STACKS

NEW YORK STATE JOURNAL OF MEDICINE

JANUARY 1985 • Volume 85, Number 1

Contents

COMMENTARIES

The emergency medical services system in New York State: time for a change 1
MARK C. HENRY, MD
BEATRICE KRESKY, MD

The need for routine sonography prior to late abortion 4
FRANK A. CHERVENAK, MD, SALLY FAITH DORFMAN, MD, CHARLES BOWERS, MD, RICHARD L. BERKOWITZ, MD, ANGELA R. HOLDER, LLM, RICHARD A. EHRENKRANZ, MD, REGINA FURLONG, ASCW, JOHN C. HOBBINS, MD

The new "regulators" of health care 6
JOHN T. PRIOR, MD

RESEARCH PAPERS

Seroepidemiology of *Legionella pneumophila* serogroup 1 in healthy residents of New York City 10
IQBAL A. POSHNI, PhD,
STEPHEN J. MILLIAN, PhD

Use of health services by merchant seamen 14
HOWARD R. KELMAN, PhD
FLORENCE KAVALER, MD

REVIEW ARTICLE

Neuro-ophthalmologic aspects of occlusive cerebrovascular disease 18
ARTHUR H. WOLINTZ, MD

FROM A COMMITTEE CHAIRMAN

The role of the Medical Society of the State of New York in workers' compensation 24
JOHN H. MORTON, MD

THE CPC

A 39-year-old man with thrombocytopenia, cough, fever, and shaking chills 27

CASE REPORTS

Factitious hypoglycemia associated with eosinophilic leukemoid reaction 34
ALAN T. LEFOR, MD, MYRON MILLER, MD

Retroperitoneal cystic lymphangioma 36
VINAY K. KAPOOR, MD,
SUKANTO K. DAS, MD,
ASHOK K. MUKHOPADHYAY, MD,
LALIT K. SHARMA, MS

Fibroma of the lung 37
HELEN Y. KINAS, MD, ROGER L. GARCIA, MD, EMMA LUGO, MD

Treatment of hemoptysis via bronchial artery embolization 38
MICHAEL TAFRESHI, MD, STEVEN SOBO, MD, LEON ADLER, MD,
ARTHUR LISS, MD,
GEORGE PILLARI, MD

Disseminated toxoplasmosis with symptoms of a cerebral lesion 40
IRIS FLETCHER NORSTRAND, MD, PhD,
KAZEM FANI, MD, FIDELIO A. JIMENEZ, MD, JOANNA HOLLENBERG SHER, MD

LETTERS TO THE EDITOR

Offshore medical schools 7
MORRIS ALPERT, MD
JULIUS E. STOLFI, MD

Effects of present and past occupational exposure 8
ALF FISCHBEIN, MD

Fiscal and moral considerations of chronic brain syndrome 9
GERALD L. GLASER, MD

1984 INDEX **follows p. 28**

AN APPRECIATION **42**
Our referees

ESSAY REVIEW
Reforming Medicine: Lessons of the Last Quarter Century 45
ALLEN D. SPIEGEL, PhD

BOOK REVIEWS **47**

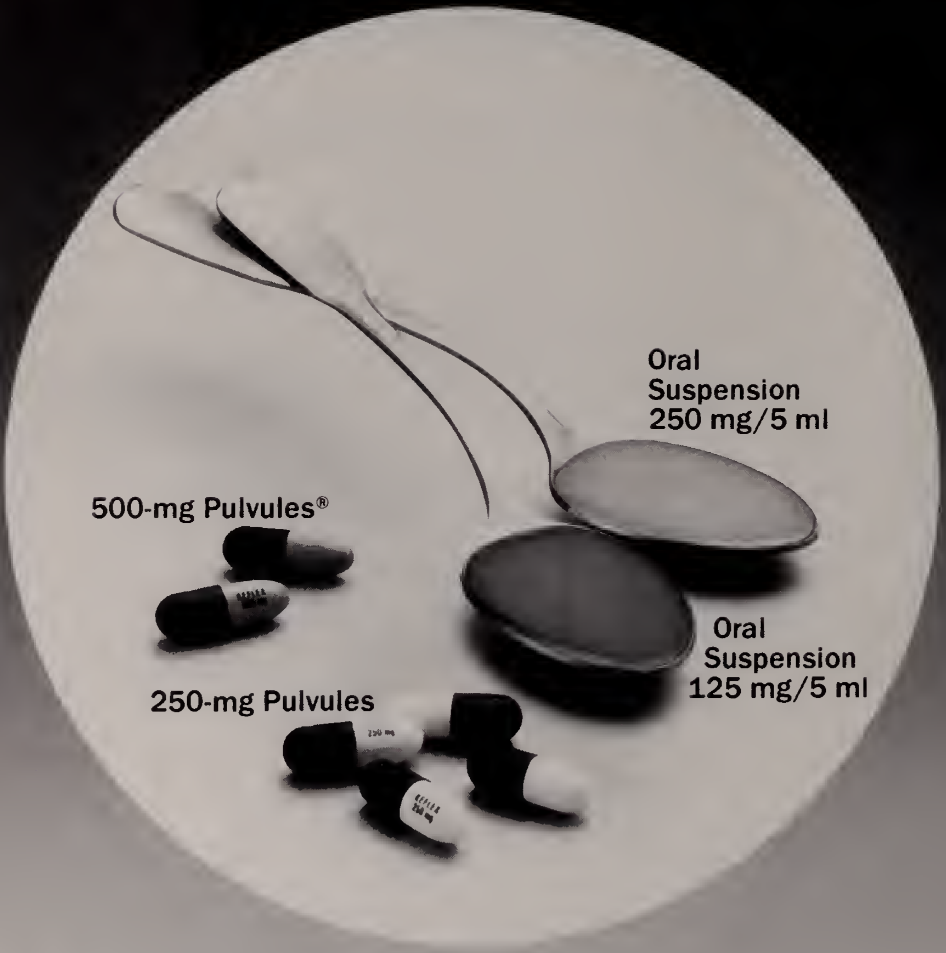
MEDICAL DIRECTORY ERRATA 50
OBITUARIES 51
GUIDELINES FOR AUTHORS

M. D. S.

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
JAN 28 1985

Library of the College Physicians
of Philadelphia Dec. 85
19 S. 22nd St.
Phila, PA 19103

Easy To Take



Keflex®
cephalexin

Additional information available to the profession on request.

DISTA
420113

Dista Products Company
Division of Eli Lilly and Company
Indianapolis, Indiana 46285
Mfd. by Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

New studies uncover the potassium effects of beta-2 blockade

Clinical pharmacology data from *The New England Journal of Medicine:*

"...when normal young men are given infusions of epinephrine at levels such as those that circulate in patients with myocardial infarction, their serum potassium concentrations fall by about 0.8 [mmol] per liter. Hypokalemia is prevented by selective beta-2 blockade."[1]

Evidence that all beta blockers are not created equal.

Right from the start in hypertension...

Once-daily INDERAL LA (propranolol HCl) for smooth blood pressure control without the potassium problems of diuretics

Once-daily INDERAL LA (propranolol HCl) avoids the risk of diuretic-induced ECG abnormalities due to hypokalemia.[2,3] In addition, INDERAL LA preserves potassium balance without additive agents or supplements while providing simple, well-tolerated therapy with broad cardiovascular benefits.

Once-daily INDERAL LA for the cardiovascular benefits of the world's leading beta blocker

Simply start with 80 mg once daily. Dosage may be increased to 120 mg to 160 mg once daily as needed to achieve additional control.

Like conventional INDERAL tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.

For beta-1/beta-2 blockade

Once-daily
INDERAL® LA
(PROPRANOLOL HCl) LONG-ACTING CAPSULES

80 mg 120 mg 160 mg

The appearance of these capsules is a registered trademark of Ayerst Laboratories.

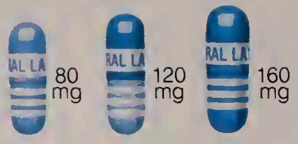
Please see brief summary of prescribing information on the next page for further details.

Once-daily

For beta-1/beta-2 blockade

INDERAL® LA

(PROPRANOLOL HCl) *LONG ACTING CAPSULES*



The appearance of these capsules
is a registered trademark
of Ayerst Laboratories.

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR)*
INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use Effects on plasma volume appear to be minor and somewhat variable INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential The significance of the membrane action in the treatment of arrhythmias is uncertain

The mechanism of the antimigraine effect of propranolol has not been established Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

Propranolol is not significantly dialyzable

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)— PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes In these patients, it may be more difficult to adjust the dosage of insulin

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General* Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure

Clinical Laboratory Tests Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy Pregnancy Category C INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose

There are no adequate and well-controlled studies in pregnant women INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers. INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman

Pediatric Use Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular bradycardia, congestive heart failure, intensification of AV block, hypotension, paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type

Central Nervous System lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue, reversible mental depression progressing to catatonia, visual disturbances, hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics

Gastrointestinal nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory bronchospasm

Hematologic agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION—*Dosage must be individualized* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic The dosage may be increased to 120 mg once daily or higher until adequate blood pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

REFERENCES

1. Epstein FH, Rosa RM Adrenergic control of serum potassium. *N Engl J Med* 1983, 309 1450-1451. **2.** Holland OB, Nixon JV, Kuhnert L Diuretic-induced ventricular ectopic activity *Am J Med* 1981,70.762-768 **3.** Holme I, Helgeland A, Hjermann I, et al: Treatment of mild hypertension with diuretics. The importance of ECG abnormalities in the Oslo study and in MRFIT *JAMA* 1984,251:1298-1299

9411/1184

Ayerst®
AYERST LABORATORIES
New York, N.Y. 10017

Copyright © 1984 AYERST LABORATORIES
Division of AMERICAN HOME PRODUCTS CORPORATION

Kodak presents...

New KODAK EKTACHEM DT60 Analyzer

A timely invest

The KODAK EKTACHEM DT60 Analyzer creates an extra service for your patients without extra investment in labor. And because it can pay for itself in three months, it's a timely investment in your future.

The chemistry tests you need

With the DT60 Analyzer you perform key chemistry tests in your own office instead of using an outside laboratory. Available tests include glucose, cholesterol, triglycerides, BUN, uric acid, sodium, and potassium, with total hemoglobin and bilirubin coming soon.

The time you need

Get test results in five minutes or less; perform up to 75 tests an hour. Save time waiting for results to assist in your diagnosis, and on follow-up phone calls.

The accuracy you need

The DT60 Analyzer uses proven technology and methodology from the KODAK EKTACHEM 400 and 700 Analyzers, which

© Eastman Kodak Company, 1984

ment for your office.

provide millions of accurate, precise results to clinical laboratories nationwide.

The simplicity you need

The DT60 Analyzer, compact as a personal computer, features dry slide technology to eliminate wet reagents. It is automated to free up your staff, and training takes only minutes. From the finger-stick sample to results printout, the DT60 Analyzer is simplicity itself.

To see what the DT60 Analyzer can do for you, write Eastman Kodak Company, Dept. 740-B, 343 State Street, Rochester, NY 14650, or call **1 800 44KODAK,** Ext 423 (1 800 445-6325, Ext 423) today.

Leading the way in healthcare technology for over 100 years.

KODAK EKTACHEM Clinical Chemistry Products

MAY NOT BE AVAILABLE IN ALL AREAS

NEW YORK STATE JOURNAL OF MEDICINE

LIBRARY, ARCHIVES, AND PUBLICATIONS COMMITTEE

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
ELIZA H. CALDWELL, MD
URSULA MATULONIS (Medical Student)
GERALD P. MURPHY, MD
RICHARD B. NOLAR, MD
CARL E. POCHEDLY, MD
JOHN T. PRIOR, MD

Editor	ALAN BLUM, MD
Deputy Editor and Book Review Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Managing Editor	VICKI GLASER
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Administrative Assistant	IMELDA MORRISSEY
Editorial Assistant	JEANNE CONNERY
Librarian	ELLA ABNEY
Library Assistant	ELEANOR BURNS
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1984

RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MARK C. HOMONOFF, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
STEPHEN NORDLICHT, MD
IRA A. POLISAR, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. Synergistic Media Sales, 50 East 42nd St., Suite 504, New York, N.Y. 10017. Telephone: (212) 682-2200.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

ALLISON B. LANDOLT, MD, Westchester	*President*
BERNARD J. PISANI, MD, New York	*Past-President*
DANIEL F. O'KEEFFE, MD, Warren	*President-Elect*
JOHN A. FINKBEINER, MD, New York	*Vice-President*
JOHN H. CARTER, MD, Albany	*Secretary*
GEORGE LIM, MD, Oneida	*Assistant Secretary*
CHARLES D. SHERMAN, JR, MD, Monroe	*Treasurer*
MORTON KURTZ, MD, Queens	*Assistant Treasurer*
JOHN L. CLOWE, MD, Schenectady	*Speaker*
CHARLES N. ASWAD, MD, Broome	*Vice-Speaker*

Councilors

Term Expires 1985	JAMES H. COSGRIFF, JR, MD, Erie SAMUEL M. GELFAND, MD, Nassau RICHARD A. HUGHES, MD, Warren ANTONIO F. LASORTE, MD, Broome
Term Expires 1986	FREDERIC W. HOLCOMB, JR, MD, Ulster ROBERT A. MAYERS, MD, Westchester THOMAS D. PEMRICK, MD, Rensselaer RALPH E. SCHLOSSMAN, MD, Queens STANLEY A. STECKLER, MD, Suffolk
Term Expires 1987	DAVID M. BENFORD, MD, New York DUANE M. CADY, MD, Onondaga JAMES M. FLANAGAN, MD, Wayne M. THEODORE TANENHAUS, MD, Brooklyn

Trustees

CARL GOLDMARK, JR, MD, New York, *Chairman*
G. REHMI DENTON, MD, Albany
RALPH M. SCHWARTZ, MD, New York
EDGAR P. BERRY, MD, New York
RICHARD D. EBERLE, MD, Onondaga
JOSEPH F. SHANAPHY, MD, Richmond
VICTOR J. TOFANY, MD, Monroe

Executive Vice-President	MILTON ROSENBERG, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Associate	IRMA A. ERICKSON
Director, Division of Scientific Publications	ALAN BLUM, MD
Director, Division of Management Services	GEORGE FORREST, JR
Assistant Director, Division of Governmental Affairs	JERRY S. HOFFMAN, ESQ
Director, Division of Communications	EDWARD HYNES
Director, Division of Insurance and Membership Benefits	BERNARD M. JACKSON
Director, Division of Membership	EUNICE SKELLY
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Scientific Activities	GEORGE J. LAWRENCE, JR, MD
Director, Division of Advertising-Sales and Meeting Services	ROBERT MILLER
General Counsel	DONALD R. MOY, JD
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Medical Services	ROBERT J. O'CONNOR, MD
Director, Division of Long-Range Planning	DEBORAH R. SIEGEL
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. The proceedings of the Society's House of Delegates meeting are published in March as a separate issue of the *Journal* with limited circulation. Editorial material published in the *Journal* is covered by copyright. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

Classified Advertising

PHYSICIANS WANTED

INTERNIST WANTED, NASSAU COUNTY, NY Board certified/eligible, with/without subspecialty to join growing private practice. Reply Dept. 358, c/o NYSJM.

FAMILY PHYSICIAN—Northeastern New York Board Family Physician wanted to join active, private, group practice of family medicine, all board certified family physicians, including obstetrics and hospital care in an excellent local community hospital. Suburban Albany area. Contact: Business Manager 1-(518) 785-4471.

EMERGENCY PHYSICIANS—Upstate New York Syracuse area hospital with 18,000 visits and moderate trauma. Immediate need for physicians offering above average hourly income. Finger Lakes region, Corning, NY. Opportunity for F/T experienced emergency physician Director in progressive community hospital. ED volume 12,500 visits with steady growth in income for past 5 years. Please call or write, Ken Baker, Director of Physician Recruitment, Fischer Mangold Group, P.O. Box 788, Pleasanton, CA 94566, (800) 227-2092.

ORTHOPEDIC SURGEON: Excellent opportunity for board eligible/certified Orthopedic Surgeon to replace same in medium-sized city in progressive/rural Eastern Pennsylvania. Candidate should have significant interest in sports medicine. Call Judith Kennedy at 1-800-441-0996 or in PA at 215-896-5080 collect.

PHYSICIAN WANTED TO SHARE OFFICE SPACE with an older general practitioner with intention of sale later on retirement. Pleasant, rural community in Finger Lakes area. Situated on lake. Hospital nearby. Terms negotiable. (315) 536-4456, nights (315) 536-8084.

PHYSICIAN DURING JULY AND AUGUST, 1985 for children's camp located at Beach Lake, PA, accommodates 350 campers, age 6–16; complete modern Health Center; 2 R.N.'s in attendance; will accept one M.D. for each month; no children accepted who are of camp age. Camp opens June 30 and closes August 24. Private room and facilities. Write to: Trail's End Camp, c/o Beach Lake, Inc., 215 Adams Street, Brooklyn, NY 11201 and include your phone number.

OBSTETRICIAN/GYNECOLOGIST: Attractive opportunity for board eligible/certified OB/GYN to establish practice in medium-sized city in progressive/rural Eastern Pennsylvania. Candidate must be supportive of hospital midwifery program. Call Judith Kennedy at 1-800-441-0996 or in PA at 215-896-5080 collect.

SEMI-RETIRED INTERNIST OR GP to join a busy Internist in Upstate New York largely office based practice needed in February 1985. Send CV to Dept. 371, c/o NYSJM.

PHYSICIANS WANTED—CONT'D

PHYSICIAN FOR CHILDREN'S SUMMER CAMP. Located in lower NYS Catskills, CAMP ECHO seeks Physicians for July and August 1985. Physicains for two or four week periods, with or without camp age children. Excellent medical facility, 2 R.N.'s + aide, private rooms and facilities for M.D. Camp accommodates 350 boys and girls from 6-16 yrs. of age. Write CAMP ECHO, P.O. Box 255, Roslyn, NY 11576 or call (516) 742-2679.

GENERAL/FAMILY PRACTITIONERS, if you are looking for an opportunity to be in the forefront of medical care, practice preventive medicine, work with other innovative professionals, and earn a comfortable living in pleasant surroundings, send your curriculum vitae to Physician Placement Dept.—38. An equal opportunity employer. CIGNA Healthplans of California, 700 N. Brand Blvd., Suite 500, Glendale, CA 91203.

OBSTETRICIAN/GYNECOLOGIST: Second OB/GYN needed to join this rural group practice of 10 physicians. Primary care is emphasized. Formal tertiary linkages to Rochester General Hospital for high risk. Base salary, then income tied to productivity. Contact: Richard F. Endres, M.D., Sodus Health Center, P.O. Box A, Sodus, NY 14551. EOE.

FAMILY PRACTICE GROUP seeks BC/BE Family Practitioner. Early Partnership, five weeks vacation. Excellent hospitals. Beautiful college town in the Hudson Valley vacation area 90 miles north of N.Y.C. Address CV to Administrator, Medical Associates of New Paltz, PC, 40 Sunset Ridge, New Paltz, NY 12561.

IMMEDIATE OPENING with competitive salary, excellent fringes, and scheduled time off for an energetic, BE/BC Primary Care Physician with E.D. Training or experience. Regional control of "Paramedics" requires excellent command of written and spoken English. Inhouse Dialysis, Cardiologist, and upcoming new Emergency Department with 20,000 visits per year. Summer (Thousand Islands) and Winter (Lake Placid) sports and recreation well known. Growing town of +30,000 with service area of +80,000. Send CV to Phillip Tatnall, M.D., Director, Emergency Services, Mercy Hospital of Watertown, Watertown, New York, 13601. (315) 782-7400.

GERIATRICIAN/INTERNIST—Full time Senior Physician to teach in fellowship and student programs, conduct research and participate in incidental clinical responsibilities. Requires physician with 4 year's geriatric experience in acute, long term, home care and out-patient programs. Should qualify for appointment at ass't professor level. Competitive salary. Send c.v. to Conn J. Foley, M.D., Medical Director, Jewish Institute for Geratric Care, 271-11 76 Avenue, New Hyde Park, N.Y. 11042.

BOARD CERTIFIED INTERNIST: Preventive, Occupational Health—a leader in field offers varied opportunities (full-time, half-time or per diem) at our clinics and corporate medical units. Send C.V. to Executive Health Examiner, 777 Third Ave., N.Y., N.Y. 10017, Attn: Personnel Director. "EOE M/F."

PHYSICIANS WANTED—CONT'D

WANTED: INTERNIST CARDIOLOGIST. Must be board certified or eligible City of 25,000 in beautiful Upstate New York. Excellent 200 bed local hospital a ½ block away. Small group with large practice, well-established for 40 years. Permanent placement. Complete in-house clinical lab, x-ray and physical fitness lab. Opening must be filled by July 1, 1985. Send CV to 110 No. Main St., Cortland, New York 13045.

BOARD CERTIFIED DIAGNOSTIC RADIOLOGIST for large radiology practice serving multiple office and hospital locations in Northeast, Ohio. The successful candidate should preferably have 1–3 years post-residency experience as a fellow or on staff. Remuneration, benefits, and vacation time are all excellent. Reply Dept. 373 c/o NYSJM.

GASTROENTEROLOGIST wanted to associate with two board certified Gastroenterologists in upstate NY close to mountains, skiing and recreational areas. Must be university trained and proficient in all procedures. Located in close proximity to university centers. Reply Dept. 375 c/o NYSJM.

OFFICE SPACE, three (13′ x 14′) offices, with connecting hall, adjacent fully equipped diagnostic center. Common reception area. Plainview, Long Island. Seeking Board Certified Internist, Orthopedic Surgeon and Cardiologist. Will consider all specialties. Opportunity to work part-time for diagnostic center. Use of diagnostic center equipment and space for private practice negotiable. Call for appointment (516) 822-4600.

PHYSICIANS needed for 75 residential children's camps located in Northeast. July and/or August. Contact: Association of Independent Camps (M), 60 Madison Ave., New York, NY 10010. (212) 679-3230.

POSITIONS WANTED

INTERNIST—seeking a position in a group practice that is looking for an energetic, bright, young physician recently graduated from an excellent Connecticut residency program, Yale affiliated. Interested in area that is commuting distance to Manhattan as wife is employed there. Eventual partnership opportunity a must. Reply Dept. 370, c/o NYSJM.

AP/CP CALIFORNIA PATHOLOGIST, 36, seeks to relocate near NYC, small or medium-sized hospital. Strong community-type background, eight years experience. Well grounded in medical staff and clinical laboratory functions. C.V. on request. J. Vasser, M.D., 7816 Summerplace Drive, Citrus Heights, Calif. 95621.

RESIDENCY TRAINED FAMILY PHYSICIAN, Chief resident, Board Eligible July 1985. Columbia P and S graduate, trained in medicine, pediatrics, office gynecology, geriatrics, office surgery. Seeking practice opportunity Nassau/Queens County this July. C.V. and references furnished upon request. Contact: Monte Nussbaum, M.D., 23 Upper Overlook Road, Summit, NJ 07901, (201) 522-9537.

New variable rate
U.S. Savings Bonds
guarantee
investment growth...

The employees at BURLINGTON INDUSTRIES keep their future plans on target when they rely on the Payroll Savings Plan.

Nancy B. Turner — "Currently, these bonds are accumulating for a college fund for our son. And too, we feel it's necessary for each of us to re-invest in America."

John Maier — "I would tell other employees that it is an easy way to use small amounts of money through payroll deduction to build toward a lump sum gift for a child or loved one where that person benefits from the earnings."

Lois Bailiff — "In a small way we are helping the future of our country as well as building up our savings for retirement. Bonds have given our family extra money to buy items for our home and for graduation and wedding gifts."

Take stock in America.

Director of Sales
U.S. Savings Bonds Division
Department of the Treasury
Washington, D.C. 20226

Yes, please send me *Free* information about the Payroll Savings Plan.

Name ______

Position ______

Company ______

Address ______

City ______ State ______ Zip ______

Ad Council — A public service of this publication and the Advertising Council

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meetings and lectures unless the information is submitted at least two months prior to the event.*

FEBRUARY 1985

AROUND THE STATE

MANHATTAN

Feb 6. **Personal Computing for Physicians.** 4 Cat 1 credits. Info: Beverley R. Baptiste, Post-Graduate Education Office, Beth Israel Medical Center, Nathan D. Perlman Place, New York, NY 10003. Tel: (718) 420-2849.

Feb 11–15. **Head & Neck Surgery.** 40 Cat 1 credits. Mount Sinai Medical Center. Info: Page & William Black Post-Graduate School of Medicine, One Gustave L. Levy Place, New York, NY 10029. Tel: (718) 650-6737.

Feb 19. **Depression, Dementia and Non-Compliance in Patients Undergoing Programmed Electro-Psychological Arrhythmia Induction.** Lenox Hill Hospital. Info: Mrs V. Matthes, 542 Baldwin Ave, Baldwin, NY 11510. Tel: (516) 379-1787.

Feb 19. **New York Downstate I & II Regional Meeting of the American College of Physicians.** Grand Hyatt Hotel. Info: Arthur C. Fox, MD, 550 First Ave, New York, NY 10016. Tel: (718) 340-6405.

Feb 23. **The Use of Countertransference Data in Psychotherapy.** 5 Cat 1 credits. Info: William Alanson White Institute of Psychiatry, 20 W 74th St, New York, NY 10023.

BROOKLYN

Feb 6. **Cancer in Infants.** *Feb 13.* **Chemotherapy of Non-Small Cell Lung Cancer.** *Feb 20.* **AIDS in Children—Epidemiology, Immunology, and Treatment.** *Feb 27.* **Colo-Rectal Cancer.** Info: Interfaith Medical Center, 555 Prospect Place, Brooklyn, NY 11238. Tel: (718) 240-1211.

Feb 17. **Update on the Management of Thromboembolic Disorders.** Info: Hazel Spadafora, Medical Society of the County of Kings, 1313 Bedford Ave, Brooklyn, NY 11216. Tel: (718) 467-9000

NASSAU

Feb 7. **Insulin Therapy.** *Feb 14.* **Nutrition in the Critically Ill Patient.** *Feb 21.* **Hydrocephalis and Vasospasm Following Subarachnoid Hemorrhage.** *Feb 28.* **The Mast Cell: Its Role in Health and Disease.** East Meadow. Info: Lectures in CME, Nassau County Medical Center, 2201 Hempstead Turnpike, East Meadow, NY 11554. Tel: (516) 542-0123.

Feb 7. **Meeting the Mental Health Needs of Children and the Family.** *Feb 14.* **It's a Virus.** *Feb 21.* **Diagnosis of Inherited Metabolic Diseases.** *Feb 28.* **Infant Intervention: Methods and Efficacy.** East Meadow. Info: Lectures in Continuing Pediatric Education, Nassau County Medical Center, 2201 Hempstead Turnpike, East Meadow, NY 11554. Tel: (516) 542-0123.

QUEENS

Feb 27. **Medical Problems in the Geriatric Patient.** Flushing. 3 Cat 1 credits. Info: Luisa Gonzalez, Flushing Hospital and Medical Center, 146-01 45th Ave, Flushing, NY 11355. Tel: (718) 670-5741.

Feb 20–June 12. **The Fifteenth Annual Pediatric Board Review Course.** 32 Cat 1 credits. New Hyde Park. Info: Ann J. Boehme, CME Long Island Jewish-Hillside Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-2114.

ERIE

Feb 19. **Radiation Effects.** 5 Cat 1 credits. Buffalo. Info: Ms. Gayle Bersani, Education Office, Roswell Park Memorial Institute, 666 Elm St, Buffalo, NY 14263. Tel: (716) 845-2339.

AROUND THE NATION

CALIFORNIA

Feb 11–15. **San Diego Abdominal and Obstetrical Imaging Symposium Update.** San Diego. Info: Dawne Ryals, PO Box 610203, DFW Airport, TX 75261-0203. Tel: (214) 659-9590.

Feb 13–15. **Recent Advances in Neurology.** 16 Cat 1 credits. San Francisco.

FOR SEVERE COUGHS, CAN YOU REMEMBER THESE LETTERS?

You know Robitussin®, and the "DAC" formula gives you just what you need to manage those severe coughs complicated with nasal congestion . . . Decongestant And Codeine!

Robitussin®-DAC Ⓥ

Each 5 ml (1 teaspoonful) contains:

Guaifenesin, USP	100 mg
Pseudoephedrine Hydrochloride, USP	30 mg
Codeine Phosphate, USP	10 mg

(Warning: May be habit forming)
Alcohol 1.4 percent

℞ ONLY

A-H-ROBINS
Pharmaceutical Division
Richmond, Virginia 23261-6609

New study reveals no interaction between

Ativan® (lorazepam) and Darvon® (propoxyphene HCl) Ⓒ

In a study evaluating the influence of propoxyphene coadministration on the pharmacokinetics of the oxidatively metabolized benzodiazepines Xanax® (alprazolam) Ⓒ and Valium® (diazepam) Ⓒ, and a benzodiazepine metabolized by conjugation, Ativan® (lorazepam), the following results were reported:

with Xanax, propoxyphene caused a large and highly significant prolongation of half-life and impairment of total metabolic clearance.[1]

in the case of Valium, propoxyphene produced a small but not statistically significant impairment of clearance.[1]

propoxyphene had no apparent effect on the distribution, half-life or clearance of Ativan.[1]

In this randomized crossover study, eight healthy male and female volunteers received single oral doses of alprazolam (1 mg), six received single IV doses of diazepam (10 mg), and five received single IV doses of lorazepam (2 mg), once in a drug-free control state and again during coadministration of propoxyphene (65 mg q6h). Consistent with previous findings, this study evidences that Ativan does not interact with drugs that undergo oxidative metabolism.[2-5] In contrast to most other benzodiazepines, Ativan does not compete for the cytochrome P-450 enzyme system.

The clinical implications of the pharmacokinetic interaction, or non-interaction, of propoxyphene with benzodiazepines are not established by this study. Even without a pharmacokinetic interaction, propoxyphene and benzodiazepines share central depressant properties and therefore should be coadministered with suitable caution. A concurrent pharmacokinetic interaction indicates a need for even further caution. Coadministration of propoxyphene and alprazolam, for example, would produce not only the expected pharmacodynamic interaction, but also whatever additional central depressant effect would be produced by the elevated steady-state plasma concentrations of alprazolam due to its impaired clearance.

Caution should also be observed when propoxyphene is prescribed for patients who use alcohol to excess.

References

1. Abernethy DR, Greenblatt DJ, Shader RI: Data on file, Wyeth Laboratories.
2. Patwardhan RV, Yarborough GW, Desmond PV, et al: Gastroenterology *79*:912-916, 1980.
3. Abernethy DR, Greenblatt DJ, Divoll M, et al: Psychopharmacology *80*:275-278, 1983.
4. Abernethy DR, Greenblatt DJ, Divoll M, et al: N Engl J Med *306*:791-792, 1982.
5. Ochs HR, Greenblatt DJ, Abernethy DR: Data on file, Wyeth Laboratories.

Wyeth Laboratories
Philadelphia, PA 19101
TM

© 1984, Wyeth Laboratories.

Ativan® for (lorazepam) Ⓒ Anxiety

See important information on following page.

DISTINCTIVE DESIGN® 0.5, 1.0 and 2.0 mg

The appearance of these tablets is a registered trademark of Wyeth Laboratories

Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Transient amnesia or memory impairment has been reported in association with the use of benzodiazepines.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® Ⓒ IV
for (lorazepam)
Anxiety

DOSAGE: Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.

HOW SUPPLIED: 0.5, 1.0 and 2.0mg tablets.

Wyeth Laboratories
Philadelphia, PA 19101
TM

POSITIONS WANTED—CONT'D

GASTROENTEROLOGIST, 30. Completing my fellowship at Nassau County Medical Center in June, 1985. Hahnemann Graduate, Board Certified Internal Medicine. Well trained in all procedures. Desires position. Contact: Daniel E. Shufler, M.D., 200 Carman Ave. #20B, East Meadow, NY 11554, (516) 794-2370 (Home) or (516) 542-2573 (work).

FAMILY PHYSICIAN/GENERAL PRACTITIONER temporary or permanent affiliation. Full-time preferred, but can accommodate part-time hours. No obstetrics. Minimum 3 months, to start at once. Astoria, Queens, (718) AS8-1909.

PEDIATRICIAN: University trained, Board eligible, 3 years private practice experience (NJ). Wishes to relocate for better opportunities. Hospital, Health Organization or partnership potential with private practitioner. Available immediately. Reply Dept. 377 c/o NYSJM.

PRACTICES AVAILABLE

OPHTHALMIC SURGI-CENTER and large eye clinic available at once. Capacity for over 400 major and 900 minor surgical procedures. Eye clinic capacity 12,000 per year. Deluxe living quarters. N.A. Karakashian, M.D. (215) 423-4477.

FOR SALE: Lucrative, long established family practice in suburban area 35 miles north of N.Y.C. Fully equipped office complete with X-ray and EKG, attached to large comfortable four bedroom home. Acreage also available. Reason for sale Retirement. Reply Dept. 372 c/o NYSJM.

INTERNAL MEDICINE PRACTICE FOR SALE. North Grand Concourse, Bronx. Fully equipped, X-Ray, ECG, Lab., etc. Retiring, call (914) 277-4430 after 5 P.M.

MEDICAL PRACTICES FOR SALE—Are you contemplating selling or buying a medical practice? We can help. PRACTICE FINDERS, INC. 45 East 89th Street, New York, NY 10028, Phone (212) 860-2206.

PRACTICES WANTED

YOUNG INTERNIST, wishes to purchase Nassau County office based or nursing home based private practice. Available immediately. Strictly confidential. Reply Dept. 365, c/o NYSJM.

INTERNIST wishes to purchase or join private practice in Eastern Nassau County or Western Suffolk. Available July 1985. Reply Dept. 374 c/o NYSJM.

From Blue Cross and Blue Shield of Greater N.Y.

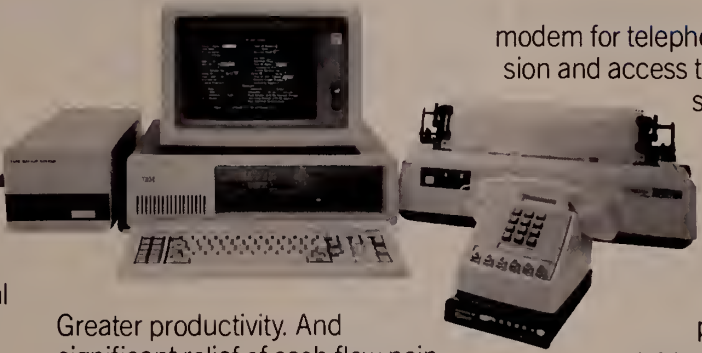
The last claim form!

Available with the new IBM® PC-AT!

Introducing

Amicus 1™

MEDICAL OFFICE MANAGEMENT SYSTEM



A computer system that does something you could never do before

For over 50 years, Blue Cross and Blue Shield of Greater New York has been the most consistently innovative force in helping physicians and patients meet the financial demands of progressive medical care. Now, with advances in the technology of computer networking, we can help you do something never before possible. Amicus 1 lets you transmit claims directly by phone to our computer for faster, more efficient processing of our medical and Medicare B* claims.

Direct communication provides better service

Just as direct communication between people is preferred for accuracy, computer-to-computer communication reduces the potential for errors and delays. When you purchase Amicus 1, you gain significant benefits. Faster collections and fewer errors. Greater productivity. And significant relief of cash flow pain.

Handles most of your accounting and financial management functions

Of course, Amicus 1 does more than just send claims to our computer. It assists you in all your billing and accounts receivable needs. It provides up to the minute financial reports on your practice. And more. All with just a few short entries.

A complete system featuring the IBM® Personal Computer XT

We've developed a ready-to-use system featuring an IBM Personal Computer XT. In addition, Amicus 1 includes a high speed printer; a tape backup storage unit; and a fast modem for telephone data transmission and access to medical and financial services. The Amicus 1 system includes installation with a three-day training program for your office staff. It also includes an on-site service contract plus a special telephone response line available during business hours.

Fill out your last claim form.

Just complete and mail the coupon below. Or, call us at **212 490-4180** to learn how Amicus 1 can help reduce your paperwork, increase your productivity and improve your cash flow.

Tell me how Amicus 1 can work for my practice.
☐ Yes, I'd like to see a personal demonstration.
☐ Please send me more information. NYSJM

1. NAME
2. SPECIALTY
3. ADDRESS *(Street, city, state, ZIP code)*
4. TELEPHONE ( )
5. OFFICE MANAGER

Send completed form to: **Medical Business Services**
P.O. Box 1707 Grand Central Station
New York, N.Y. 10163

Medical Business Services
Div. of Blue Cross. Blue Shield. of Greater New York

*Applies to the 16 counties where Blue Cross and Blue Shield of Greater N.Y. is the Medicare carrier
IBM® is a registered trademark of International Business Machines Corporation
Amicus 1™ is a trademark of Blue Cross and Blue Shield of Greater New York

1985 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
New York Hilton, New York City

FRIDAY, JUNE 7

8:00 a.m. - 10:00 a.m.	Meet the Professor Breakfast Meetings (5)
10:00 a.m. - 1:00 p.m.	Cardiovascular Diseases Pediatrics/Dermatology Obstetrics/Gynecology Neurosurgery
2:00 p.m. - 5:00 p.m.	Neurology Medical Legal & Workers´ Compensation Cancer Update Chest Diseases Computers in Medicine

SATURDAY, JUNE 8

10:00 a.m. - 1:00 p.m.	Ophthalmology (All day) Urology/Pathology Internal Medicine (All day) General Surgery/Orthopedic Surgery/Gastroenterology Anesthesiology/Radiology Psychiatry/Alcoholism (All day)
2:00 p.m. - 5:00 p.m.	Ophthalmology Otolaryngology Emergency Medicine Internal Medicine Psychiatry/Alcoholism

SUNDAY, JUNE 9

8:00 a.m. - 10:00 a.m.	Meet the Professor Breakfast Meetings (5)
10:00 a.m. - 1:00 p.m.	Allergy & Immunology Women´s Medical Society of New York State/Family Practice The Doctor as a Witness

◆

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis.

◆

Technical Exhibits

NEW YORK STATE
JOURNAL OF MEDICINE

January 1985 • Volume 85, Number 1

COMMENTARIES

The emergency medical services system in New York State: time for a change

Of the 6,215,560 patients cared for in emergency departments in New York State in 1980, approximately 912,000 patients required emergency admission to a hospital, accounting for 33% of all hospital admissions.[1,2] Heart disease, cerebrovascular disease, and accidental injuries are the leading causes of sudden death and disability.[2] An emergency medical services system (EMSS) to curtail avoidable deaths and injuries is comprised of the following components: public education regarding availability and access to emergency care facilities as well as techniques for assisting fellow citizens; first aid measures by police and fire units; prompt application of sophisticated life-support measures by ambulance personnel; well-designed means of transportation; and hospitals with appropriate staffing and resources.[3–5] All of these components should be under medical supervision. An EMSS recognizes that emergency medical care is a continuum from the scene of the accident or sudden illness through the definitive phases of critical hospital intervention. To provide the public with an effective emergency medical response, efforts and resources must be committed that are commensurate with the magnitude of the problem and the number of patients who require emergency care. This goal, although feasible, still remains in its developmental stages in New York and most other states.

The concept of and the need for an EMSS were well expressed in 1966 in a paper entitled "Accidental Death and Disability: The Neglected Disease of Modern Society" prepared by the National Academy of Sciences/National Research Council Committees on Shock and Trauma.[6] Initiated in large part as a response to this document and encourged by early efforts in systematic delivery of trauma care in Illinois and Maryland,[7,8] the federal Emergency Medical Services System Act of 1973 was passed "to provide assistance and encouragement for the development of comprehensive area wide Emergency Medical Services Systems."[9] In addition to addressing unnecessary deaths due to trauma and accidental injuries, the act also recognized the importance of EMSS in treating patients with other categories of critical illnesses such as myocardial infarction or poisonings.

Many regions in New York State began initial EMSS development as a result of this federal legislation. In 1974, Article 30 of the New York State Public Health Law was amended to permit the provision of advanced life support measures by paramedics, to establish state and regional emergency medical services councils, and to charge the state council with "the development of a statewide emergency medical services system." However Article 30 did not address emergency patient care in the hospital phase.

Based on the results to date in New York State, it is clear that further change is necessary in the existing legislation to establish an emergency medical services system. It must be recognized that whereas significant activity took place in many of the 16 emergency medical services regions that were established in the state, many of the regions did not receive federal monies, and program development in these areas has lagged behind. Furthermore, under the present structure, there are impediments that limit future progress statewide. This is understandable from a historical perspective. When one considers that Article 30 was written at a time when the EMSS concept was still being developed, it was appropriate to define emergency medical service as "initial emergency medical assistance"; this left the system concept—which includes the continuum through the hospital and definitive phases of emergency care—as a developmental task. Now, nearly 10 years later, there is enough experience both nationwide and statewide to acknowledge that Article 30 requires updating to promote the systematic delivery of emergency medical care through the "prehospital" and hospital phases.

Major legislative changes necessary to implement an EMSS must address state and regional authority as necessary to carry out the functions of an EMSS including authority to categorize (review against a set of standards) and designate (select for purposes of patient referral and transfer) the most effective emergency hospital resources; the inclusion and coordination of all prehospital and hospital providers with appropriate standards of care for both; and formation of state and regional medical boards that will apply medical standards and medical control systems to ensure that the medical care delivered in the prehospital and hospital phases results in optimal benefit to patients.

The term prehospital care, now used by specialists in emergency medicine and allied personnel, refers to initial medical care delivered prior to arrival at a hospital by personnel with various degrees of training. This includes care delivered by "first-responders" (for example, police and firemen), good Samaritans, emergency medical technicians, and paramedics who have received varying degrees of medical training as well as by physicians traveling to the scene. In New York State most prehospital care is provided by nonphysician personnel.

Standards for prehospital care providers were developed through Article 30 of the public health law and subsequent regulations. However, this statute specifically exempted existing volunteer ambulance crews from meeting any minimum standards of training. Since 82% of the prehospital providers in New York State are volunteers, this exemption makes it difficult to define the capabilities of a large portion of the first medical response to emergency situations. In 1983 only 33% of the ambulance services in New York State were certified as meeting appropriate standards of staff training and ambulance equipment.[10] It is difficult to request ambulance personnel to follow treatment protocols when their level of training is not clearly defined. All ambulance personnel must be certified as meeting minimum levels of training at the emergency medical technician-ambulance level. Funds must be appropriated for this training. All ambulance corps should be certified and incorporated into the regional delivery systems. Exceptions to standards can allow inadequately trained individuals to serve as ambulance personnel and can result in poor patient care.

Changes are also required to provide for sound medical control of all prehospital ambulance services, including those that provide basic as well as advanced life support. Certified basic life support services are staffed by emergency medical technicians with at least 110 hours of training. Advanced life support services are staffed by advanced emergency medical technicians with extra training of up to nearly 900 hours. Present regulations require physician direction and supervision for advanced life support services only. Physician involvement has tended to focus on these advanced life support systems, which constitute considerably less than half of the prehospital services in the state. Development of advanced life support prehospital care should continue to be encouraged, since it is directed toward patients with life threatening conditions that can cause death within minutes if left untreated. The advanced life support systems extend physician-level care to critical patients in the field through a physician's orders to appropriately certified personnel. Whereas advanced life support services should continue to be developed, physicians must become just as involved with the basic life support services used in the prehospital sector to treat the majority of the patients in the state.

The federal legislation stimulated categorization and designation of hospitals to identify the facilities most able to care for patients with selected categories of critical illnesses and injuries. Once hospitals have been categorized and designated, treatment and transfer policies can be effected to deliver the patient from the scene to the most appropriate facility to treat his or her condition. It was recognized that the closest hospital was not necessarily the best hospital to go to in time of certain emergencies. Therefore designation of both emergency receiving hospitals (for initial evaluation, resuscitation, and stabilization) and specialty referral centers (for definitive care of selected categories of illness or injury) was needed. In New York State the Governor's Task Force on Emergency Medical Services recommended in 1979 that guidelines for the categorization process be developed "so that hospitals may be categorized according to their ability to care for the patient in need of critical care, and in a manner to assure a comprehensive range of care within a defined geographical region." Such guidelines were developed and approved by the State Emergency Medical Services Council in 1981.[11]

The categorization process was undertaken in nearly half the regions within the state. Standards of care for emergency departments and specialty referral hospitals—trauma centers—were developed, hospitals were audited, and hospitals were designated in the respective regions, usually by lead EMS agencies who were recipients of state or federal grants. The results were uneven and incomplete in most of the regions. The main reason for this was the lack of clear cut authority for the hospital categorization and designation process. With no federal or state requirements, hospital participation was strictly voluntary.[11] This has led to uneven hospital participation in many regions and no activity at all in others. Other factors impeding the process include political forces, reimbursement issues, and the general lack of priority of emergency services—factors that have been noted as problems in other states as well.[12] The lack of uniform voluntary participation has prevented the public from knowing in advance which hospital to go to in the event of a life threatening emergency.

A legal authority such as the State Health Department must assume responsibility for categorizing and designating emergency receiving hospitals in order for EMSS development to continue. Moreover, regional plans for hospital care must address bed and staffing needs, critical care resources, and reimbursement issues that result from the regionalization of delivery of emergency care and changes in patient referral patterns that accompany EMSS development. Regional EMSS councils are appropriate bodies to carry out the categorization process in each region, in conjunction with the respective health systems agencies and hospital planning councils. The number of regional councils should be reduced to six, equivalent to the regional areas of the State Health Department, or eight, equivalent to the Health Systems Agency areas for purposes of hospital categorization. Their recommendations will require approval by the State Health Department, which should have ultimate authority over the EMSS of New York State.

The standard of care in many regions where the categorization process was voluntarily undertaken has improved considerably. Major changes included provision of adequate staffing in numbers sufficient to meet anticipated patient volumes. Qualifications for physician, nursing, and ancillary staff, as well as the ratio of staff to patient volume were clearly delineated in some regional standards.[13] Another improvement was the acquisition of necessary equipment. Although the need for emergency care to an individual patient is episodic, to an institution and health planners it is predictable. Hospitals are aware of the numbers of patients who seek emergency care daily. Linking staffing and equipment requirements to patient volume allows for efficient allocation of resources.

The improvements in the delivery of hospital emergency medical services required additional resources in many cases. However, one questions whether the standard of care is adequate in those hospitals that do not meet local standards. Not all hospitals in a given region must meet the highest standard. Duplication of effort and resources is unnecessary if there are ample and readily available facilities to deal with expected patient volumes. Several levels of standards may be appropriate. However, all hospitals should be reviewed to see that they meet minimum standards for emergency care. Transfer agreements between hospitals can ensure that the patient arriving by ambulance or other transportation will be expeditiously referred to the appropriate facility when a hospital meets the limits of its ability to care for certain patients. Such transfer mechanisms are well known in the care of burn patients who are referred to burn centers. Similar arrangements are just as applicable to other patients, such as those with multiple trauma, poisonings requiring dialysis, and high risk neonates. All hospitals must participate in the Emergency Medical Services System according to their ability.

Public education should be offered on a regional basis, since varying geographic resources and time factors will dictate the most prudent action to be taken by the patient in need of immediate care. Furthermore, care must be taken to avoid negative implication for hospitals not designated as emergency receiving hospitals or specialty referral centers.[14]

Emergency Medical Services Medical Boards must be established at state and regional levels to ensure sound medical control of all aspects of the system. To avoid the fragmentation of care that can result otherwise, treatment, triage, transport, and transfer protocols are required. These protocols should be formulated by regional medical boards working together with regional EMS councils. The medical boards should be comprised of physicians who are providers within the emergency care community and who are involved and familiar with prehospital and hospital emergency and critical care. Regulations must be developed to ensure accountability by a physician for the medical conduct of all prehospital personnel.

There should be at least one physician-advisor available for every ambulance corp to ensure consistency and appropriate supervision of clinical instruction, case review, continuing education, and evaluation of effectiveness. Medical direction works best when an individual physician accepts responsibility for a limited number of emergency medical technicians. While medical boards can work toward uniformity within the state and its regions, the individual physician functioning as the medical director of the local ambulance corp is the cornerstone of medical control.

Medical boards should also develop or approve medical standards for categorization and designation of emergency receiving hospitals and specialty referral centers. Medical involvement in this stage is vital since EMSS development sets the standards of care for the most critically ill and injured patients in the community. Each regional body should appoint a medical director responsible for the day-to-day operation of the system, implementation of the policies decided upon by the regional council and medical board, and ongoing supervision of the system of medical control for prehospital care.

Emergency medical services system development is a public health issue that requires the cooperative efforts of the entire community. Because of the comprehensive scope of emergency medical care, many forces must join together to effect the necessary legislative and health policy changes in order to move forward. Currently the State EMS Council, the Medical Society of the State of New York, regional EMS councils, health planning bodies, and individuals are working on plans and legislative proposals to accomplish many of these goals.[2] Because physicians have the major responsibility for patient care, they must assume a leadership role in the development and implementation of an emergency medical services system in their own communities.

MARK C. HENRY, MD
Director
Department of Emergency Medicine
Booth Memorial Medical Center
Main St at Booth Memorial Ave
Flushing, NY 11355

BEATRICE KRESKY, MD
Chairman
Department of Ambulatory Care
The Jamaica Hospital
89th Ave and Van Wyck Expressway
Jamaica, NY 11418

1. *Statewide Planning and Research Cooperative System Annual Report Series 1980.* Albany, SPARCS, New York State Department of Health, 1980. (This was the last year that these statistics were compiled by SPARCS.)

2. *The New York State Emergency Medical Services System Plan.* Albany, New York State Emergency Medical Services Council, November 1984.

3. Eisenberg M, Bergner L, Hallstrom A: Paramedic programs and out-of-hospital cardiac arrest: I. factors associated with successful resuscitation. *Am J Public Health* 1979; 69:30–38.

4. West JG, Trunkey DD, Lim RC: Systems of trauma care. A study of two counties. *Arch Surg* 1979; 114:455–460.

5. Li TC, Phillips MC, Shaw L, Cook EF, Natanson C, Goldman L: On-site physician staffing in a community hospital intensive care unit. *JAMA* 1984; 252: 2023–2027.

6. *Accidental Death and Disability: The Neglected Disease of Modern Society.* Washington, DC, National Academy of Sciences/National Research Council, 1966.

7. Lowe RJ, Baker RJ: Organization and function of trauma care units. *J Trauma* 1973; 13:285–291.

8. Crowley RA: A study of shock and trauma in man utilizing the resources of a clinical shock trauma unit. *Md State Med J* 1967; 16:65–66.

9. Boyd DR: Trauma—a controllable disease in the 1980's. *J Trauma* 1980; 20:14–24.

10. *Ambulance Services Statistics in New York State.* Albany, Bureau of Emergency Health Services, New York State Department of Health, June 29, 1983. [Memorandum]

11. *Guidelines for Emergency Medical Services System Categorization Process.* Albany, New York State Emergency Medical Services Council, 1981.

12. Panel: Current status of emergency medical services. *J Trauma* 1981; 21: 196–203.

13. *Emergency Department Standards.* Maspeth, Standards Committee of the 911 Receiving Hospital Committee/New York City EMS Staff. 4th ed. March 1983.

14. Baker RJ: Emergency medical services categorization and regionalization: an accomplished fact. *Arch Surg* 1978; 113:1133–1134.

The need for routine sonography prior to late abortion

In 1973, the landmark decision of the United States Supreme Court in *Roe v Wade* established a woman's right to abortion.[1] Fetal viability—the age at which the fetus is "potentially able to live outside the mother's womb, albeit with artificial aid"—was determined to be a critical point in gestation. Prior to viability outside the womb, the pregnant woman had a fundamental right to abortion, with the state able to intervene only to protect her health. However, after viability outside the womb was established, the state's interest in protection of fetal life became compelling, and the proscription of abortion under most circumstances was permissible. The Court placed viability "usually at about seven months (28 weeks) but may occur earlier, even at 24 weeks."[2] In the subsequent decision of *Coluatti v Franklin*, the Court decided that "viability is reached when, in the judgment of the attending physician on the particular facts of the case before him, there is a reasonable likelihood of the fetus' sustained survival outside the womb, with or without artificial support."[3] Recently, the Supreme Court has upheld the previous decision concerning a woman's right to abortion and has decided that not all second-trimester abortions need be performed in hospitals.[4,5] The purpose of this communication is to encourage the routine use of sonographic assessment of gestational age prior to elective late abortions for both maternal and fetal interests.

Viability, when used to describe a fetus in utero, is a presumptive and predictive term. In the absence of clear criteria for determining nonviability, one presumes that a fetus in utero will be viable *ex utero.* A fetus is "previable" when its departure from the mother will certainly result in its death.[6] Therefore, correlations with survival outside the womb must be examined in order to assess in utero viability.

Due to the lack of standardization of gestational age assessment in extremely premature newborn infants, survival is usually expressed as a function of birth weight. Although survival of a 450-g infant has been documented,[7] it is most unusual for an infant with a birth weight of less than 600 g to live. However, a healthy neonate with a birth weight greater than 600 g has a reasonable chance of survival if a modern intensive care unit is available. From 1975 to 1978, for example, the University of Colorado Medical Center reported a 40% survival rate for neonates that weighed between 600 g and 700 g.[8] At Yale-New Haven Medical Center there is a 30% survival rate for neonates that weighed between 600 g and 800 g. According to intrauterine and extrauterine growth curves, this birth weight best corresponds to a gestational age of 23 to 24 weeks.[9-11] However, the infants who survive at very low birth weights likely represent more mature fetuses with intrauterine growth retardation.

Dillon and Egan[12] have recently reported on the survival of 62 infants with gestational ages of 24 to 27 weeks, determined before birth, who were managed from 1977 to 1980 at Children's Hospital in Buffalo. Survival at the time of discharge from the hospital ranged from 36% at 24 weeks of gestation to 76% at 27 weeks of gestation. However, since the birth weights of the four survivors with a gestational age of 24 weeks were larger than expected, they were probably one or two weeks older. During a comparable period at Yale-New Haven Medical Center, the youngest survivor was an infant of 25 weeks gestational age.

Until recently the antenatal determination of viability outside the womb has been dependent on menstrual history and clinical assessment of fetal size. For early abortions, these are sufficient variables to exclude a viable fetus. However, for late abortions, the inherent inaccuracies of menstrual history and clinical sizing do not preclude the possibility of confusing a fetus that is not yet viable with a potentially viable fetus. For example, in upstate New York during a two and a half year period before the availability of antenatal ultrasound, 27 live fetuses resulted from saline abortions. Of these, 21 had birth weights greater than 600 g, 12 greater than 800 g, 8 greater than 1,000 g, and 2 greater than 1,500 g.[13] Undoubtedly, there were also fetuses of increased weight that were dead on abortion during this period. Furthermore, the possibility of deliberate underestimation of gestational age by the obstetrician or the woman requesting the abortion cannot be discounted.

However, since obstetric ultrasound is now readily available, determination of biparietal diameter prior to late second trimester abortions seems warranted. Determining fetal age with sonography is more accurate than clinical estimation, as a biparietal diameter measurement between 18 and 24 menstrual weeks is reliable within 10 days using 95% confidence limits.[14,15] Ultrasonic determinations of biparietal diameter would also provide a more objective assessment of gestational age, since menstrual history or clinical estimation of uterine size is subject to a patient's recollection and a clinical impression—both sources of bias. In addition to biparietal diameter, other sonographic measurements, including femur length,[16,17] head perimeter,[18] and orbital measurements,[19] used separately or in combination, may be valuable in assessing gestational age.

Inaccuracies in clinical gestational age assessment are not limited to the antenatal determination of fetal viability. Iatrogenic prematurity due to elective delivery of a fetus thought to be at term has been identified as a major health care problem.[20] Menstrual history and clinical estimators of gestational age are not sufficient to define fetal maturity, and at least one corroborative ultrasound assessment is necessary.[21] Although the usefulness of combined clinical observations in gestational age assessment has recently been stressed,[22] they are of limited value because of inherent error. To determine fetal maturity or fetal viability more accurately, sonographic assessment of gestational age is necessary.

Maternal interests are also served by sonographic assessment of fetal age. Respect for both maternal autonomy and current law requires that accurate information about the gestational age of the fetus be available, so that an informed decision regarding termination of pregnancy is possible. A pregnancy in a uterus enlarged by myomas may be terminated at an early stage, whereas a more advanced pregnancy, otherwise underestimated by clinical assessment, may not. For maximum efficiency and safety, the method of termination should be based upon accurate assessment of the duration of the pregnancy. Inaccuracies in assessing gestational age are related to increased maternal morbidity and mortality from induced abortion—an otherwise relatively safe procedure.[23] Again, it is emphasized that even in experienced hands, clinical assessment of gestational age may be inaccurate.[13] The recent Supreme Court decision that all second trimester abortions need not be performed in hospitals accentuates the responsibility of the obstetrician to identify late gestations, for which inpatient termination may be medically preferable.

We feel that the potential benefits of accurate assessment of gestational age warrant the cost and inconvenience of sonographic evaluation prior to late abortion. Sonographic evaluation between 12 and 18 weeks would be helpful in choosing a method and place of termination, while sonographic evaluation after 18 weeks would be valuable in determining the viability of a fetus.

Based on available data it is not possible to define an exact cutoff for viability outside the womb. Although sonographic estimation of gestational age is more accurate in predicting viability than are clinical variables, cases in which viability is uncertain would not be eliminated—merely better defined and perhaps reduced. In these borderline cases the sonographic estimation of fetal weight[24] may be of value. At the present time, elective termination of pregnancy is permitted at The Mount Sinai Medical Center and Yale-New Haven Hospital, provided the sonographic and clinical assessment of gestational age is less than 24 weeks. Based on biometric studies, a biparietal diameter of 57 mm to 61 mm[14,15] and a femur length of 42 mm to 44 mm[17,25] correspond to a mean gestational age of 24 weeks. If future advances in neonatal medicine enable survival of more immature neonates outside of the womb, a reevaluation of this policy would be in order.

Acknowledgment. Robert J. Levine, MD and Margaret Farley, PhD gave their support and encouragement in the preparation of this manuscript.

FRANK A. CHERVENAK, MD
SALLY FAITH DORFMAN, MD
CHARLES BOWERS, MD
RICHARD L. BERKOWITZ, MD
Department of Obstetrics, Gynecology,
and Reproductive Science
The Mount Sinai Medical Center
One Gustave L. Levy Place
New York, NY 10029

ANGELA R. HOLDER, LLM
Department of Medical Legal Affairs

RICHARD A. EHRENKRANZ, MD
Department of Pediatrics

REGINA FURLONG, ASCW
Department of Obstetrical Social Work

JOHN C. HOBBINS, MD
Department of Obstetrics and Gynecology
Yale-New Haven Medical Center
New Haven, CT

1. *Roe v Wade*, 410 US 113, 1973.
2. *Roe v Wade*, 410 US 160, 1973.
3. *Coluatti v Franklin*, 439 US 379, 1979.
4. *City of Akron v Akron Center for Reproductive Health, Inc.*
5. *Planned Parenthood Association of Kansas City, Missouri, Inc v Ashcroft.*
6. Levine RJ: Viability and death of the human fetus: biologic definitions. AFCR Symposium. *Clin Res* 1975; 23:211–216.
7. Lelek K. Limanowski J, Hager-Malecka B: A three-month-old infant with birth weight 450 g. *Pediatr Pol* 1973; 48:221.
8. Bowes Jr, WA, Simmons MA: Improved outcome in very low-birth weight infants. *Am J Obstet Gynecol* 1980; 136:1080–1081.
9. Brenner WE, Edelman DA, Hendricks CH: A standard of fetal growth for the United States of America. *Am J Obstet Gynecol* 1976; 126:555–564.
10. Williams RL, Creasy RK, Cunningham GC, Hawes WE, Norris FD, Tashiro M: Fetal growth and perinatal viability in California. *Obstet Gynecol* 1982; 59: 624–632.
11. Ott WJ, Doyle S: Normal ultrasonic fetal weight curve. *Obstet Gynecol* 1982; 59:603–606.
12. Dillon WP, Egan EA: Aggressive obstetric management in late second trimester deliveries. *Obstet Gynecol* 1981; 58:685–690.
13. Stroh G, Hinman AR: Reported live births following induced abortion: two and one-half years' experience in upstate New York. *Am J Obstet Gynecol* 1976; 126:83–90.
14. Jeanty P, Cousaert E, Hobbins JC, Tack B, Bracken M, Cantraine F: A longitudinal study of fetal head biometry. *Am J Perinatol* 1984; 1:118–128.
15. Shepard M, Filly RA: A standardized plane for biparietal diameter measurement. *J Ultrasound Med* 1982; 1:145.
16. Jeanty P, Kirkpatrick C, Dramaix-Wilmet M, Struyven J: Ultrasonic evaluation of fetal limb growth. *Radiology* 1981; 140:165–168.
17. Hadlock FP, Harrist RB, Deter RL, Park SK: Fetal femur length as a predictor of menstrual age: sonographically measured. *AJR* 1982; 138:875–878.
18. Hadlock FP, Deter RL, Harrist RB, Park SK: Fetal head circumference: relation to menstrual age. *AJR* 1982; 138:649–653.
19. Mayden KL, Tortora M, Berkowitz RL, Bracken M, Hobbins JC: Orbital diameters: a new parameter for prenatal diagnosis and dating. *Am J Obstet Gynecol* 1982; 144:289–297.
20. Maisels MJ, Rees R, Marks K. Friedman Z: Elective delivery of the term fetus: an obstetrical hazard. *JAMA* 1977; 238:2036–2039.
21. Chervenak FA, Shamsi HH: Is amniocentesis necessary before elective, repeat cesarean section? *Obstet Gynecol* 1982; 60:305–308.
22. Anderson HF, Johnson TRB, Flora JD, Barclay ML: Gestational age assessment. II. Prediction from combined clinical observations. *Am J Obstet Gynecol* 1981; 140:770–774.
23. Cates Jr W, Grimes DA: Deaths from second trimester abortion by dilatation and evaluation: causes, prevention, facilities. *Obstet Gynecol* 1981; 58:401–408.
24. Shepard MJ, Richards VA, Berkowitz RL, Warsof SL, Hobbins JC: An evaluation of two equations for predicting fetal weight by ultrasound. *Am J Obstet Gynecol* 1982; 142:47–54.
25. Jeanty P: Fetal limb biometry. *Radiology* 1983; 147:601–602.

The new "regulators" of health care

In the past decade attempts by corporations to control rapidly escalating employee health costs have centered on such programs as wellness seminars, hypertensive screening, employee health education, and generic drug promotion. But in more recent years, corporations have taken an increasing interest in peer review programs, reduction in length of hospital stay, outpatient surgery, second opinions, and hospital and doctor profile disclosure through purchase of information. Big business, which does not believe that medicine polices itself, has an equal distrust of fiscal intermediaries. As industry develops new health programs, some of which necessitate the employee paying first dollar coverage (a deductible), there is interest in both concurrent and pre-admission review. As employees become increasingly involved in their health programs, more companies are providing their workers with information on the least expensive hospitals and on which procedures can be done on an outpatient basis. Similarly, many companies have contracted with selected providers to make their services available at a reduced cost—the so-called preferred provider organization.

Industry is convinced that escalating medical care budgets are not being spent judiciously, and the statistics they are releasing lend credence to these claims. Chrysler Corporation, for example, states that $600 of the price of every new product is earmarked for employee health. Dupont reports spending $150 million a year on their health benefits. General Motors, which employs about half a million people, expected to pay $2.2 billion for health care in 1983, or about $4,400 per employee.[1] International Harvester provided $90 million in health benefits in 1983, and an increase was predicted for 1984. John Deere spends $100 million a year on health care and states that inappropriate use of hospitals is the major cause of these high costs. In 1973 Honeywell's medical costs were 2.6% of total payroll costs; by 1981 they had doubled to 5% of payroll. The $124 million that Honeywell spent on health care in 1983 is an impressive figure, but if costs continue to rise at the characteristic rate of 19% per year, Honeywell will spend three-quarters of a billion dollars on health care by 1993.[1] These grim statistics need to be countered by pointing out that business, itself, must accept the major responsibility for this situation. Most benefits managers will admit that they have "given away the store." The benefits they have offered employees encourage consumption of health care services without regard to cost.

The formation of health care coalitions made up of the purchasers of health care services and the employers in the community appears to be business's answer to the health care problems.[2] Physicians and hospitals are not included in most of the coalitions at this time, although, ironically, the American Medical Association has been a leader in the establishment of medicine and business committees nationwide. Nearly all of the coalitions have instituted systems to review the use of health services, including preadmission certification for elective procedures, certification of the expected length of stay in the hospital, and concurrent review. Some systems require a second opinion on certain elective surgical procedures.

The coalitions are very interested in the development of data bases in their respective communities. One cannot rebut their philosophy that to control costs there must be a clear understanding of where the money is going. Pooling of claims data can tell business how many days they paid for per 1,000 employees and dependents, the average length of stay per diagnosis, and how many admissions there were per diagnosis. Depending on the sophistication of the data base, they can discover the average cost for a given diagnosis for each hospital in the region, and sometimes for each physician. The data will help businesses redesign their insurance plans: many employers are backing off from first-dollar 100% insurance coverage and are instituting deductibles and copay plans that cause the employee to use physician and hospital services sparingly. Statistics will also help identify procedures that can be done safely on an outpatient basis. With the help of this data benefits managers can plan alternative delivery methods. Once the hospitals and providers that offer cost-efficient services are identified, contracts for health maintenance organizations and preferred provider organizations will follow. Physicians are properly concerned that such practices have the potential for compromising quality, as industry concentrates on cost containment. In short, the "buyers guide" that industry proposes to provide its employees may not fulfill the consumer's expectations.

An alternate form of corporate involvement in the provision of medical services is in place for 2,600 salaried employees of Zenith's seven Chicago plants.[3] Zenith has employed a utilization review specialist who must be contacted before employees can enter the hospital. He has data on the average cost per day for Chicago's hospitals, the average length of stay per procedure, and the amount the employee himself can expect to pay. If the employee is embarrassed to query the physician on cost-cutting procedures, the utilization specialist contacts the physician. Under the Zenith plan, 100% of the costs of second opinions, testing, and skilled nursing care are covered. During the first two months of this program Zenith saved about $4,500 per week.

Overzealous business coalitions whose only concern is the bottom line may emerge. Are they banking on litigation to take care of quality? It would seem that they have paraphrased the old saw "he who pays the fiddler calls the tune" to read "he who has access to the data calls the tune."

JOHN T. PRIOR, MD
Medical Director
ESPRO of Central New York
Community General Hospital
Broad Road
Syracuse, NY 13215

---

1. Lefton D: Business aims to trim health care costs. *Am Med News*, November 18, 1983, p 1.
2. MDs seek bigger role in coalition. *Am Med News*, April 2, 1982, p 1.
3. Lefton D: Zenith plan taps Blues' cost data. *Am Med News*, December 2, 1983, p 1.

LETTERS TO THE EDITOR

This section is designed to offer rapid publication (usually within 1-3 months) of new clinical, epidemiologic, and laboratory observations, as well as of comments on articles published in the Journal *or on any topic related to the role of the physician. Letters should be typed double-spaced and should include the professional affiliation, highest academic degree, address, and signature of each author. Preference is given to letters not exceeding 450 words. All letters are personally reviewed and acknowledged by the Editor. Address correspondence to Editor,* New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042.*

Offshore medical schools

TO THE EDITOR: In the July 1984 issue of the *New York State Journal of Medicine*, titled "The Offshore Medical Schools," an article from *The Lancet* by F. J. Woodroffe (1982; 2:546-547) is quoted. The article is obsolete, and there were several areas where wrong inferences were made and erroneous conclusions drawn. For example, the price of housing in St. Vincent has been alleviated under the building of three dormitories and a long-term lease on an apartment complex.

The issue of cultural deprivation is best answered by quoting from a personal communication of a student who recently transferred to an American medical school after two years at St. George's:

> St. George's gave me the opportunity to begin my medical education and my dream of becoming a doctor, in addition to the personal growth of strength, determination and flexibility. I have gained a great deal from this very unique life experience that others cannot even imagine. It has taught me good medicine as well as perseverence and strength to meet any challenge in life. I will be forever grateful for all that it has given me.

As for costs, you can always be sure that the high cost of a St. George's education will be quoted; however, the fact that private medical schools in the United States are often twice as costly and in some cases three times as much is conveniently omitted.

I do not have the time to comment fully on the St. Vincent experience that the writer knows so much about, but much of it is quite wrong. He ought to do what a British consultant in medicine did: he came and taught Physical Diagnosis for four weeks and then wrote about his experience from firsthand information and not hearsay:

> The University seems to have gained tremendous momentum under your guidance and with your future plans, the hospital picture and teaching arrangements will become very strong indeed. I also understand the system more clearly which I think will influence the way I think and teach the chaps in the U.K.

We are a school for the rejected, and there is a lot of talent in that group. We offer them their last hope, and what we offer is an opportunity to prove that the student has what it takes, not a guaranteed degree. Maybe that is why 30% to 35% of our students transfer into American medical schools. We are dedicated to high standards and quality education.

A few years ago, an associate editor of the *New York State Journal of Medicine* wrote an article about the neglected and unqualified students in the Caribbean.* He was never in the Caribbean himself, and he quoted a physician who also never saw St. George's. So much for objectivity.

MORRIS ALPERT, MD
Dean of Faculty
St. George's University
School of Medicine
Kingston Medical College
PO Box 885
St. Vincent, West Indies

* *The author may be referring to the article "Resurrect Flexner" by Julius E. Stolfi,* MD (NY State J Med *1979; 79: 1679*) *—Editor*

In reply: The alleged obsolescence of the comments made in *The Lancet* in 1982[1] was not apparent to the ministers of health of the Caribbean countries who met at a two-day conference in Dominica in August 1984.[2] They issued a declaration concerning the operations of offshore medical schools. In this declaration they emphasized the inadequacies of these schools—the lack of proper training facilities, the use of itinerant lecturers, and the unstable nature of these schools because they have been established by entrepreneurs who will abandon them as soon as they are not viable financially.

Participants in the conference recommended that the activities of offshore schools be limited to preclinical training, that the establishment of new schools be viewed with extreme caution and, significantly, that Caribbean nationals themselves be discouraged from attending these schools. Further, the health ministers recommended that scholarships offered to Caribbean nationals by offshore schools be used at the University of the West Indies or another school chosen by the government, rather than at the sponsoring school.

The recent declaration of the Caribbean ministers of health confirms that the comments made in *The Lancet* in 1982 are not dated.

PASCAL JAMES IMPERATO, MD
Deputy Editor

1. Woodroffe FJ: Offshore medical schools. *Lancet* 1982; 2:546-547.
2. Bartholomew C: Offshore medical schools. *Lancet* 1984; 2:580.

TO THE EDITOR: I read with great interest the July issue of the *Journal:* "The Offshore Medical Schools—Current Issues and Controversies." I was surprised by the moderate tone used by the authors of the papers published. The problem is distasteful and threatens the quality of medical care, especially in our own state. Of course, those not affected have little or no say in the matter. The sick and injured have no way of knowing the credentials of those involved in their care in New York hospitals.

The opening overview nicely summarized the present state of affairs, but the sections "The Current Situation" and "Present and Future Problems" omitted a discussion of the poor training in basic and clinical medicine received by those students who are being allowed to take the licensing examinations in New York State. California, for example, has excluded them. Apparently, the Regents of New York State were influenced by the threat of litigation, and that was tragic.

A physician's problem solving ability

cannot be adequately developed without the firm foundation provided by basic science training. Without anatomy, histology, biochemistry, pharmacology, physiology, and microbiology, medical graduates would be mere technicians or tradesmen and not professionals trained to understand and apply the basic science information so necessary to the practice of clinical medicine.

Monahan describes well the categories of individuals who, under the New York State education laws, can practice medicine without a license. They are students enrolled in "medical schools that meet standards acceptable to the New York State Education Department and who are performing clinical clerkships in New York State Hospitals." Most are from medical schools registered by the department or accredited by the Liaison Committee on Medical Education. He admits that barring students from unregistered and unaccredited institutions was ruled out on "legal and practical" grounds.

In 1981, at one of the hearings held by the Board of Regents, I testified against opening the doors of licensing in New York State for students who, in my opinion, were poorly prepared by the offshore schools. In editorials[1,2] I expressed a strong objection to the Regents' permissive regulations. The Regents did change the regulations later by restricting students from the schools in question to clerkships in teaching hospitals with approved residency training programs. Many of the hospitals so classified are only "paper" affiliations, and the protection that this affords patients is questionable.

The bottom line is quality medical care and, those exposed to care rendered by students from entrepreneurial schools is at best suspect. Passing an examination is not proper protection for patients.

We must admit to the practice of medicine in New York State only those students enrolled in schools accredited by the Liaison Committee on Medical Education or an analogous body outside of the United States. Only those so qualified should be allowed to sit for the licensing examination. Flexner's committee essentially established that in 1910, and it is important to maintain high standards in 1984. If our state regulatory bodies and the medical profession would support these requirements, the present crop of diploma mills would be eliminated.

The Board of Regents should act for the common good without surrendering to the fear of litigation. Our hospitals, medical schools, state societies, and county societies can help by supporting this stance and by carefully monitoring the credentials of students applying for admission, as well as candidates for membership and residency training programs.

Drs Pierson and Riddle properly evaluated the present situation. Their presentations and Woodroffe's article published in *The Lancet* (1982; 2: 546–547) should be required reading for all Regents.

Having received all of my post-graduate training in Kings County Hospital in Brooklyn, I sympathize with Dr Boufford's concerns, but I disagree with her statement that the municipal hospital system is not in the medical education business. In medicine during the postgraduate period it is as impossible to separate service from education as it is to separate experienced teachers from continuous life-long learning. Teaching is educational for those who teach. Her statement in the last paragraph of the paper is a paradox. How can the Health and Hospitals Corporation "support a health personnel policy that is committed to identifying and developing a cadre of excellent physicians regardless of their educational origins?" I submit that their educational origins determine to a great extent excellence. Qualified institutions for the training of students are plentiful, and there is no valid reason for allowing students from unqualified (non-registered and non-accredited) schools to practice on unknowing patients. The increased output from properly accredited schools in the United States makes it more likely for good programs in all approved residencies to recruit graduates. The Health and Hospitals Corporation would do well to improve its training programs to meet the competition in this country.

JULIUS E. STOLFI, MD
10590 Lindberry Drive
Boynton Beach, FL 33436

1. Stolfi JE: Medical news—good and bad. *NY State J Med* 1981; 81:14–15
2. Stolfi JE: Why, Regents, why? *NY State J Med* 1981; 81:1176–1177.

Dr Stolfi served as Associate Editor of the Journal *from 1973 to 1982.*

Effects of present and past occupational exposures

TO THE EDITOR: The importance of environmental and occupational factors in the causation of disease is now recognized. Moreover, since the work environment can be quite complex, workers are at risk of developing multiple occupational diseases. This is illustrated by the following case report.

Case Report: A 50-year-old iron worker sought medical attention because of concern about excessive lead exposure. One month prior to the examination he was engaged in bridge repair work—cutting and burning through lead-painted metal structures using an oxyacetylene-propane torch. Despite frequent use of protective respiratory equipment (positive pressure respirator), he had inhaled a large amount of lead oxide fumes.[1]

He complained of nonspecific gastrointestinal and neurologic symptoms that had lasted for a few weeks. The symptoms included nausea, anorexia, vomiting, and epigastric discomfort; fatigue, headache, and sleep disturbances were the main neurologic symptoms. Auscultation of the chest revealed scattered dry rales bilaterally along the middle axillary lines. Physical examination was otherwise noncontributory. Laboratory findings included an elevated blood lead level of 54 μg/100 mL. (A blood lead level of 15.3 ± .32 μg/100 mL [mean ± standard error of the mean] was reported in a recent United States National Health and Nutrition Examination Survey for the age group 45 to 54 years; the Occupational Safety and Health Administration's blood lead standard is 40 μg/100 mL.) A chest radiograph showed bilateral thickening of the pleura.

In reviewing the occupational history of this patient, it was found that he had been an iron worker for the past 25 years. During the period from 1952 to 1956, he had served in the Navy as a firefighter on ships and was frequently in shipyards while maintenance work was being performed.

Discussion: Although the two medical problems diagnosed in this case, elevated blood lead level and bilateral pleural thickening, represent effects of two "classical" occupational exposures, the report illustrates the importance of giving attention to a worker's total work experience or lifetime occupational history, even when evaluating a single acute occupational disease. The acute problem is often easily diagnosed because of the temporal relationship between exposure and disease; however, the effects of some occupational exposures become clinically evident only after a latency period of 20 years or more.

In this case, a detailed history revealed the most plausible explanation for the bilateral pleural thickening: work in shipyards from 1952 to 1956. This is in accordance with the well-known occurrence of asbestos-associated diseases in former shipyard workers, many of whom did not use asbestos themselves but were indirectly exposed ("bystander's disease").[2]

The short-term effect of lead poisoning is readily handled by discontinuation of lead exposure or chelation therapy, if necessary. The discovery of long-term effects of asbestos exposure should also alert the physician to inform the patient about the synergism between asbestos exposure and cigarette smoking; in addition to minimizing exposure to asbestos, discontinuation of cigarette smoking is a key factor in reducing the extraordinarily high lung cancer risk that individuals exposed to asbestos experience.[3] The detailed occupational history established the cause and effect relationship between previous work in shipyards and the pleural disease, with obvious implications for future medical surveillance.

We have recently encountered a case of malignant pleural mesothelioma in an iron worker from the same local union of which this patient is a member; he gave a history of working as an iron worker in a shipyard during the 1940s. Shipyard work was common among iron workers during this period, and physicians should be aware that asbestos-related diseases may occur with previously unanticipated high frequencies in this occupational group.

ALF FISCHBEIN, MD
Assistant Professor
Environmental Sciences Laboratory
The Mount Sinai Medical Center
1 Gustave L. Levy Place
New York, NY 10029

---

1. Fischbein A, Leeds M, Solomon SJ: Lead exposure among iron workers in New York City. *NY State J Med* 1984; 84:445-448.
2. Selikoff IJ, Lilis R, Nicholson WJ: Asbestos disease in United States shipyards. *Ann NY Acad Sci* 1979; 330:295-311.
3. Hammond EC, Selikoff IJ, Seidman H: Asbestos exposure, cigarette smoking and death rates. *Ann NY Acad Sci* 1979; 330:473-490.

Fiscal and moral considerations of chronic brain syndrome

TO THE EDITOR: Every two months, as required by state law, I visit several inhabitants of local nursing homes. One of these, in particular, stands out in my mind. Formerly a bright, cheerful, and witty lady, she has for years led a totally non-sentient existence, not recognizing even her children and requiring little medical attention but total nursing care and help with basic bodily functions.

What sets her apart in my mind is the appearance of the nursing home in which she resides—long corridors lined wall to wall with aged, demented patients in wheelchairs, seemingly unchanged year by year, unable to think, often unable to feel or to speak, and no longer a part of intelligent life as we know it. I invariably leave the place with a feeling of relief mixed with vague foreboding. This and similar experiences have led me to consider the complex role of the patient with chronic brain syndrome in our society.

As national expenditures for health care last year reached the staggering total of $335 billion,[1] we are told by opinion makers that costs have risen too high too fast. Emphasis has been placed on the high cost of technology. Its use "to prolong dying" is being questioned on ethical as well as economic grounds. It has been pointed out that we spend 28% of Medicare funds on the 6% of patients who die each year.[1] Courts have ruled that treatment may be discontinued if there is "no reasonable possibility of a patient returning to a cognitive, sapient state." [1]

However, any primary care physician that deals with patients in nursing homes is aware of the equally vexing and serious problem of patients with advanced chronic brain syndrome. These make up an estimated 50% to 80% of the nursing home population.[2] They usually require a great deal of nursing and custodial care, but relatively little physician care and no expensive technology. They continue to exist for many years, often becoming mute, mindless, and incontinent—a great strain on their families and a clear-cut burden on the tax payer. Yet, in my opinion, this problem has largely escaped serious public discussion, possibly because no easy suggestions, such as the "right to die," are available.

In 1983 one out of nine Americans was age 85 and over.[3] By the year 2030 this number is estimated to rise to one in 5.[3] By the year 2000, it is estimated that 43% of the 65 and over age group will be age 75 and above, requiring an estimated 66% increase in nursing home beds, assuming present standards of care.[2]

With the cooperation of Monroe County administrative personnel, I have attempted to obtain some figures to help put the local situation into perspective. The population of Monroe County is approximately 705,000, with a total county budget of about $412 million in 1984. The county Medicaid budget stands at approximately $41 million, which pays only a quarter of incurred cost, the rest coming from state and federal funds. Although I cannot obtain definite figures, I believe that the total Medicaid-eligible population in Monroe County is about 41,000, of which a relatively stable group—2,800 to 3,000—reside in nursing homes (approximately 80% in skilled nursing facilities and 20% in health related facilities). Nursing home turnover stays level at about 10% to 15% per year.

Of this $41 million budget, about $12.5 million goes to skilled nursing facilities, and $2.9 million to health related facilities, for an approximate total of 37.5%. Moreover, these figures only include straight service costs and not administrative overhead. Thus, an estimated 7.3% of the eligible population, if my calculations are correct, is responsible for incurring anywhere from 37.5% to over 45% (depending on calculations of overhead) of available Medicaid expenditures.

Such allocation of public funds raises complex questions for the makers of public policy, both for now and, with the inevitable graying of the population, in the future. Surely the aged deserve the best care that society is capable of providing, but so do children, pregnant adolescents, and those with chronic diseases. Furthermore, when we attempt to alter the definition of life from a quantitative one of absence of death to a more qualitative one, we are faced with numerous, subjective uncertainties. It may at times be possible, with consent of family, to delay antibiotic treatment of pneumonia in the severely demented, but what is to be done about the same patients who do not get pneumonia?

At this time I am unable to offer any solutions and I have no strong opinions to present. I merely hope, with the help of a few statistics, to present a difficult subject for consideration and for more informed public discussion.

GERALD L. GLASER, MD
Department of Medicine
The Genesee Hospital
224 Alexander St.
Rochester, NY 14607

---

1. Lamm RD: *The New Republic*. August 27, 1984, p 20.
2. Wershow H: In *Controversial Issues in Gerontology*. New York, Springer Publishing Co, 1981, p 177.
3. Tibbitts SJ: Future belongs to new multihealth corporations; hospitals should join now. *Modern Health Care*, September 1984, p 208.

Seroepidemiology of *Legionella pneumophila* serogroup 1 in healthy residents of New York City

IQBAL A. POSHNI, PHD, STEPHEN J. MILLIAN, PHD

ABSTRACT. **During a three-year period from 1979 to 1981, 4,320 serum specimens from asymptomatic persons living in New York City were examined by the indirect fluorescent antibody technique for antibodies against serogroup 1 of *Legionella pneumophila*. An overall prevalence rate of 23.6% was observed among the 4,320 persons tested, using 1:64 as the cutoff titer. The prevalence in the population tested declined from a high of 31.8% in 1979 to a low of 16.7% in 1981. There were no significant differences associated with age or sex. In colder months—December, January, and February—significantly higher prevalence rates (average rate 32.5%) prevailed than in the warmer months (average rate 17.5% for June, July and August). Prevalence rates ranging from 5% to 41.7% persisted throughout the year, confirming the hyperendemicity of legionellosis in New York City. Serologic cross reactivity of *Legionella* species is discussed.**

(NY State J Med 1985; 85:10–14)

INTRODUCTION

A sudden outbreak of legionellosis in the garment district of New York City during the fall of 1978[1] prompted us to initiate an extensive seroepidemiologic survey for antibody against *Legionella pneumophila.* Several investigators[2,3] used the indirect fluorescent antibody technique (IFA) and microagglutination (MA) methods to test apparently healthy populations for the prevalence of *Legionella pneumophila* antibody. These researchers failed to detect variations in prevalence rates between the sexes or among different age groups. On the other hand, Helms et al[4] found that among individuals 40 years of age or older, higher titers were observed more frequently in men than in women (2.7:1). In another study,[5] significantly higher titers were observed in the 50 to 59 year age group than in the 80 to 89 year group. The present study was implemented to determine the influence of age, sex, and season on the prevalence of *L pneumophila* in New York City.

PATIENTS AND METHODS

Indirect fluorescent antibody testing (IFA) was initially performed according to the procedure outlined by McDade et al[6] and subsequently by Wilkinson et al.[7] An IFA titer of ≥1:64 was considered significant for this seroepidemiologic study. Statistical significance was calculated using the chi-square test at a 5% level of significance.

Antigens. The Philadelphia strain of *Legionella pneumophila* was obtained from the Biological Products Division of the Centers for Disease Control in Atlanta, GA. Antigen from heat-killed organisms grown on Feeley Gorman medium was used during the 1979 and 1980 part of the study. From January 1981 on, heat-killed organisms grown on Charcoal Yeast Extract medium were used as antigens. To our knowledge, there is no published report of a difference in serologic sensitivity or specificity, or both of the legionella organisms grown on the two media mentioned above.

Preparation of the slides. Twelve-welled, teflon-coated slides obtained from Cell-Line Associated Inc, Neufield, NJ, were stored at −21°C to preserve shelf life. Just before use these slides were removed from the freezer and held at room temperature for 10 minutes. Each slide was carefully wiped, and a drop of the mixed test antigen suspension was placed in each well and spread evenly. The slide was air-dried for 15 minutes and then fixed in acetone for 10 minutes.

Initially, two-fold serial dilutions of the test and control sera were made using 10% normal yolk sac suspension. After May 1979, a 3% suspension was used following a recommendation from the Centers for Disease Control.

Fluorescein isothiocyanate-labelled rabbit antihuman globulin, purchased from Sylvana (Cat. # 112-3120), was used throughout the study at titers from 1:100 to 1:400 depending upon the lots of antigens used.

Observations were made using a Leitz-Ortholux binocular microscope with a periplan GF 10× eye piece and a 54× oil immersion fluorite lens. The light source was a mercury lamp, HBO W LII, incorporated into an incident light fluorescence illuminator system (Ploem). The excitation filter was a 2× interference blue filter, KP 490 (FITC), used in conjunction with a K 510 barrier filter.

The positive control sera were supplied by the Centers for Disease Control and were precalibrated at a dilution of 1:256 to 1:512. Lot numbers 78-006K, 79-0103K, and 80-0101 were used throughout this investigation to standardize in-house control sera.

Each month, sera from 10 men and 10 women, for each specified age group (<20, 20–29, 30–39, 40–49, 50–59, and ≥60 years) were sequentially selected from a pool of rapid plasmin reagin negative specimens. These blood samples were submitted to the Syphilis Serology Unit as part of preemployment, premarital, or routine physical examinations. According to the records accompanying the specimens, all subjects were healthy and were not visiting the clinic for any specific complaint. Approximately 8% of the blood specimens were obtained from healthy

---

From the Division of Immunology and Virology, Bureau of Laboratory Services, New York City Department of Health, New York.

Address correspondence to Dr Millian, Director, Immunology and Virology Division, Bureau of Laboratory Services, New York City Department of Health, Room 1273, 455 First Ave, New York, NY 10016.

women during their periodic prenatal or postnatal visits to the clinics. Since pregnant women might be considered a special group, we tested a number of prenatal specimens and found no difference between their results and those of other groups used in this study. The number of specimens in each age group was not adjusted to reflect its true ratio in the total population. We did not know the true age and sex distribution in the general population of the New York City metropolitan area. Therefore, we took equal numbers for all age groups for both men and women. There is no reason to believe that the proportion of different age groups significantly changed during any of the three years of the study. As the same number of specimens in each age group was tested in each year, it is unlikely that any slight difference in age distribution would have a significant effect on the results.

RESULTS

Sex and age. The age distribution of the specimens for the three-year period are presented in Table I. Higher prevalence rates were observed in the 20 to 29-year-old group (188/720, 26.1%) and in the 40 to 49-year-old group (193/720, 26.8%). The lowest rate, 17.4% (125/720), was observed in the less than 20-year-old group.

When the age groups were banded into less than or equal to or greater than 50 years of age classifications, and the data were evaluated by sex, significant differences were not observed. Except for the 30 to 39-year-old group, each male group was observed to have a slightly higher prevalence rate than the respective female group. Chi-square tests failed to demonstrate any statistically significant variation.

Among men the prevalence rates seem to rise and fall for alternate age groups. The lowest rate was observed for the youngest group (<20 years) and the highest for the ≥60-year-old group. Although the rate for women under 20 years was also the lowest for that sex, a pattern comparable to that seen in the men was not observed.

Seasonal differences and annual variations. Figure 1 shows the three-year average monthly prevalence rates for men and women and the combined population. In general, the male seasonal trend was similar to that of the female group. While men had higher rates than women for six months of the year (March, May, June, July, September, and November), no difference was observed for April, August, or December. For the months of January, February, and October, the female rate was found to be in excess of that of the men.

For both sexes combined the peak prevalence occurred during the winter months: January, 34.7%; February, 32.5%; and December, 30.3%. The lowest prevalence rates occurred during the summer months: June, 16.4%; July, 17.8%; and August, 18.3%. The results for each year were similar to one another—that is, higher and lower prevalence rates were observed during the winter and summer months, respectively.

Overall prevalence of legionellosis declined from a high of 31.7% in 1979 to a low of 16.9% in 1981. The yearly prevalence rates for both male and female groups declined from a high of 32% to a low of 19% for men and 15% for women. For each sex the year to year decline was statistically significant, except for the male group from 1981 to 1983. A drop in the prevalence rate for the study period was observed in all age groups; the largest decline (21.7%) was observed in the ≥60-year-old group from 1979 to 1981. The smallest decline (7.5%) occurred in the 50 to 59-year-old group.

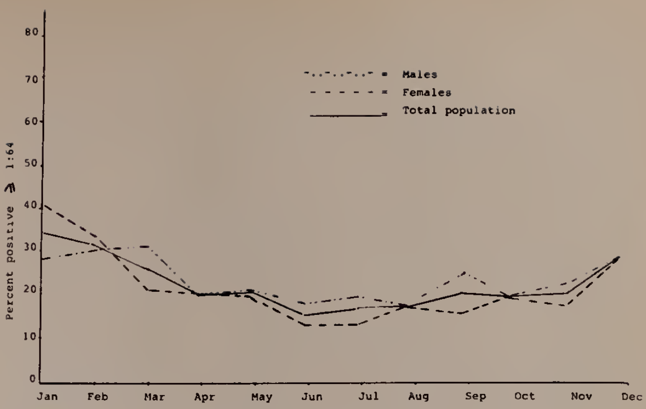


FIGURE 1. Average prevalence rates by sex and month, 1979–1981.

A year by year comparison of the prevalence rates for men and women of various age groups is presented in Figure 2. Among the women, all age groups showed a steady decline in prevalence rates from 1979 to 1981. Among the men, the 1979 rates were the highest in all age groups, but in 1980 and 1981 the differences were less marked and the relationship was reversed in the three age groups.

DISCUSSON

The objectives of this study were to determine whether or not seasonal fluctuations and year to year trends in *Legionella pneumophila* antibody prevalence occurred, and if the variations were a function of age or sex. While seroconversions in conjunction with clinical findings provide the best documentation of disease incidence, a single convalescent serum can provide valuable information once the upper level of normality has been established.[8]

Factors such as serogroup used, growth conditions, dilution of the conjugate, the inactivation procedure, method used for the preparation of the antigen, and the criteria for reading the end titers can influence the upper

TABLE I. Prevalence of Significant Antibody Titers Against *Legionella pneumophila* (Philadelphia 1) by Age and Year (1979–1981)

Year		1979		1980		1981		1979–1981	
	Age group (yr)	+/total*	%+**	+/total	%+	+/total	%+	+/total	%+
Total population	<20	59/240	24.6	35/240	14.6	31/240	12.9	125/720	17.4
	20–29	81/240	33.8	58/240	24.2	49/240	20.4	188/720	26.1
	30–39	75/240	31.3	60/240	25.0	28/240	11.7	163/720	22.6
	40–49	87/240	36.3	56/240	23.3	50/240	20.8	193/720	26.8
	50–59	68/240	28.3	58/240	24.2	50/240	20.8	176/720	24.4
	≥60	88/240	36.7	51/240	21.3	36/240	15.0	175/720	24.3
Total		458/1440	31.8	318/1440	22.1	244/1440	16.9	1020/4320	23.6

* = Number positive (≥1:64)/total population
** = Percentage positive

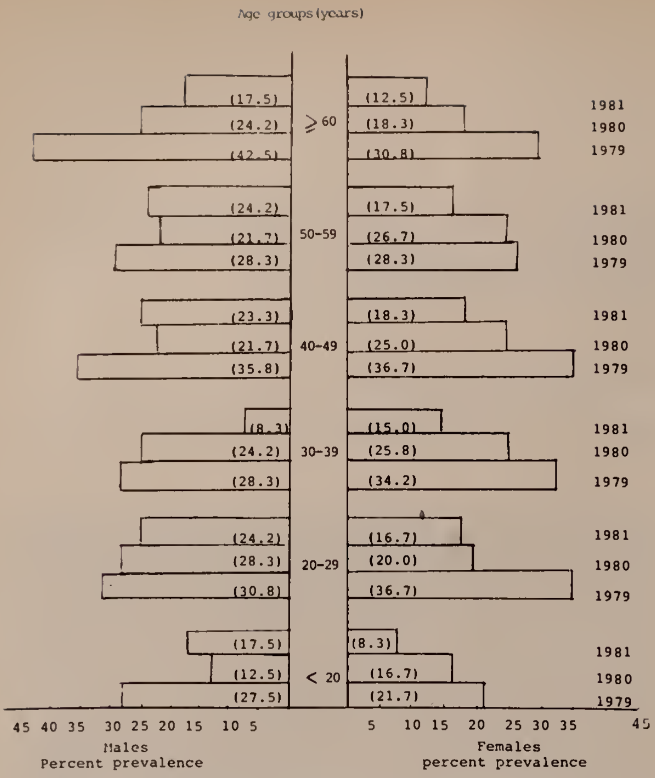


FIGURE 2. Percent prevalence rates (≥ 1:64) of selected age groups by sex and year, 1979–1981.

normality limit.[2,9] A recent outbreak of legionellosis in a community may also effect the upper normality titer. The results of this study indicate that during 1979, the year following an explosive outbreak of Legionnaires' disease in New York City, the prevalence rate was 31.8%. The following year it dropped to 22.1%, and two years later it declined still further to 16.9%. In view of these observations, it is essential that each laboratory conducting seroepidemiologic studies establish its own critical value. The variations in legionella prevalence, ranging from 0.4% to 32.0% (Table II), may be attributable to one or more of the above mentioned factors, despite the attempted standardizations by different investigators. Most of the studies in the United States and elsewhere have shown that men, particularly those aged 40 to 65 have significantly higher rates of legionella infection than women of comparable ages.[10,11] The difference has been shown to be as much as two to three fold.[4,12] On the other hand, most worldwide seroepidemiologic studies failed to reveal any age or sex related prevalence differences for *Legionella pneumophila*.[2,3,5,13-15]

Edson et al[16] have reported that age, sex, or geographic location of the persons tested had no effect on the prevalence rates of 1,200 apparently healthy Michigan residents. Yonke et al,[13] using the indirect hemagglutination test, found no significant differences in the prevalence rates of men and women, but noted a trend toward decreasing prevalence in the >50 years of age group in both sexes. It is interesting to note that Helms[4] reported that men 40 years

TABLE II. Summary of the Prevalence Rates of *Legionella pneumophila* by Various Authors

Location	Author (reference)	No. Tested	Percent Positive	Titer	Test System	Strain/ serogroup
New York City	Cordes et al (1)	198	24.7	≥1:128	IFA*	Philadelphia 1
New York City	Cordes et al (1)	199	14.6	≥1:128	IFA	Philadelphia 1
Four locations in US	Storch et al (2)	1143	1.7	≥1:64	IFA	Philadelphia 1
Iowa	Helms et al (3)	517	7.0	≥1:32	MA†	serogroup I,II,III,IV
Iowa	Helms et al (4)	517	32.0	≥1:64	IFA	Philadelphia
Memphis, TN	Smalley et al (5)	1000	5.3	≥1:16	MA	Philadelphia 1
Atlanta, GA	Farshy et al (8)	135	3.0	≥1:40	Elisa	Philadelphia 1
Atlanta, GA	Farshy et al (8)	135	5.2	≥1:40	MA	Philadelphia 1
Los Angeles, CA	Haley et al (11)	116	4.0	≥1:128	IFA	Knoxville
Vermont	Broome et al (12)	110	15.0	≥1:128	IFA	Philadelphia 1 & 2
Michigan	Yonke et al (13)	1200	16.8	≥1:16	IHA‡	Philadelphia 1
New Zealand	Bettelheim et al (15)	500	9.4	≥1:64	IFA	Philadelphia 1
Michigan	Edson et al (16)	1200	22.5	≥1:16	IHA	Detroit, Flint
New Zealand	Bettelheim et al (17)	100	18.0	≥1:64	IFA	Philadelphia 1
Nottingham, England	McRae et al (23)	2023	1.5	≥1:32	IFA	Pontiac
Benidorn, Spain	Grist el al (24)	21	19.0	≥1:128	IFA	Legionnaires' disease bacterium
Larimer, CO	Collins et al (25)	286	0.4	≥1:64	MA	serogroup II Togus 1
Larimer, CO	Collins et al (25)	286	0.4	≥1:64	MA	serogroup III Bloomington 1 & 2
Bristol, TN	Dondero et al (26)	122	5.7	≥1:128	IFA	Philadelphia 1 & 2
Pittsburgh, PA	Elder et al (27)	129	7.0	≥1:160	Elisa	Knoxville
Ohio	Snowman et al (28)	588	20.0	≥1:128	IFA	Philadelphia 1
US	Klein et al (29)	548	4.7	≥1:32	MA	Knoxville
Italy	Re et al (30)	51	17.6	≥1:64	IFA	serogroup I & II

* = Indirect Fluorescent Antibody Test; † = Micro Agglutination Test; ‡ = Indirect Hemagglutination Assay

of age or older had prevalence rates 2.7 times higher than women of comparable ages.

Edson et al[16] suggested that as in the case of varicella, primary exposure to legionella infection probably occurs in children less than 15 years of age. The data reported by Yonke,[13] however, did not support this hypothesis. Our own results are compatible with Yonke's observations. We also found that for both men and women the youngest population group studied (≤19 years) had the lowest prevalence rates. We did not find any significant sex or age related differences in the prevalence rates of antibodies to *Legionella pneumophila.* Men had a slightly higher prevalence rate than women in all age groups, except the 30 to 39 year age group, where women had a higher prevalence. Despite the variations observed, none of the differences were significant. Bettelheim et al[19] also observed no differences in the prevalence rates between persons under and over 40 years of age, but in another geographic region, persons <40 years were found to have a higher rate (32.5% at 1:64) than those >40 years of age (2.5% at 1:64). In addition Foy et al[14] found no significant difference in the prevalence rates of children and adults. In the present study no significant difference was observed in the prevalence rates between population groups under and over 50 years of age when compared within the same sex, or between men and women of comparable ages.

There was a gradual drop in the prevalence rates during the period from 1979 to 1981 for both men and women. The drop was observed not only in the total population but also for the age-specific groups within each sex.

This study was implemented the year following an outbreak of legionellosis in the garment district of New York City.[1] The highest prevalence rate in the total population (31.8%) was in 1979. Subsequently the rates dropped to 22.1% and 16.9% in 1980 and 1981 respectively. The higher rate of 1979 may be attributed to the 1978 legionella outbreak. Observed drops in prevalence were probably due to the absence of continued or repeated exposure to the organisms.

Most of the prior seroepidemiologic investigations had not considered the effect of seasonal variance on the prevalence rates. Of the studies that did consider seasonal influences, two[13,16] found higher prevalence rates during the period of summer and autumn. Another investigation[17] reported that the prevalence rate was higher immediately after a severe winter than during the summer. Our results corroborate the finding of higher rates during the colder months. The higher winter rates in the present study may be due to other strains of *Legionella* or antigenically related organisms. Heterologous responses have been reported with legionella-like organisms and other organisms possessing common antigens.[18-20]

In a concurrent study (unpublished) involving suspected clinical cases of legionellosis, the incidence rate was found to be significantly higher during the warmer months than the colder months. A difference in the peaks and troughs of the prevalence and incidence rates may be due to the persistence of high titers of antibody and may actually reflect an infection that occurred months earlier. Brown et al[21] reported that during a nosocomial legionella infection in a Pennsylvania Veterans Administration hospital, both plasmid-bearing and plasmidless strains of *Legionella pneumophila* were isolated. After the epidemic subsided the investigators could isolate only plasmid-bearing strains. It is interesting to note that during the epidemic, the attack rate of legionellosis was 10 times higher in the wards where the plasmidless strains had been isolated than in the wards with plasmid-bearing strains. Furthermore, all clinical isolates were plasmidless. These investigators also observed that antisera prepared by injecting mice with plasmid-bearing strains lost their reactivity when they were first absorbed with plasmid-bearing strains; the same was not true following prior absorption with plasmidless strains. From these results they concluded that some antigenic factor besides the presence of a plasmid may also be responsible for the difference in virulence or environmental adaptability, or both. Plouffe and coworkers[22] have reported three subtypes of *Legionella pneumophila* serogroup 1 with different virulence characteristics.

In this seroepidemiologic study, a difference in the pattern of prevalence rates in different months of the year was observed. If the hypothesis presented by Brown et al[21] is correct, the plasmid-bearing legionella strains may be able to adapt better to cooler temperatures. This would result in higher prevalence rates during the winter, due to the organisms low virulence. On the other hand, the more virulent plasmidless strains could cause higher incidence rates, due to their better adaptability to summer and autumn environmental conditions.

This seroepidemiologic study provides evidence for a high (average 16% to 32%) year-round prevalence of *Legionella pneumophila* serogroup 1. This prevalence was found to be independent of age or sex. The data also established that seasonal changes significantly influence the prevalence rate. At present, the causal relationship cannot be established for the occurrence of higher prevalence (and incidence) rates in the different months of the year, although cross-reactivity with other strains of *Legionella pneumophila* and related organisms may be a factor. This apparent anomaly may be resolved by studies over an extended period of time in different environmental settings and by studying the prevalence of strains antigenically related to *Legionella pneumophila* serogroup 1.

Acknowledgments. The late Sol Blumenthal, PhD, Executive Director of Biostatistics, New York City Department of Health, provided useful suggestions, and Helen Kravetz, Austin Swaby, Remona Clark, and Minnie Barnes offered technical assistance.

REFERENCES

1. Cordes LG, Goldman WD, Marr JS, et al: Legionnaires' disease in New York City, August-September 1978. *Bull NY Acad Sci* 1980; 56:467–482.

2. Storch G, Hayes PS, Hill DL, Baine WB: Prevalence of antibody to *Legionella pneumophila* in middle-aged elderly Americans. *J Infect Dis* 1979; 140: 784–788.

3. Helms CM, Johnson W, Renner ED, Hierholzer Jr WJ, Wintermeyer LA, Viner JP: Background prevalence of microaggultination antibodies to *Legionella pneumophila* serogroups 1, 2, 3 and 4. *Infect Immun* 1980; 30:612–614.

4. Helms CM, Renner ED, Viner JP, Hierholzer Jr WJ, Wintermeyer LA, Johnson W: Indirect immunofluorescence antibodies to *Legionella pneumophila:* frequency in a rural community. *J Clin Microbiol* 1980; 12:326–328.

5. Smalley DL, Ourth DD: Seroepidemiology of *Legionella pneumophila.* A study of adults from Memphis, Tennessee, USA. *Am J Clin Pathol* 1981; 75: 201–203.

6. McDade JE, Shepard CC, Fraser DW et al: Legionnaires' disease. Isolation of a bacterium and demonstration of its role in other respiratory disease. *N Engl J Med* 1977; 297:1197–1203.

7. Wilkinson HW, Fikes BJ, Cruce DD: Indirect immunofluorescence test for

serodiagnosis of Legionnaires' disease: evidence for serogroup diversity of Legionnaires' disease bacterial antigens and for multiple specificity of human antibodies. *J Clin Microbiol* 1979; 9:379-383.

8. Farshy CE, Klein GC, Feeley JC: Detection of antibodies to Legionnaires' disease organism by microagglutination and micro-enzyme-linked immunosorbent assay tests. *J Clin Microbiol* 1978; 7:327-331.

9. Lattimer GL, Cepil BA: Legionnaires' disease serology. Effect of antigen preparation on specificity and sensitivity of the indirect fluorescent antibody test. *J Clin Pathol* 1980; 33:585-590.

10. England III AC, Fraser DW: Sporadic and epidemic nosocomial legionnellosis in the United States. Epidemiologic features. *Am J Med* 1981; 70:707-711.

11. Haley CE, Cohen ML, Halter J, Meyer RD: Nosocomial Legionnaires' disease: a continuing common-source epidemic at Wadsworth Medical Center. *Ann Intern Med* 1979; 90:583-586.

12. Broome CV, Goings SAJ, Thacker SB, et al: The Vermont epidemic of Legionnaires' disease. *Ann Intern Med* 1979; 90:573-577.

13. Yonke CA, Stiefel HE, Wentworth BB, Wilson DL: Prevalence of antibody to serogroups 1-4 of *Legionella pneumophila*. A seroepdemiologic study using the indirect hemagglutination test. *Am J Epidemiol* 1982;115:633-639.

14. Foy HM, Broome CV, Hayes PS, Allan I, Cooney MK, Tobe R: Legionnaires' disease in a prepaid medical-care group in Seattle 1963-75. *Lancet* 1979; 1:767-770.

15. Bettelheim KA, Metcalfe RV, Sillars H: Levels of antibody against *Legionella pneumophila* serotype 1 in healthy populations in five areas in New Zealand. *J Clin Microbiol* 1982; 16:555-557.

16. Edson DC, Stiefel HE, Wentworth BB, Wilson DL: Prevalence of antibodies to Legionnaires' disease. A seroepidemiologic survey of Michigan residents using the hemagglutination test. *Ann Intern Med* 1979; 90:691-693.

17. Bettelheim KA, Metcalfe RV, Sillars H: Antibodies to *Legionella pneumophila* serogroup 1 among the healthy New Zealand population. *NZ Med J* 1981; 93:259-261.

18. Wilkinson HW, Farshy CE, Fikes BJ, Cruce DD, Yealy LP: Measure of immunoglobulin G-, M-, and A-specific titers against *Legionella pneumophila* and inhibition of titers against nonspecific, gram-negative bacterial antigens in the indirect immunofluorescence test for legionellosis. *J Clin Microbiol* 1979; 10:685-689.

19. Ormsbee RA, Peacock MG, Lattimer GL, Page LA, Fiset P: Legionnaires' disease: antigenic peculiarities, strain differences, and antibiotic sensitivities of the agent. *J Infect Dis* 1978; 138:260-264.

20. Edelstein PH, McKinney RM, Meyer RD, Edelstein MA, Kraus CJ, Finegold S: Immunologic diagnosis of Legionnaires' disease: cross-reactions with anaerobic and microaerophilic organisms and infections caused by them. *J Infect Dis* 1980; 141:652-655.

21. Brown A, Vickers RM, Elder EM, Lema M, Garrity GM: Plasmid and surface antigen markers of endemic and epidemic *Legionella pneumophila* strains. *J Clin Microbiol* 1982; 16:230-235.

22. Plouffe JF, Para MF, Mather WE, Hackman B, Webster L: Subtypes of *Legionella pneumophila* serogroup 1 associated with different attack rates. *Lancet* 1983; 2:649-650.

23. Macrae AD, Appleton PN, Laverick A: Legionnaires' disease in Nottingham, England. *Ann Intern Med* 1979; 90:580-583.

24. Grist NR, Reid D, Nájera R: Legionnaires' disease and the traveller. *Ann Intern Med* 1979; 90:563-564.

25. Collins MT, Cho SN, Reif JS: Prevalence of antibodies to *Legionella pneumophila* in animal population. *J Clin Microbiol* 1982; 15:130-136.

26. Dondero Jr TJ, Clegg 2d HW, Tsai TF, et al: Legionnaires' disease in Kingsport, Tennessee. *Ann Intern Med* 1979; 90:569-573.

27. Elder EM, Brown A, Remington JS, Shonnard J, Naot Y: Microenzyme-linked immunosorbent assay for detection of immunoglobulin G and immunoglobulin M antibodies to *Legionella pneumophila*. *J Clin Microbiol* 1983; 17:112-121.

28. Snowman WJ, Holtzhauer FJ, Haplin TJ, Correa-Villasenor A: The role of indoor and outdoor occupations in the seroepidemiology of *Legionella pneumophila*. *J Infect Dis* 1982; 145:275.

29. Klein GC, Jones WL, Feeley JC: Upper limit of normal titer for detection of antibodies to *Legionella pneumophila* by the microagglutination test. *J Clin Microbiol* 1979; 10:754-755.

30. Re MC, Baldassari B, Furlini G, Coppolecchia P, Landini MP: Legionnaires' disease in Northern Italy. *Eur J Clin Microbiol* 1983; 2:362-363.

Use of health services by merchant seamen

HOWARD R. KELMAN, PHD, FLORENCE KAVALER, MD

ABSTRACT. American merchant seamen represent a unique group in the spectrum of health care provision in the United States. Until recently they were the only civilian occupational group that was eligible to receive medical, surgical, dental, and other health care benefits under the tax-supported federal health care system. The United States Public Health Service Hospital and Clinic System, which had been in existence since 1798, originally consisted of eight acute care general hospitals, one special long-term facility for patients with Hansen's Disease, 30 out-patient clinics, and 308 contract physicians. American merchant seamen were the largest single beneficiary group and constituted 40% of the total of 500,000 eligible persons treated. A sample of healthy, actively employed seamen was interviewed, and the findings suggest that the Public Health Service system was their main source of health care, although alternate sources of care were used.

(NY State J Med 1985; 85:14–17)

INTRODUCTION

The United States Public Health Hospital and Clinic System, which was recently dismantled, represented a shrunken portion of what had been a much larger federal system of hospitals, clinics, and contact physicians, which at one time spanned the country. The rationale for reducing the system rested on evidence of a steady decline in the use of its services by eligible beneficiary groups.[1,2] Studies completed by the Health Services Research Unit at the Staten Island Public Health Service Hospital in 1978 revealed the following trends in use of Public Health Service health care facilities by seamen for the period 1971 to 1975:[3] (1) The volume of use of hospital care by seamen had declined as reflected in decreasing numbers of inpatient admissions and discharges and in the use of hospital based outpatient clinics. (2) In contrast to this pattern, the volume of use of

---

From the Division of Sociomedical Sciences, Department of Social Medicine, Montefiore Medical Center (Dr Kelman), and the Department of Preventive Medicine and Community Health, SUNY-Downstate Medical Center (Dr Kavaler), New York.

Dr Kelman was formerly consultant and chief, Health Services Research Unit, United States Public Health Service Hospital, Staten Island, NY; Dr Kavaler was formerly assistant surgeon general and director, United States Public Health Service Hospital, Staten Island, NY.

Address correspondence to Dr Kavaler, Professor, Department of Preventive Medicine and Community Health, SUNY-Downstate Medical Center, 450 Clarkson Ave, Brooklyn, NY 11203.

TABLE I. Selected sociodemographic characteristics of seamen respondents

Characteristics	Percent of Total	Percent Unlicensed	Percent Licensed
No. of respondents	1130	923	207
Age			
18–39 yr	38.6	40.2	31.4
40–54 yr	31.7	33.6	23.2
55+ yr	29.6	26.1	44.9
Marital status			
Never married	37.3	38.8	30.4
Currently married	37.9	34.3	53.6
Previously married	24.6	26.5	16.0
Ethnicity/race			
Hispanic	13.6	15.5	5.3
Black	13.1	15.8	1.0
White	70.1	65.0	92.8
Other	3.2	3.7	1.0
Education			
Grade school or less	18.3	21.8	2.9
Some high school	22.9	26.1	8.7
High school graduate	28.1	28.8	24.6
Some college or more	29.7	22.3	62.8
Days worked in 1978			
None	8.5	8.7	7.7
Less than 180	42.2	41.3	44.4
180 or more	49.6	50.1	47.9
Income			
Under $10,000	35.8	41.2	12.1
$10,000–14,999	24.6	28.2	8.7
$15,000–24,999	26.4	23.1	41.1
$25,000+	10.4	4.6	36.3

TABLE II. Proportion of Reported Hospital, Ambulatory and Dental Service use Resulting from Shipboard Health Problems by Seamen Respondents

Medical and Dental Care Reported	Total	Unlicensed	Licensed
Total no. of hospital admissions*	272	236	36
Attributed to shipboard health problems			
No.	122	114	8
Percent	44.9	48.3	22.2
Ambulatory medical care† (Total no.)	716	583	133
Attributed to shipboard Health problems			
No.	285	234	51
Percent	39.8	40.1	38.3
Dental care† (Total no.)	499	362	137
Attributed to shipboard Health problems			
No.	132	110	22
Percent	26.4	30.4	16.1

* Hospital admissions reported for previous 24 months.

† Care received during previous 12 months.

community based contract physicians increased during this time period. Fewer seamen were admitted to hospitals for accidents and for infectious, circulatory, digestive, or genitourinary diseases, whereas admissions increased for neoplasms, nervous system disorders, ill-defined symptoms, and for special conditions.[4,5]

It is unlikely that this decline in use of the Public Health Service facility by seamen was due to a decrease in the size of the eligible population or to reduced needs for health care, especially since the seamen have become older.[6] In 1975, the Public Health Service estimated that 213,300 seamen were eligible for care. This represented an increase of more than 15,000 from 1974, and 23,500 from 1973.[7] Alternative explanations—lessened demand as a function of access problems or as a consequence of obtaining care outside the Public Health Service system—could not be investigated with the available data.

This study was initiated to provide more comprehensive information on the health characteristics of merchant seamen and their health services utilization patterns. Available data on seamen's health care needs were sparse and were limited to statistical information on seamen who had obtained health care from the Public Health Service hospital and clinic health care system, or to tabulations of injuries and illnesses that occurred aboard ship and were reported to the Marine Index Bureau.[8]

PATIENTS AND METHODS

Population Samples. In this interview study, data were obtained from seamen who were not sick, in the hospital, or clinic patients at the time of the study. Information was gathered on their perceived health status, and the volume, types, and auspices of health care they had obtained from sources both inside and outside of the Public Health Service while ashore and at sea.

Data were gathered through personal interviews with 1,130 seamen conducted by trained interviewers in union hiring halls in New York City, San Francisco, New Orleans, Chicago, and St. Louis. The cooperation of several maritime unions facilitated the data collection effort. The interviews were conducted in a private setting in each of the hiring halls, using a structured questionnaire that had been pretested. Periodic rechecks on randomly chosen seamen were performed for quality control.

The study included unlicensed able-bodied seamen interviewed at three deep seaport cities where there was a local Public Health Service Hospital (New York, San Francisco, and New Orleans), and two inland port cities without a local Public Health Service Hospital (Chicago and St. Louis). The total sample included a group of licensed—officers and mates—seamen in New York City and in San Francisco. The resulting sample consisted of presumably well, able-bodied union seamen, and represented a population that was "broader" than patient groups served by the Public Health Service health care system or other health care facilities.

In addition to identifying demographic and social data, information was sought on occupational background, use of inpatient and outpatient health services, sources and sites of treatment, present health and dental status, status of chronic conditions, knowledge of health benefits and eligibility for Public Health Service care, and satisfaction and problems with care received.

RESULTS

Characteristics of the respondents. Interviews were obtained from 923 unlicensed seamen and 207 licensed seamen. The sociodemographic characteristics are shown in Table I. It should be noted that licensed seamen were generally older, had higher incomes, were predominantly white, and had achieved higher levels of education than did the unlicensed seamen.

TABLE III. Percentage Distribution of Reported Chronic Conditions and Whether Treated of Seamen Respondents*

	Total			Unlicensed			Licensed		
Chronic Conditions	No.	Percent reported	Percent treated	No.	Percent reported	Percent treated	No.	Percent reported	Percent treated
Respondents reporting one or more conditions	564		53.0	457		54.0	107		48.6
Hypertension	174	30.9	57.5	141	30.9	60.3	33	30.8	45.5
Arthritis or rheumatism	118	20.9	37.3	102	22.3	39.2	16	15.0	25.0
Impairment of back or spine	92	16.3	28.3	73	16.0	30.1	19	17.8	21.1
Hearing impairment	58	10.3	17.2	42	9.2	19.0	16	15.0	12.5
Stomach ulcer or trouble	56	9.9	57.1	45	9.8	62.2	11	10.3	36.4
Asthma or lung trouble	50	8.9	36.0	42	9.2	38.1	8	7.5	25.0
Diabetes	47	8.3	61.7	41	9.0	65.9	6	5.6	33.3
Drinking problems	45	8.0	6.7	41	9.0	7.3	4	3.7	—
Visual impairments	45	8.0	40.0	36	7.9	44.4	9	8.4	22.2
Heart conditions	41	7.3	65.9	33	7.2	63.6	8	7.5	75.0
Dental problems	130	23.0	30.1	116	25.4	30.2	14	13.1	28.6
All other conditions	178	31.5	32.0	132	28.9	31.1	46	43.0	34.8

* Percent based on number of persons reporting one or more conditions (N = 564).

Sources and volume of obtained health services. Two hundred and seventy-two inpatient hospital stays—a third due to accidents and related conditions—were reported for the previous 24-month period. Unlicensed seamen reported more admissions for accidents, and for digestive, respiratory, and mental conditions, and they experienced longer hospital stays, than did licensed seamen.

More than half of all reported hospital stays were in Public Health Service Hospitals, a third in non-Public Health Service Hospitals in the United States, and just over 10% were in hospitals in foreign ports. In port cities where there was a Public Health Service Hospital available, two-thirds of all hospital stays for unlicensed seamen and nearly 40% of hospital visits for licensed seamen were in Public Health Service facilities.

In the preceding 12-month period, seamen reported a total of 716 episodes of illness for which they received ambulatory care. Accidents and related conditions were the most frequently reported condition for unlicensed seamen. For licensed seamen, "check-ups" exceeded other conditions as a reason for obtaining ambulatory care, and the number of accidents reported by them was less than half the number reported by unlicensed seamen. Two-thirds of these episodes were treated in Public Health Services facilities such as hospital outpatient departments and clinics, and by contract physicians. Unlicensed seamen used Public Health Service sources more frequently than did licensed seamen (73% versus 42%). The fewest number of such events reported and the smallest proportion of use of Public Health Service sources occurred in Chicago and St. Louis, cities in which a Public Health Service hospital was not available.

Nearly half of all respondents obtained dental care during the preceding 12-month period. Most dental treatment was obtained from non-Public Health Service sources—85% for licensed and 54% for unlicensed seamen. Unlicensed seamen sought treatment for dental problems more frequently than licensed seamen; dental care for the latter group was nearly evenly divided between dental treatment and checkups.

TABLE IV. Percentage Distribution of Reported Health Insurance Coverage of Seamen Respondents

	Respondent Group		
Type of Coverage	Percent total	Percent unlicensed	Percent licensed
(No. of respondents)	(1130)	(923)	(207)
Union health plan only	56.0	58.0	46.4
Union and veterans administration	16.6	17.6	12.1
Union and Medicaid or Medicare	4.1	3.0	8.7
Union and other type of third party coverage	8.8	4.6	27.5
All other coverage (non-union)	4.5	4.8	2.9
None	4.9	5.5	1.9
Do not know	5.1	6.5	.05

Seamen—unlicensed more so than licensed—attributed much of their health service use and need for medical care to events that occurred aboard ship: 45% of the 272 hospital admissions; 40% of the ambulatory care conditions; and 26% of the reported dental conditions (Table II).

Health status. About 50% of the respondents reported one or more chronic conditions, and 25% reported two or more. In nearly half of these instances the seamen were receiving care; unlicensed seamen were more likely than licensed seamen to be receiving care for chronic conditions (Table III).

Hypertension, arthritis and rheumatism, impairments of the back and spine, hearing difficulties, asthma, and diabetes were the most frequently reported conditions. Age was significantly associated with the number of chronic conditions reported and with the receipt of care. Despite the prevalence of chronic disease reported, three-fourths of the respondents who reported such conditions also reported no disabling consequences, and 85% of all respondents assessed their own health as being either good or excellent.

Income, absence of chronic conditions, and identification of a non-Public Health Service source of regular care were associated with good or excellent assessments of health.

Factors related to use of Public Health Service facilities. While 90% or more of the respondents considered themselves eligible for Public Health Service care and were knowledgeable about specific Public Health Service services, nearly all respondents reported some form of health insurance coverage, including their union pension and health plan, and many reported multiple sources (Table IV).

Three-fourths of the unlicensed seamen and a third of the licensed seamen regarded the Public Health Service, mainly the hospital outpatient department, as their regular source of health care. The major reasons reported for use of Public Health Service facilities were (in order of frequency) financial, referral, accessibility, and quality of care. Reasons reported for use of non-Public Health Service facilities were accessibility, emergency care, and dissatisfaction with Public Health Service care.

Public Health Service hospital and ambulatory care facilities were used more frequently by seamen in this study who had the following characteristics: were unlicensed; interviewed in cities where a Public Health Service Hospital is located; were members of minority groups; earned less than $25,000 per year; were older and less educated; and reported one or more chronic conditions.

DISCUSSION

The results of this survey indicate that although the

Public Health Service was the predominant source of health care, a significant number of seamen availed themselves of other sources of care. Unlicensed and older seamen, those with lower incomes, and seamen who reported having one or more chronic conditions were more likely to have been regular users of the Public Health Service system for financial, accessibility, and quality of care reasons.

The dismantling of the Public Health Service system, coupled with the nearly universal availability (in this sample) of union-sponsored health benefits, will result in the incorporation of this occupational group into the available health and medical care resources utilized by the general population. As a consequence, we may lose sight of the particular health care related problems and characteristics of an occupational group that spends a considerable portion of its working time at sea away from homes, families, and shore-based sources of medical care.

Although it may be hazardous to generalize from this sample population to all merchant seamen, it is important for physicians and other health care providers to note that this group reports high rates of hypertension, arthritis, rheumatism, diabetes, ulcers, and other chronic conditions—only half of which, on average, are reported to be under care. Yet seamen describe their health as good, and also ascribe much of their use of hospital, ambulatory, and dental services to shipboard related illnesses and accidents. Their reported patterns of health care use and health conditions suggest the need to develop procedures to monitor and manage these disorders, especially the chronic conditions, taking into account the long periods of time spent at sea. These considerations will effect employment decisions, the coordination of medical management, and attention to matters of diet, medication, and care for acute illness while at sea as well as ashore.

Finally, it should be noted that the data reported here were derived from a sample of healthy, actively employed seamen, and, therefore, they do not reflect the medical care needs of seamen who are severely ill, disabled, or in the hospital.

REFERENCES

1. US House of Representatives: Hearings before the Committee on Merchant Marine and Fishermen, 94th Congress, 2nd Session, June 11 and 14, July 26, 1976.
2. Office of Science and Technology, Public Health Service Hospital Study Committee: Webster Report, 1965.
3. USPHS Hospital Staten Island: Progress Report Health Services Research Unit. New York, Bureau of Medical Services, Department of Health, Education, and Welfare, 1978.
4. Barclay RW, Gray EA: Health problems of American seamen as measured by hospital statistics. *Public Health Reports* 1958; 73:585–589.
5. National Maritime Research Center: *Injuries and Illness on U.S. Ships.* Department of Commerce Maritime Administration, October 1978, NMRC-KP-193.
6. Maritime Transportation Research Board: *Shipboard Manpower.* National Research Council, Washington, DC, 1968.
7. Bureau of Medical Services: *Annual Statistical Summary Fiscal Year 1975.* Washington, DC, USPHS.
8. *Statistical Analysis No. 65*, Marine Index Bureau, New York, April 2, 1976.

ASSISTANCE FOR VICTIMS OF FAMINE

> *All deaths are hateful to miserable mortals, but the most pitiable death of all is to starve.*
>
> *Homer*
> The Odyssey, *XII, 341*
> (*translated by W.H.D. Rouse*)

Donations to the Ethiopian famine victims can be sent to Interaction, a coalition of more than 20 humanitarian and church relief groups:

Interaction
200 Park Ave S
New York, NY 10003
(212) 777-8210

Neuro-ophthalmologic aspects of occlusive cerebrovascular disease

ARTHUR H. WOLINTZ, MD

Occlusive cerebrovascular disease is an important cause of stroke. Occlusive disease of the carotid and vertebrobasilar arteries may produce the following visual defects:[1] amaurosis fugax; unilateral loss of vision caused by occlusion of a central retinal artery, one of its branches, or of the artery to the optic nerve; binocular visual defects secondary to occlusion of a middle cerebral artery or its branches with resulting incongruous homonymous hemianopia; binocular visual defects secondary to occlusion of a posterior cerebral artery with resulting congruous homonymous hemianopia; hypotensive manifestations in the retina on the affected side, including cotton-wool patches in the retina, widening of the arterioles and venules, and venous-stasis retinopathy; hemorrhagic glaucoma; bright embolic cholesterol plaques or fibrin platelet aggregates in the retinal arterioles; lowering of the ipsilateral retinal artery pressure; or Horner's syndrome.

The signs and symptoms that occur with occlusive disease are caused by local tissue ischemia of the eye or of the visual and oculomotor pathways. Blood flow to a given area that has been partially compromised by an atherosclerotic process in the local circulation can be further impaired by hypotension, decreased cardiac output, vasospasm, transient failure of the collateral circulation, or narrowing of the lumen of the extracranial vessels in the neck or thorax.

Plaques can also cause ischemia and can produce signs and symptoms when they occur at various sites in the aortocranial arteries: bifurcation of the common carotid artery; angulations, bifurcations, and areas of dilation or tortuosity—the carotid artery siphon, for example; the arch of the aorta; subclavian arteries; carotid sinus; vertebral arteries, between transverse processes of cervical vertebrae (especially C6); proximal portions of anterior, middle, and posterior cerebral arteries; and the intracranial vertebral, posterior inferior cerebellar, or basilar arteries.

SIGNS AND SYMPTOMS OF OCCLUSIVE DISEASE

Almost 90% of the patients with carotid artery insufficiency will have ocular signs or symptoms.[2] The transient attack of ischemia is a short episode of neurologic dysfunction that, when confined to the distribution of the carotid system, may produce temporary blurring of vision, headaches, focal seizures, hemiplegia, hemisensory defects, and aphasia.

The ocular hallmark of occlusive carotid disease is amaurosis fugax (fleeting blindness) in which the patient suddenly loses vision in one eye, usually for up to 10 minutes, after which it gradually returns. Amaurosis fugax may be total or partial; the dimming of vision can occur in the upper or lower half of the field of vision or the sensation may be that of specks of bright light fleeting across the visual field. The patient may describe a curtain or shade that obscures part or all of the vision in one eye. These episodes may also include burning or watering of the involved eye, transient paresthesias of the medial brow and forehead in the distribution of the terminal branches of the ophthalmic artery, and peculiar feelings behind the eye preceding the "blackout."

The fleeting monocular blindness is caused by a combination of atheromatous disease of the carotid artery in the neck with superimposed hypotension or vasoconstriction, producing local ischemia. In addition, mural thrombi may be dislodged from plaques in the carotid vessels of the neck, flow downstream to small peripheral vessels, cause transient signs, and then fragment and disappear in the bloodstream.

Shiny yellow particles, considered to be cholesterol crystals, within the lumen of a retinal arteriole, usually at a bifurcation, are frequently found in patients with a history of amaurosis fugax. White nonreflecting plugs of material may also be seen within the lumen of the retinal vessels (Fig 1). These are thought to be composed of platelets and fibrin, as well as fats.[3] Hollenhorst[4] found that 30% of patients with cerebrovascular insufficiency had one or more orange, yellow, or copper-colored intraluminal plaques lodged at a bifurcation of the retinal arterioles. He also found that 20% of patients undergoing endarterectomy had such particles within the retinal vasculature, and he presumed that these were flakes of cholesterol that had been washed into the retinal vascular tree from ulcerating atheromatous lesions in the neck, usually a stenotic lesion at the bifurcation of the carotid artery.

These transient ischemic attacks usually cease after the

Address correspondence to Dr Wolintz, Interim Director, Department of Ophthalmology, SUNY-Downstate Medical Center, 450 Clarkson Avenue, Box 58, Brooklyn, NY 11203.

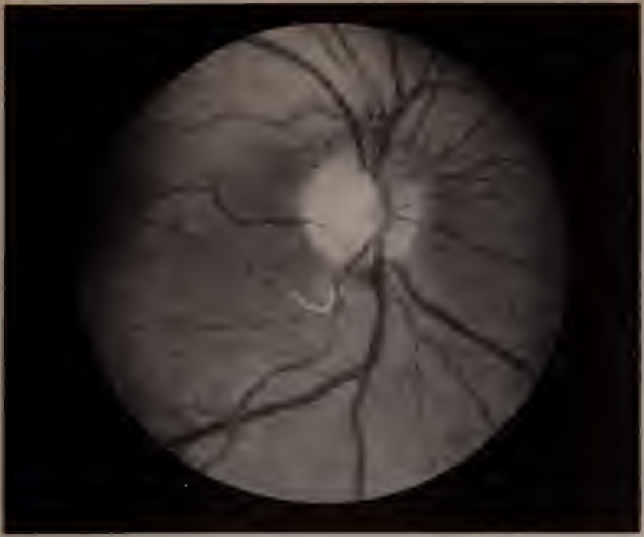


FIGURE 1. Photograph of fundus showing plaque of the inferior temporal arteriole with extension to secondary bifurcation.

carotid artery has become completely occluded. Vision may then remain good if there is collateral circulation through the circle of Willis or the external carotid artery.

Transient loss of vision can also be caused by cardiac arrhythmias, giant cell arteritis, hypertensive crises, increased venous pressure, and fluctuating retinal perfusion secondary to an impending central retinal vein occlusion, or by an intermittent increase in intraocular pressure. Episodic loss of vision is also found in patients with polycythemia vera, severe anemias, and thrombocythemia. Transient amaurosis may be produced by emboli that occur in valvular heart disorders such as rheumatic heart disease and subacute bacterial endocarditis, as well as in the mitral valve prolapse syndrome.[5]

VENOUS STASIS RETINOPATHY

Venous stasis retinopathy was initially described by Hedges[6] in 1962 and by Kearns and Hollenhorst[7] in 1963.

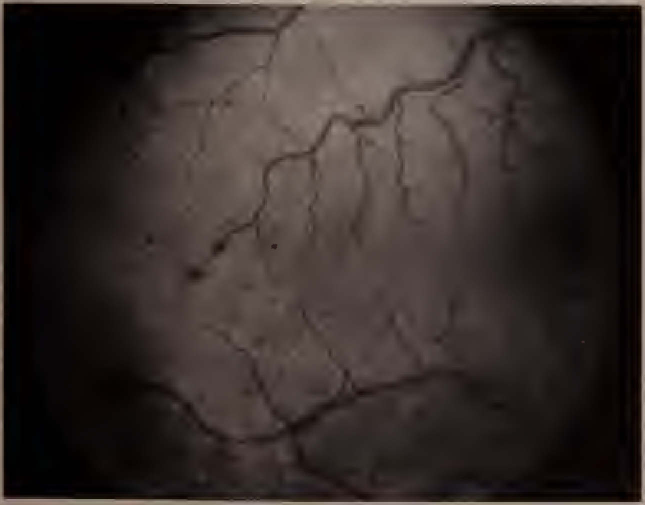


FIGURE 2. Photograph of fundus of patient demonstrating features of venous stasis retinopathy, including microaneurysms, capillary dilation, and retinal hemorrhages.

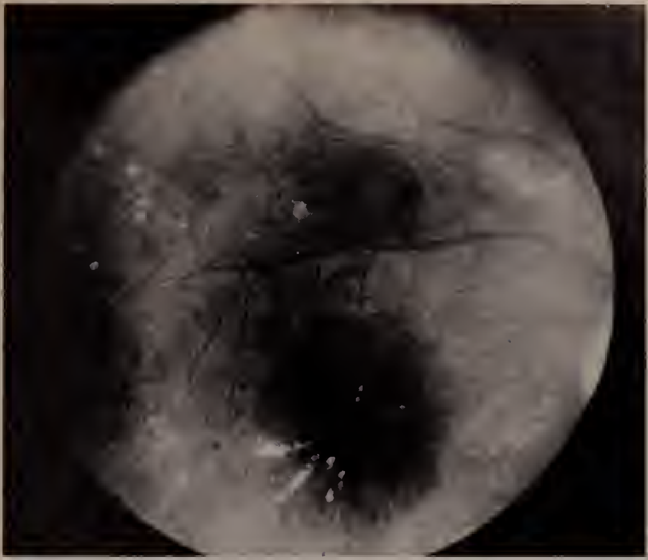


FIGURE 3. Photograph of fundus indicating some features of patient with diabetic retinopathy in posterior pole.

These retinal changes consist of microaneurysms, punctate areas of capillary dilation, small retinal hemorrhages, and, in severe cases, dilation, darkening, and irregularity of caliber of the major retinal veins (Fig 2). Venous stasis retinopathy resembles diabetic retinopathy. Both are caused by ischemia and resulting hypoxia, although the site of the obstruction to blood flow is different. In diabetes the obstruction is caused by altered hemodynamics in the retinal microcirculation,[8] whereas in carotid occlusive disease it is caused by a reduction in perfusion pressure from atheromatous obstruction proximal to the retinal microcirculation.

Diabetic retinopathy is usually bilateral, whereas venous stasis retinopathy is usually unilateral. However, in bilateral occlusive disease of the carotid arteries, bilateral venous stasis retinopathy does occur. The early retinal changes of venous stasis retinopathy are first observed in the mid-peripheral retina, as opposed to the early retinal changes of diabetic retinopathy that are first seen in the macula and the posterior pole of the eye (Fig 3). The finding that best differentiates the two is a low retinal artery pressure. Venous stasis retinopathy occurs only with severe occlusive disease, and, in most instances, the carotid artery will be occluded on the affected side.[9]

Venous stasis retinopathy may also be confused with occlusion of the central retinal vein. The microaneurysms and punctate dilation of the capillaries are similar; however, patients with venous occlusions usually have more hemorrhage (Fig 4). The veins may also be dark and engorged, but they do not become irregular in caliber as they often do in patients with venous stasis retinopathy. As in diabetic retinopathy, the retinal artery pressure will always be normal in patients with a central retinal vein occlusion and low in patients with venous stasis retinopathy.

Cotton-wool patches in the retina on the affected side have been described in patients with carotid artery insufficiency.[10] They can also be found in patients with arteriosclerotic and hypertensive retinopathy and in patients with venous stasis retinopathy, diabetes, and with a central retinal vein occlusion. They result from small infarcts secon-

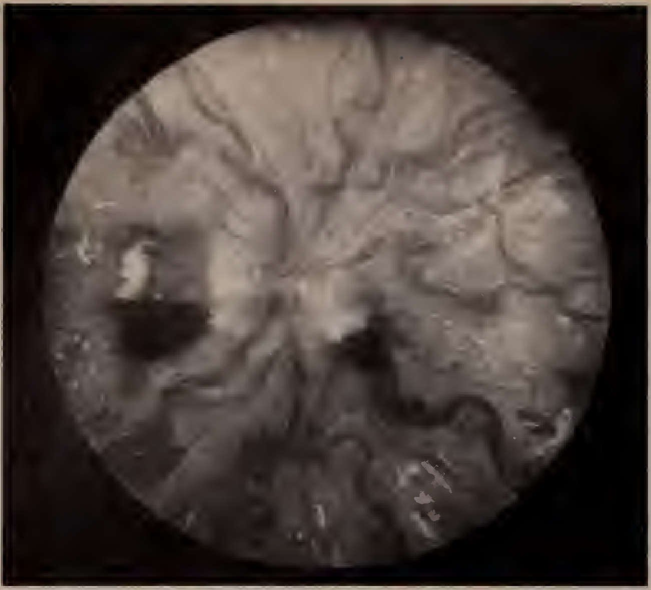


FIGURE 4. Photograph of fundus demonstrating recent occlusion of the central retinal vein with hemorrhages, distention of veins, and edema of the disc.

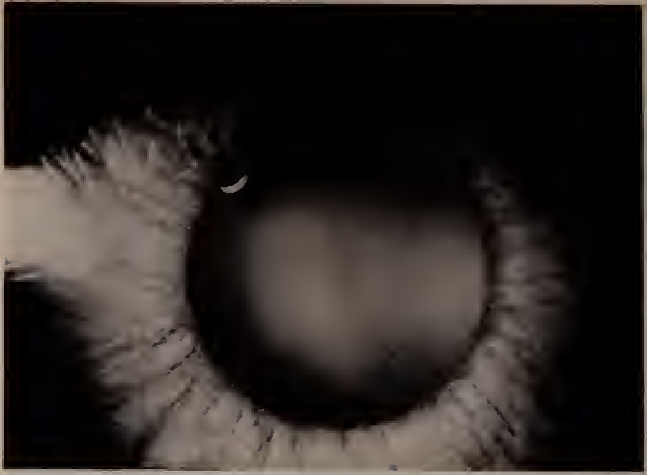


FIGURE 5. Photograph of patient with rubeosis iridis showing new vessel formation on the anterior surface of the iris.

dary to vascular insufficiency. The presence of one or more such patches in one eye may be the initial clue to the diagnosis of ipsilateral carotid arterial occlusive disease.

In the more advanced type of venous stasis retinopathy, rubeosis iridis, neovascularization of the anterior chamber angle, and secondary glaucoma may develop (Fig 5). Proliferating neovascular tissue on the surface of the ischemic iris may occlude the anterior chamber angle and produce a secondary "neovascular glaucoma." As in other forms of neovascular glaucoma, a cataract usually develops and the retinopathy is obscured. Thus, the ophthalmologist who encounters a patient with neovascular glaucoma should consider the possibility of carotid occlusive disease in the differential diagnosis.[11] If such a patient is considered to have an idiopathic occlusion of the central retinal vein with secondary glaucoma, and if carotid occlusive disease is overlooked, a catastrophe may result. For example, neovascular glaucoma can be found in a patient with contralateral hemiplegia and homonymous hemianopia.

In the late stages of severe ischemia, there may be conjunctival hyperemia, corneal edema with folds in Descemet's membrane, vitreous hemorrhage, posterior synechiae, and iris atrophy.

Venous stasis retinopathy is present in approximately 20% of patients with carotid occlusions. Some decrease in venous stasis retinopathy has been observed after successful superficial temporal artery-middle cerebral artery bypass surgery.[12,13] Progressing venous stasis retinopathy with subsequent neovascular glaucoma is an indication for the performance of such a bypass procedure.

CAROTID ARTERY OCCLUSION

Patients with occlusion of an internal carotid artery may also demonstrate a peculiar retinal change that is thought to be secondary to a collateral flow mechanism. A significant percentage of patients with occlusion of an internal carotid artery show retrograde filling of the anterior and middle cerebral arteries by way of the external carotid, orbital, and ophthalmic arteries. In 1976, Neupert[14] and associates reported on the rapid resolution of a retinopathy consisting of a delicate network of new vessels extending from the right optic disc into the vitreous cavity. The retinal veins were dilated and irregular in caliber. Retinal hemorrhages and microaneurysms were also present. The patient described had an occlusion of the left internal carotid artery and 99% stenosis of the right internal carotid artery. After a right carotid endarterectomy the new vessels disappeared from the optic disc within 48 hours. The investigators believed that although the venous stasis retinopathy was induced by the almost occluded right internal carotid artery, the neovascularization on the optic disc was induced by a collateral flow mechanism through the external carotid, orbital, and ophthalmic arteries. This has been referred to as "collateral flow retinopathy."

Asymmetrical retinal vascular changes may indicate the presence of carotid artery disease. A unilateral internal carotid artery occlusion can protect the ipsilateral eye from the vascular effects of systemic hypertension. Thus, the presence of uniocular hypertensive vascular retinopathy suggests occlusion of the contralateral internal carotid artery[15] (Fig 6). Ischemic pain in the orbit in patients with carotid occlusion was described in 1963 by Kearns and Hollenhorst.[7] The pain is described as a constant aching over the orbit, the upper part of the face, and the temple; typically, it is worse when the patient is sitting or standing. Lying down tends to lessen or relieve the pain. It is thought that the pain is produced by ischemia resulting from the carotid occlusion. Gravitational factors explain its worsening when the patient is in the upright position and its decrease on lying down.

An alternate explanation could be dilation secondary to collateral flow through the external carotid (superficial temporal) artery. It should be noted that the pain usually occurs in patients who do not have signs of glaucoma or of ocular disease other than retinal arterial hypotension and venous stasis retinopathy. Patients who have vertebrobasilar disease may have pain in the cranial vertex or in the occipital or suboccipital regions. The pain that occurs in patients with occlusive vascular disease may arise from local low blood pressure and the resulting stasis of blood flow,

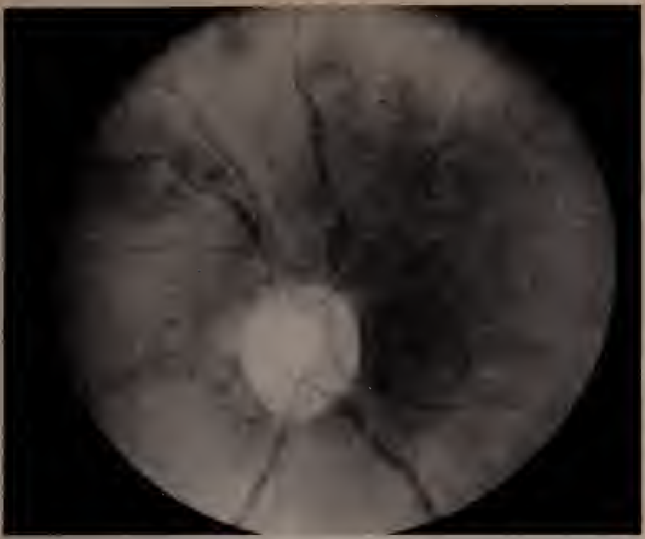


FIGURE 6. Photograph of fundus showing some features of hypertensive retinopathy, including narrowing and irregularity of retinal arteries and punctate and splinter hemorrhages.

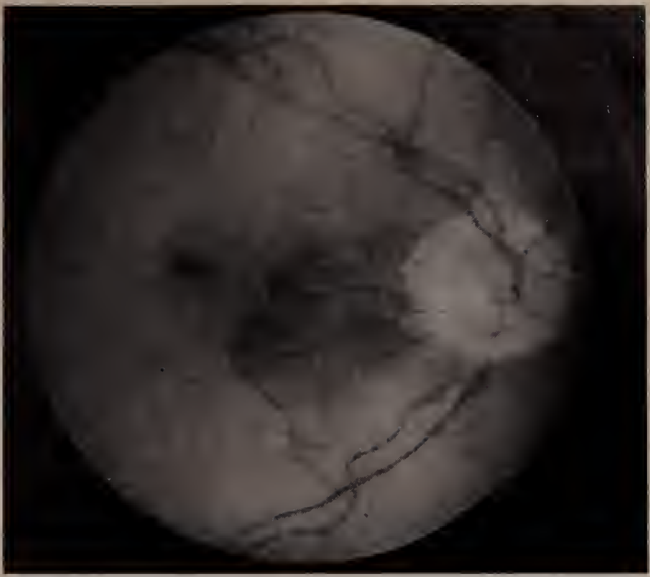


FIGURE 7. Photograph of fundus of patient with central retinal artery occlusion of the right eye. Noteworthy is the cherry-red spot in the macula with partial sparing of the peripapillary area.

local tissue hypoxia, and dilation of the capillaries and veins.

In 1979, Furlan and associates[16] described five patients with unilateral visual loss in bright light associated with carotid artery occlusive disease. It was suggested by these investigators that the loss was secondary to retinal ischemia of hemodynamic origin, such that the regeneration of visual pigment was slowed after bleaching by bright light. The most common change on angiography was ipsilateral occlusion or tight stenosis of the internal carotid artery. In one patient there was associated venous stasis retinopathy. Although venous stasis retinopathy and light-induced amaurosis fugax both reflect retinal ischemia secondary to carotid artery occlusive disease, one of these conditions does not necessarily accompany the other. Venous stasis retinopathy may also exist without visual symptoms. Although the presence of venous stasis retinopathy may be evidence of generalized retinal ischemia, the ischemia in the macular region may not be sufficient to produce visual symptoms when the patient is exposed to bright light. Thus, patients with light-induced visual symptoms but without venous stasis retinopathy have relative focal ischemia of the macular region.

VISUAL-EVOKED RESPONSES

Donnan, Sharbrough, and Whisnant[17] described four patients in whom the symptom of visual blurring occurred in the presence of bright light. These patients were studied with the use of visual-evoked response recordings before and after exposure to an artificial source of bright light. The study documented an abnormality of the visual-evoked responses associated with light-induced amaurosis fugax in patients with severe carotid artery occlusive disease.[16] Symptoms of visual blurring were reproduced in the patients by exposure to a source of artificial light. Immediately thereafter attenuation of the visual-evoked response was noted. The waveform usually returned to normal within two minutes after the cessation of exposure to the light. The visual-evoked response primarily reflects activity originating in the central three degrees of the visual field, and the activity of the cones of the fovea is the main component within this portion of the visual field. Since visual loss in bright light is most often associated with disease of the macula, the attenuation of the visual-evoked response in this study most likely represents change in function of the macular zone, presumably because of the reduction in local retinal blood flow, causing an inadequate regeneration of retinal pigments.

Improvement of the visual-evoked response with light after carotid endarterectomy is consistent with the previous reports of resolution of venous stasis retinopathy[14] and elimination of light-induced amaurosis fugax after endarterectomy.[16] Improvement in blood flow to the retina is probably the mechanism involved and may also be the basis for resolution of venous stasis retinopathy after superficial temporal-middle cerebral artery bypass surgery.[13]

PROFOUND VISUAL LOSS

About 20% of the patients who suffer from occlusive disease of the carotid arteries may experience the onset of visual loss of a more profound degree. The visual deficit usually appears suddenly and tends to persist, although in some patients it may resolve within 24 to 48 hours. Visual field defects that are attributable to occlusive disease of the carotid artery may appear as partial or complete loss of vision in the ipsilateral eye caused by occlusion of a central retinal artery or branch retinal artery, or by occlusion of a vessel to the optic nerve (Fig 7).

An important cause of sudden persistent monocular visual loss is infarction of the optic nerve—ischemic optic neuropathy. This is produced by occlusion of either a posterior ciliary artery or several posterior ciliary arteries that perfuse the optic nerve head. Ischemic optic neuropathy may be caused by microemboli from atherosclerotic plaques in the carotid arteries. The patient experiences sudden loss of vision or a visual field defect that tends to involve the upper or lower half of the visual field, particularly the lower

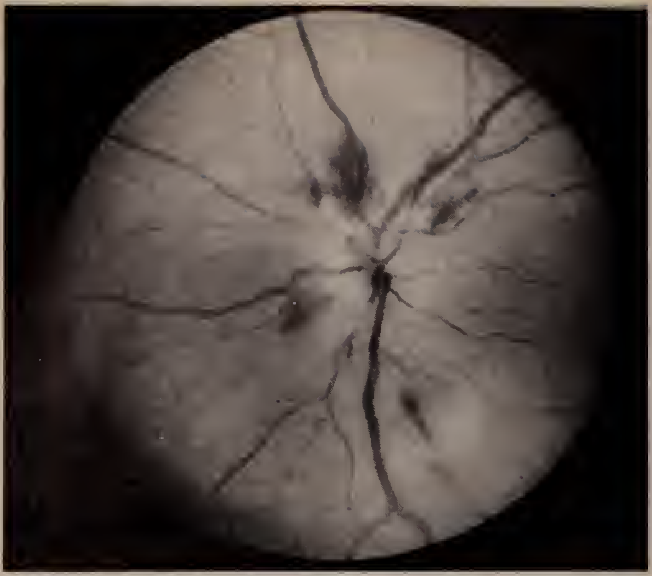


FIGURE 8. Photograph of fundus of patient with ischemic optic neuropathy involving the right eye. Important features include edema of the optic nerve, peripapillary flame-shaped hemorrhages, and cotton-wool spots.

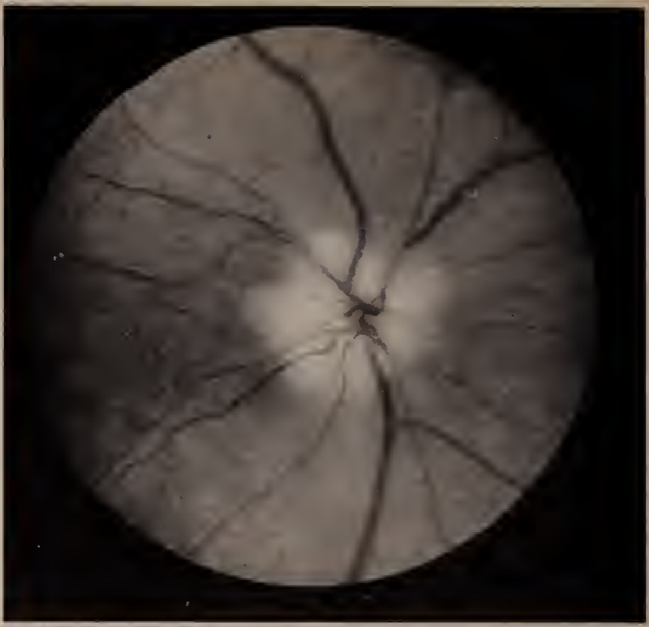


FIGURE 9. Photograph of fundus of patient with ischemic optic neuropathy showing significant pale edema of optic nerve.

half. The patient manifests decreased perception of colors and a positive swinging flashlight sign. The positive swinging flashlight sign is elicited when the affected pupil is stimulated with light after stimulating the preserved eye. Instead of pupillary constriction, the pupil on the affected side dilates. The optic nerve head appears elevated and edematous. Flame-shaped hemorrhages may be present on the surface of the optic nerve (Fig 8). The optic disc may also appear pale and edematous with the surrounding retina being relatively normal (Fig 9). With a central retinal artery occlusion the optic disc is initially normal and the surrounding retina appears pale and edematous. The visual field deficit with ischemic optic neuropathy is usually permanent, even though, occasionally, significant improvement may occur. The edema of the optic nerve head usually resolves, producing pallor, which may be segmental.

Partial or complete loss of vision in the ipsilateral eye and of the temporal field of the contralateral eye, resulting from involvement of the arteries to the ipsilateral retina combined with simultaneous or consecutive thrombosis of the homolateral middle cerebral artery, may also occur. Another finding may be an incongruous homonymous hemianopia caused by occlusive disease of the middle cerebral artery.

Ischemia of the optic chiasm may produce bitemporal visual loss. Ectatic arterial dilation may cause ischemia through the mechanism of vascular compression and small vessel occlusion. Atherosclerotic plaque formation in the carotid siphon and atherosclerotic narrowing may produce ischemic infarction to the chiasm and pituitary gland.

About 80% of patients with vertebrobasilar arterial stenosis will have ocular signs and symptoms.[18] Transient ischemic episodes involving the vertebrobasilar arterial system produce bilateral blurring or dimming of vision almost as frequently as they do vertigo. Signs and symptoms secondary to ischemia of the brainstem include episodic diplopia, oscillopsia, internuclear ophthalmoplegia, nystagmus, and extraocular muscle palsies.

Ischemia in the distribution of the posterior cerebral artery can produce transient homonymous hemianopia or transient blindness, if it occurs bilaterally. The pupillary responses are retained.

An isolated homonymous hemianopia of sudden onset is the hallmark of vascular disease in the occipital lobe that usually involves the posterior cerebral artery. Homonymous quadrantanopia, crossed-quadrant hemianopia, homonymous scotomas, or constriction of one or both visual fields may also occur.

Causes of transient vascular insufficiency involving the vertebrobasilar arterial system include changes in cardiac output and regional blood flow, hypertensive vascular disease causing slow reactivity of the brainstem and cortical arterioles, anema and polycythemia, microembolization, and atheromas in the vertebral or basilar arteries. Mechanical factors can also restrict the flow of blood in the vertebral arteries, including compromise of blood flow produced on head movement by cervical spondylosis and kinking, tortuosity, as well as redundancy of the extracranial portion of the vertebral arteries.

Other Causes of Occlusive Disease

Aortic arch disease may produce signs and symptoms related to the carotid and vertebrobasilar arterial systems, including ocular, cerebral, and brainstem signs.

As already discussed, occlusion or decreased perfusion of the internal carotid artery may result in an ipsilateral decrease in vision or a decrease in ophthalmic artery pressure and contralateral hemiplegia. Amaurosis fugax is the most common symptom of carotid artery insufficiency, occurring in about 40% of the cases, although it is nearly always absent after a completed stroke. About 85% of the patients with amaurosis fugax have lower ophthalmic artery pressures on that side. If, however, the loss of vision lasts longer than 20 minutes, the occlusive process is probably in the retina or optic nerve.

When the middle cerebral artery or its branches are involved a contralateral hemiplegia results, with the motor deficit more pronounced in the upper extremities. It is frequently associated with a contralateral hemisensory defect and contralateral homonymous hemianopia. If the lesion is in the dominant cerebral hemisphere aphasia may also be present. When the anterior cerebral artery is involved, there is a contralateral hemiplegia and occasionally a hemisensory defect. The defect is greater in the lower extremity.

When the posterior cerebral artery is involved there may be blurred vision due to a unilateral or bilateral visual field defect, which is frequently a contralateral homonymous hemianopia, and, in some cases, a concomitant hemisensory deficit. When other branches of the basilar artery are involved there are episodes of vertigo, blurred vision, or diplopia. There may be bilateral involvement of motor and sensory functions, slurred speech, and lack of coordination. Cranial nerve abnormalities and nystagmus may also occur.

The posterior inferior cerebellar artery or vertebral artery syndrome, characterized by defective sensation on the ipsilateral side of the face and of the contralateral extremities, is associated with ipsilateral palate and vocal cord paralysis, dysphagia, ipsilateral Horner's syndrome, ipsilateral ataxia, and nystagmus.

When the anterior inferior cerebellar artery is involved there may be ipsilateral impairment of hearing, facial paralysis, Horner's syndrome, loss of feeling on the face on the same side as the lesion, nystagmus, and ataxia. In addition, pain and temperature sensation may be impaired in the contralateral arm, leg, and occasionally the face. Insufficiency in the superior cerebellar artery is characterized by ataxia, involuntary movements, and loss of pain and temperature sensation in the contralateral half of the body, including the face.

Vertebral artery occlusion can produce episodes of vertigo with unsteadiness or falling. The syndrome of decreased blood flow through one or both posterior cerebral arteries, with resulting homonymous hemianopia, may also be present. Turning the head and neck or compressing the carotid artery may induce nystagmus.

Almost 40% of strokes result from atheromatous occlusion with thrombosis. The remaining 60% include embolic phenomena (25%), hypertensive intracerebral hemorrhage (20%), ruptures of aneurysms, angiomas, and other congenital malformations, and intracerebral hemorrhage secondary to blood dyscrasias such as polycythemia, anemia, hemophilia, and anticoagulant therapy (15%).

About 50% of the patients with intermittent insufficiency in the carotid or vertebrobasilar system will have a serious stroke in an average of three years. Another 25% will not sustain a stroke but will continue to have ischemic episodes. The remaining 25% will recover because collateral circulation has been established.

It is hoped that recognition of the signs and symptoms of cerebrovascular disease will enable the clinician to achieve the goal of stroke prevention.

REFERENCES

1. Hollenhorst RW: Ophthalmologic aspects of stroke, in *Symposium on Neuro-Ophthalmology*. Transactions of the New Orleans Academy of Ophthalmology. St. Louis, Mosby, 1976, pp 255–267.
2. Hollenhorst RW: Carotid and vertebral-basilar arterial stenosis and occlusion: neuro-ophthalmologic considerations. *Trans Amer Acad Ophthal Otolaryng* 1962; 66:166–180.
3. Fisher CM: Observations of the fundus oculi in transient monocular blindness. *Neurology* 1959; 9:333–347.
4. Hollenhorst RW: Significance of bright plaques in the retinal arterioles. *JAMA* 1961; 178:23–29.
5. Lesser RL, Heinemann MH, Borkowski Jr H, Cohen LS: Mitral valve prolapse and amaurosis fugax. *J Clin Neuro Ophthalmol* 1981; 1:153–160.
6. Hedges TR Jr: Ophthalmoscopic findings in internal carotid artery occlusion. *Bull Hopkins Hosp* 1962; 111:89–97.
7. Kearns TP, Hollenhorst RW: Venous-stasis retinopathy of occlusive disease of the carotid artery. *Proc Mayo Clin* 1963; 38:304–312.
8. Little HL: The role of abnormal hemorrheodynamics in the pathogenesis of diabetic retinopathy. *Trans Am Ophthalmol Soc* 1977; 74:573–636.
9. Kearns TP, Siekert RG, Sundt Jr TM: The ocular aspects of bypass surgery of the carotid artery. *Mayo Clin Proc* 1979; 54:3–11.
10. Hollenhorst RW: Ocular manifestations of insufficiency or thrombosis of the internal carotid artery. *Trans Am Ophthalmol Soc* 1958; 56:474–506.
11. Smith JL: Unilateral glaucoma in carotid occlusive disease. *JAMA* 1962; 182:683–684.
12. Sundt Jr TM, Siekert RG, Piepgras DG, Sharbrough FW, Hauser OW: Bypass surgery for vascular disease of the carotid system. *Mayo Clin Proc* 1976; 51:677–692.
13. Kearns TP, Younge BR, Piepgras DG: Resolution of venous stasis retinopathy after carotid artery bypass surgery. *Mayo Clin Proc* 1980; 55:342–346.
14. Neupert JR, Brubaker RF, Kearns TP, Sundt Jr TM: Rapid resolution of venous stasis retinopathy after carotid endarterectomy. *Am J Ophthalmol* 1976; 81:600–602.
15. Kirby Jr TJ, Hollenhorst RW: Asymmetric hypertensive retinal changes after ligation of the internal carotid artery. *Acta Conc Ophth* 1954; 17:335–340.
16. Furlan AJ, Whisnant JP, Kearns TP: Unilateral visual loss in bright light. An unusual symptom of carotid artery occlusive disease. *Arch Neurol* 1979; 36: 675–676.
17. Donnan GA, Sharbrough FW, Whisnant JP: Carotid occlusive disease. Effect of bright light on visual evoked response. *Arch Neurol* 1982; 39:687–689.
18. Kearns TP: Ocular manifestations of vertebral-basilar arterial thrombosis. *Med Clin North Am* 1960; 44:909–915.

The role of the Medical Society of the State of New York in workers' compensation

JOHN H. MORTON, MD

The New York State Workers' Compensation Law directs the Medical Society of the State of New York (MSSNY) to participate in a defined but limited way in the workers' compensation system in the state. MSSNY policy on all issues, including workers' compensation, is established by the House of Delegates and implemented by the Council of MSSNY. In matters of workers' compensation the Council is advised by a standing committee—the Council Committee on Workers' Compensation and Occupational Health. The state medical society and its component county medical societies are directly involved in compensation matters in three ways: physician ratings; the fee schedule; arbitration of complaints by doctors and hospitals about payment for services. In addition, the county medical society is empowered to "investigate, hear and make findings with respect to all charges as to professional or other misconduct of any authorized physician."

PHYSICIAN RATINGS

When a physician with a New York State license enters practice, he or she is eligible to apply for a compensation rating. This rating is granted by the chairman of the New York State Workers' Compensation Board, acting on the advice of the county medical society that screens the application. The county society recommends authorization either as a general physician or as a specialist or consultant in a given field.

The state medical society has established guidelines for assigning a specialty rating: an individual who has completed the training required by a specialty board is eligible for rating as a specialist in the field. Except under unusual circumstances, however, the applicant for a consultant's rating must be certified by one of the 23 specialty boards recognized by the American Board of Medical Specialties or hold a certificate of special competence issued by one of these boards. Certificates of special competence involve additional training and a written examination after initial board certification, and they are awarded in subspecialty areas such as cardiology or pulmonary medicine. If a county does not have a board-certified physician in a given specialty, the county society may recommend consultant status for an individual with extensive experience or training in the field, even if he or she is not board certified. The compensation board usually requests advice from the state medical society concerning the appropriate rating under these exceptional circumstances. A general physician receives the code letter X, followed by his authorization number. The specialist is assigned a code letter according to his field—for instance, A for general surgery or J for internal medicine. The consultant is assigned two code letters—for example, SA for general surgery and SJ for internal medicine.

In New York State the injured worker is entitled to a choice of physician within the group authorized by the chairman of the Workers' Compensation Board to provide care for patients with compensable problems. The situation in this state differs from that in 23 other states, where the employer or the state, rather than the employee, selects the physician; the patient is not given a choice.

Although every effort is made to keep the application procedure for a compensation rating simple and to expedite the process, some physicians object to the rating on general principles. These physicians argue that the compensation rating is a form of secondary licensure that is required in addition to the state license to practice medicine and surgery. They point out that in some states where a compensation rating is not required any licensed physician may treat a worker with a compensable injury. Before 1935 New York State had a panel of physicians selected by employers or their insurance companies to treat injured workers; the rating system was developed as a means of permitting all qualified physicians to participate and of allowing the patient to choose his own doctor. Since a rating is awarded only after the physician has satisfied training requirements in a field, and since it may be revoked or suspended for misconduct, the rating assures the employee that the physician selected has the requisite knowledge and experience to treat a particular injury or condition.

FEE SCHEDULE

Payment for care of injured workers in New York State is determined by a fee schedule that is issued by the chairman of the New York State Workers' Compensation Board.

Dr. Morton is the chairman of the Committee on Worker's Compensation and Occupational Health of MSSNY.

Address correspondence to Dr Morton, Professor, Department of Surgery, University of Rochester Medical Center, 601 Elmwood Ave, Rochester, NY 14642.

This schedule is revised periodically to include new procedures and to delete obsolete ones, to modify relativity between procedures, and to reflect changes in the cost of medical practice. A physician who cares for an injured worker bills the employer or the employer's insurance carrier for services rendered. According to the law, the doctor is not permitted to present a bill to the patient for a work-related illness or injury.

The compensation rating is important because the schedule provides larger payment to a specialist or consultant than to a general physician for office visits, hospital visits, and home visits. The schedule also provides higher fees for consultants in certain fields. For instance, certified diagnostic radiologists are paid more than other physicians for roentgenographic interpretation. The items listed in the schedule—the diagnostic and therapeutic procedures considered pertinent for compensable injuries—are handled in a different fashion. Any physician in a given region of the state who carries out one of these procedures is reimbursed at the same rate as any other physician in the region without regard to specialty rating. For instance, all physicians in a given region receive the same fee for interpreting an electrocardiogram or managing a Colles' fracture.

Section 13 of the New York State Workers' Compensation Law defines the role of MSSNY in determining payment for medical care of industrial illness and injury as follows:

> The chairman shall prepare and establish a schedule for the state, or schedules limited to defined localities, of minimum charges and fees for such medical treatment and care, to be determined in accordance with and to be subject to change pursuant to rules promulgated by the chairman. Before preparing such schedule for the state or schedules for limited localities the chairman shall request the president of the medical society of the state of New York to submit to him a report on the amount of remuneration deemed by such society to be fair and adequate for the types of medical care to be rendered under this chapter, but consideration shall be given to the view of other interested parties. The amounts payable by the employer for such treatment and services shall in no case be less than the fees and charges established by such schedule. Nothing in this schedule, however, shall prevent voluntary payment of amounts higher than the fees and charges fixed therein, but no physician rendering medical treatment or care may receive payment in any higher amount unless such increased amount has been authorized by the employer, or by decision as provided in section thirteen-g herein.

An Advisory Committee on the Medical Fee Schedule and Allied Problems makes suggestions to the chairman concerning compensation matters. MSSNY nominates five physicians to serve on this committee, and the state has always accepted these nominees. Other members represent the compensation board, the insurance industry, the self-insured, employers, and organized labor. Whenever the fee schedule is revised, the members of the committee attempt to reach a consensus on a proposal for the chairman to consider. Recommendations of the committee are purely advisory. The ultimate decision rests with the chairman, who, acting as the agent of the state, issues the schedule.

In the mid-1970s any fee negotiation by organized medicine that was based on a relative value schedule was stopped by threats from the Federal Trade Commission, claiming that such a schedule represented restraint of trade. It was eventually decided, however, that use of a relative value schedule when dealing with a governmental agency was not illegal. During the schedule revision process that led to a new fee schedule in 1985, the House of Delegates of MSSNY instructed the physicians on the advisory committee to negotiate only for usual and customary fees. Fees established in this manner were unacceptable to the chairman. He directed an ad hoc committee to update the fixed fee schedule, and the ad hoc committee reported the results of its deliberations to the advisory committee. However, because of MSSNY's position, the physicians on the advisory committee could not participate in the revision. They were permitted to make suggestions concerning the inclusion of new procedures and the elimination of obsolete ones. The House of Delegates changed MSSNY's positon during the 1983 annual meeting, and the physicians on the advisory committee will engage in negotiations during future fee schedule revisions.

Apart from general concerns about a fixed fee schedule, the low level of payment, the slow process of modifying the schedule, and the need for prior approval for elective procedures, physician complaints about the schedule involve three particular items in Section 13.

First, the section establishes a schedule of "minimum charges and fees." This provision was introduced in the 1930s to control price competition among physicians and to prevent restrictive arrangements between doctors and employers during the Depression. In the past, this provision has been accepted as a fixed maximum payment except under unusual circumstances when, with explanation, a higher fee can be sought. More recently some physicians have interpreted this to mean that any charge at or above the fee schedule is legal. The insurance carriers and the board have disagreed with this position, and a good deal of acrimonious discussion has resulted. MSSNY representatives believe that the schedule is established for usual procedures, and that unusual effort can and should be compensated at a higher level. Under these circumstances, higher payment may be made voluntarily by the carrier, or it may result from the decision of an arbitration panel.

Second, the section refers to "such schedule for the state or schedules for limited localities." Prior to 1980 the chairman established a single schedule for the whole state. In that year, in response to continuing complaints that the level of reimbursement was disproportionately low in comparison with usual fees for doctors in the New York City area, the chairman divided the state into four regions, using a statewide relative value schedule with a different conversion factor for each region. This change, which has resulted in payments that are significantly higher in New York City, Long Island, and Westchester County than they are elsewhere in the state, has had a mixed reception from physicians. Doctors in the metropolitan area agree with regionalization of fees, although they complain that they remain underpaid for compensation work. In establishing a regionalized schedule the chairman was making allowance for the greater practice expenses encountered by physicians in the metropolitan area. However, doctors upstate do not interpret the schedule in this way. They believe that they are not afforded equal treatment by a program that reimburses them at a lower rate than metropolitan physicians for comparable medical care. Doctors in rural areas are particularly concerned because of the problem this disparity creates when recruiting new physicians for smaller communities. After giving consideration to these objections, MSSNY's majority position favors regionalization of fees.

Third, the section states that the chairman will request an opinion from the president of MSSNY about a fair and adequate fee schedule, "but consideration shall be given to the view of other interested parties." The state has always interpreted this to mean the views of non-medical groups such as industry and labor. In recent years certain of the medical specialties have argued that this provision gives them the right to deal directly with the state, outside the medical consensus sought through MSSNY. To date, the chairman has rejected this interpretation, and MSSNY representatives do not believe this approach is in the best interests of the medical profession as a whole. It is important to understand the reason for the MSSNY attitude: the amount of money available for physician payment with any revision of the schedule is finite, and any increase for doctors in one specialty will be accompanied by a decrease in payment for physicians in other fields of practice.

ARBITRATION OF COMPLAINTS

In an emergency any licensed physician may treat an ill or injured worker without authorization, and issues of compensability are decided later. In elective situations care is rendered by a physician with a compensation rating after authorization is obtained.

Under most circumstances there is little disagreement between doctors and hospitals on the one side and the insurance carriers and the self-insured on the other. Many of the disagreements that do arise could be prevented by adequate initial documentation of the diagnostic and therapeutic measures carried out by the doctor or the hospital. When there is an argument about compensability, however, an administrative law judge makes the decision after a hearing on the issues. The judge also decides about the degree and duration of disability in contested cases.

Once compensability is established, the physician who renders care submits a bill. A clear written statement with an adequate description of the care is invaluable in justifying the claim. If the carrier does not object to the bill, by filing an A-1 (Notice to Chairman, Workers' Compensation Board, Objection to Bill for Treatment) within 30 days, the bill is due and payable. If the carrier does object, the physician has the right to request arbitration by filing an MR-54 (Application for Award Under Workers' Compensation Law) with the board.

Arbitration issues are heard by a panel that consists of three physicians: one member appointed by the compensation board, one appointed by the carriers, and one appointed by the county medical society or the district branch. A separate committee deals with hospital complaints, and the hospital association appoints the third physician under these circumstances. After reviewing C-48's (Attending Doctor's First Report), C-4's (Attending Doctor's Progress Report), and narrative reports by the physician or the hospital, the panel listens to oral arguments advanced by the doctor or the hospital and the carrier. At many of these hearings the carrier is represented by an expert in compensation law and regulations. The doctor or the hospital may decide not to attend the hearing at all, or they may be uninformed and poorly prepared. Attendance by the physician at the hearing is not mandatory, but it does help the panel if the record is confusing. After the hearing the committee reaches a conclusion about the appropriate level of payment, and the decision is binding on both parties. Occasionally, MSSNY receives a complaint from a doctor who is unhappy with the results of arbitration. Under these circumstances the society replies that the doctor has been heard by a jury of peers who were not convinced by the argument he advanced.

CONCLUSION

When New York State passed a workers' compensation law in 1914, it was one of the first states to protect its workers. The present system is increasingly expensive, which is of concern to employers and insurance carriers in the state. Physicians who treat patients with work-related illness or injury encounter problems periodically with carriers or with the board. Some patients complain that their disability payments are too slow, even when reports are filed in a timely fashion. It is generally agreed that the program must be continued and improved. MSSNY and its component county medical societies have a voice, albeit a limited one, in establishing the policies and procedures in the state. It is important to all parties that this role be preserved.

A 39-year-old man with thrombocytopenia, cough, fever, and shaking chills

CASE PRESENTATION

Evan Fliegel, MD (Medical Intern): A 39-year-old man was admitted for the second time, with chief complaints of increasing weakness, cough, fever, and shaking chills for the preceding two weeks.

The patient had been healthy until one month prior to the first admission, when he sought dental attention because of bleeding gums. On referral to a hematologist, his platelet count was found to be 1,000/mm^3; after admission to the Long Island Jewish-Hillside Medical Center, the bone marrow examination revealed a hypercellular marrow with increased numbers of megakaryocytes. A liver-spleen scan showed splenomegaly, with normal liver size. The patient was treated with platelet transfusions, steroids, and plasmapheresis. He responded well, with an increase in the platelet count to 130,000/mm^3. He was discharged on a regimen of prednisone, 20 mg four times daily, which was to be reduced gradually if the platelet count continued to increase.

He experienced a 10-lb weight loss during the month prior to his second admission; 10 days earlier, he had noted increased weakness, shaking chills, and cough productive of yellowish sputum occasionally flecked with blood. The patient had continued to take steroid medication, flurazepam hydrochloride, and diazepam. He denied any chest pain, shortness of breath, or history of pulmonary disease. He admitted to heavy alcohol intake and intravenous heroin abuse, denying any such injections, however, during the six weeks prior to his second admission.

His medical history was remarkable only for injuries sustained in an automobile accident 10 years previously, for which surgical intervention and blood transfusions were necessary. He had smoked two packs of cigarettes per day for 20 years. He was heterosexual and was married with two children. The family history was noncontributory.

Physical examination at the time of the second admission revealed an ill-appearing man in mild respiratory distress. Blood pressure was 110/60 mm Hg; pulse rate, 130/min and regular; respiratory rate, 40/min; and temperature, 106°F when taken in the emergency department and 102.4°F in the inpatient unit. The patient was diaphoretic; his cough was productive of sputum tinged with bright red blood. The funduscopic examination was not remarkable; the neck was supple. No adenopathy was evident. There was an old thoracotomy scar over the right lateral chest wall. Examination of the lungs revealed scattered rhonchi with occasional expiratory wheezes over both lung fields. The point of maximal cardiac impulse was in the fifth intercostal space in the mid-clavicular line; the heart sounds were normal, with no murmurs, rubs, or gallops. The abdominal and rectal examinations disclosed no abnormalities. The stool was positive for occult blood on guaiac test. The skin showed purpuric areas around the knees. The findings on neurologic examination were within normal limits.

The hemoglobin was 13.9 g/dL, and the hematocrit value was 39.3%. The white blood cell count was 4,000/ml^3, with a differential count of 53% polymorphonuclear leukocytes, 6% band forms, 27% lymphocytes, 1% atypical lymphocytes, 3% monocytes, and 1% metamyelocytes. The platelet count was 27,000/mm^3 (normal range, 150,000 to 350,000/mm^3). The mean corpuscular volume was 89μ^3 (normal range, 80 to 94μ^3). The prothrombin time was 13.7 sec (control, 11.2 sec); the partial thromboplastin time was 42.6 sec (control, <40 sec). The electrocardiogram showed no abnormalities; the ventricular rate was 90/min.

A chest film showed a bilateral pattern of diffuse interstitial infiltration (Fig 1).

The patient was treated with tobramycin, oxacillin, ticarcillin, isoniazid, rifampin, and intravenous trimethoprim-sulfamethoxazole.

Approximately 12 hours after the patient came to the emergency department his breathing became more labored, with increased rhonchi and wheezing, as well as a worsening alveolar-arterial oxygen gradient. Arterial blood gas determinations, with the use of a 100% oxygen non-rebreather, indicated an arterial pH of 7.21, a P_{CO_2} of 50 mm Hg and a P_{O_2} of 82 mm Hg. An endotracheal tube was inserted. On assisted control (fraction of inspired oxygen at 100%, ventilatory rate of 12/min, and tidal volume of 700 mL), the arterial blood gas determination showed a pH of 7.23, a P_{CO_2} of 40 mm Hg, and a P_{O_2} of 109 mm Hg. Moderate amounts of blood-tinged sputum drained from the endotracheal tube. Approximately four hours after intubation, cardiorespiratory arrest occurred, and the patient could not be resuscitated.

CLINICAL DISCUSSION

Carol Singer, MD (Chief, Division of Infectious Diseases,

Based on the clinicopathologic conference (CPC) held at the Long Island Jewish-Hillside Medical Center, New Hyde Park, NY, on February 17, 1984.

Moderator: Herbert S. Diamond, MD, Chairman, Department of Medicine; *Clinical Discussion:* Carol Singer, MD, Chief, Infectious Disease Division, Department of Medicine; *Pathology Presentation:* Stanley Lipper, MD, Director, Department of Laboratories, Queens Hospital Center, Long Island Jewish-Hillside Medical Center Affiliate.

Editor: JOHN T. FLYNN, MD

Address correspondence to Dr Flynn, Consulting Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Rd, Lake Success, NY 11042.

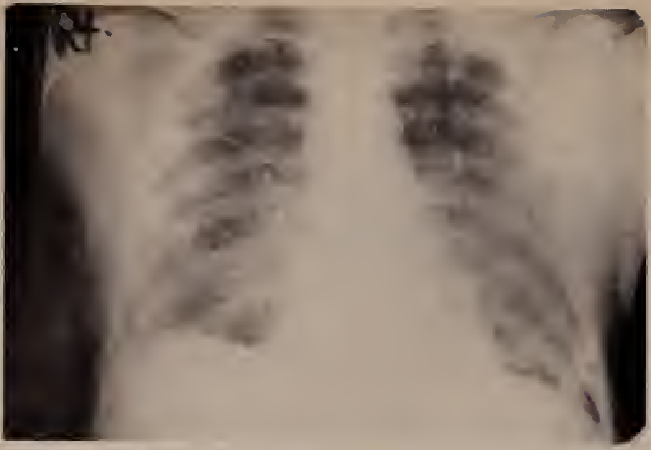


FIGURE 1. Chest film obtained on admission showing diffuse interstitial and intra-alveolar exudates.

Department of Medicine): Before beginning my general discussion, I would like to comment on the available chest films. The films taken on the first admission showed what seemed to be a pleural reaction or effusion in the lower right lung field; however this might have been related to the previous automobile accident. The heart size appeared normal. The only films from his second and final admission again showed the reaction at the base of the right lung, and a diffuse bilateral interstitial pattern of infiltration without hilar adenopathy and with normal cardiac size (Fig 1).

I have several questions for Dr Fliegel before presenting the major part of my discussion. During the first admission, was there any attempt to evaluate this pleural reaction at the base of the right lung?

Dr Fliegel: No, not during that admission.

Dr Singer: Did he have a purified protein derivative test for tuberculosis during that first admission prior to initiation of steroid therapy?

Dr Fliegel: A purified protein derivative test was ordered during his first admission, but I do not recall the results of the test.

Dr Singer: Patients who are given therapy that could have an immunosuppressive effect need to be evaluated for any evidence of active tuberculosis before such therapy is initiated. We know that this patient smoked cigarettes for many years, but do we know if he had evidence of any other pulmonary disease?

Dr Fliegel: No, we have no such history.

Dr Singer: Do we know if any immunologic work-up was performed on his first admission, in terms of T-helper cells or similar tests?

Dr Fliegel: Not to my knowledge.

Dr Singer: What was his white blood cell count on the first admission?

Dr Fliegel: His total white blood cell count was 3,900/mm^3 with 23% neutrophils, 3% band forms, 52% lymphocytes, 1% monocytes, 8% atypical lymphocytes, 1% metamyelocytes, and 2% myelocytes.

Dr Singer: Do we know if the dosage of his steroid medications was gradually reduced in the interval between his two admissions?

Dr Fliegel: We do not have that information.

Dr Singer: On his final admission, his heart size looks normal; apparently he did not have a Swan-Ganz catheter inserted. Were there signs of congestive heart failure?

Dr Fliegel: It was difficult to determine; his terminal episode was so abrupt that we did not have an opportunity to fully evaluate any evidence of congestive heart failure.

Dr Singer: Were the skin lesions around the knees actually purpuric or were they nodular-type infiltrates?

Dr. Fliegel: They were ecchymoses. I did not see any nodular infiltrations of the skin.

Dr Singer: I have asked these questions only because such information might be helpful in trying to evaluate what was wrong with this patient. A first look at this protocol impressed me with the thought that the case might be quite straightforward; subsequently, however, I was less certain as to the final diagnoses.

The patient had a relatively normal medical history, except for his drug and alcohol abuse. He was admitted initially for acute development of thrombocytopenia. Evaluation suggested that the findings were consistent with idiopathic thrombocytopenic purpura, with peripheral destruction of platelets and an increase in normal megakaryocytes in the bone marrow. One of the problems that I had with the diagnosis of idiopathic thrombocytopenic purpura was the presence of splenomegaly, evident on the liver-spleen scan. However, this might be attributable to his alcohol abuse and other factors, since splenomegaly is not generally associated with idiopathic thrombocytopenic purpura alone. Drugs can also induce thrombocytopenia, but the patient was not taking any of the drugs that commonly cause this condition. In addition, such a reaction should diminish after the discontinuance of any such medications. Thus we are left with a patient in whom a diagnosis of idiopathic thrombocytopenic purpura was made, but who had an unexplained right pleural effusion or reaction that may or may not be important. Certainly, the pleural reaction could have been long-standing and related to earlier chest trauma at the time of the automobile accident 10 years previously.

At present, we are all intensely aware of acquired immunodeficiency syndrome (AIDS), and I suspect that when this patient first entered the hospital, this diagnosis was strongly considered even though there was no definitive evaluation of his lymphocyte status at that time. Thrombocytopenia is not a common sign of AIDS, although a report in 1982 described 11 homosexual patients with multiple partners who presented with thrombocytopenic purpura.[1] Platelet counts in these patients ranged from 3,000 to 29,000/mm^3, with increased megakaryocytes in the bone marrow and with no splenomegaly. These patients had negative antinuclear antibody tests; they all responded to steroid therapy, although three of them required splenectomy. Immunologic studies showed that four of five of these patients had a decreased lymphocyte helper-to-suppressor ratio, a finding that is not limited to AIDS, but nevertheless is characteristic of the disease. Three of four patients tested had an absolute decrease in the total number of circulating helper lymphocytes, which is the immunologic hallmark of AIDS. Overall, these patients were not lymphopenic, and no clinical follow-up was given in the report. Therefore, we do not know if either Kaposi's sarcoma or the opportunistic infections characteristics of AIDS subsequently developed.

Other aspects of thrombocytopenia that I would like to emphasize include the multitude of causes of the condition. Tuberculosis has to be mentioned, because a variety of immunologic abnormalities can accompany that infection. We are most familiar with pancytopenia in tuberculosis, with or without granulomas in the bone marrow; and granulocytopenia or a leukemoid reaction can occur. We must always think of tuberculosis in the presence of atypical disease processes. Pancytopenia and thrombocytopenia may also be found in disseminated histoplasmosis. In this regard, it would be helpful to know more about this patient's background: where he was born, and whether he had lived in New York all of his life, or whether he had lived in other parts of the country.

In most of these conditions, however, thrombocytopenia has not been particularly common, except after the use of trimethoprim-sulfamethoxazole. In these patients, leukopenia and thrombocytopenia have not usually been the initial symptom prompting the patient to seek treatment. Endocarditis and vasculitis may also be associated with thrombocytopenia. In systemic lupus erythematosus, thrombocytopenia may precede other manifestations of the disease, and pleural effusions may be present. This patient apparently had no other manifestations of vasculitis, and he had a negative antinuclear antibody test.

I would like to put aside the diagnosis of thrombocytopenia for the moment and focus on the subsequent clinical events. Again, I think the differential diagnosis is complicated by the fact that this patient apparently was on a regimen of high-dose prednisone when his acute respiratory insufficiency developed. His respiratory condition progressed rapidly, unusually so for most infectious processes in the lung. We know that he had been ill for 10 days with fever, chills, and a cough productive of blood-tinged sputum; but, we do not really know his previous condition. Perhaps he always had a smoker's cough. His mild respiratory insufficiency on admission progressed rapidly to a severe, and indeed, terminal state within 12 hours. We therefore should consider the adult respiratory distress syndrome (ARDS). In that condition there is usually a normal pulmonary wedge pressure. ARDS involves non-cardiac pulmonary edema, secondary to increased capillary endothelial leakage, with hypoxemia that can progress rapidly. The infiltrate can be extensive and of the interstitial type. We have no subsequent films on this patient to determine how his pulmonary picture progressed.

There is a variety of conditions that predispose a patient to ARDS, most of which did not apply to this patient. The more common predisposing situations include Gram-negative bacteremia, which this patient may very well have had, associated with high temperature and chills. We do not know the results of the blood cultures obtained at the time of admission. Bypass surgery requiring multiple blood transfusions is another cause of ARDS; this patient was given a transfusion during his first admission but not immediately before the terminal events of his illness. There is no evidence of disseminated intravascular coagulation, although he did have a slightly increased prothrombin time; presumably the thrombocytopenia seen during the second admission was related to whatever process caused it earlier.

Pneumonias, which require intensive care, are perhaps one of the most common conditions associated with ARDS. These are usually diffuse viral pneumonias, such as influenzal pneumonia, which this patient may have had. However, there were no signs of a typical influenza-type prodrome—with severe myalgias, dry cough, and rapid progression of x-ray findings to a diffuse interstitial pattern of pulmonary infiltrate. There also was no evidence that his pulmonary findings were the result of aspiration. The fact that this patient was an intravenous drug abuser raises suspicions concerning his claim that he had not injected drugs during the preceding six weeks. In any event, neurologic signs of drug overdose were not evident at the time of his admission.

Certain metabolic disorders, as well as pancreatitis and uremia, are not considerations as possible causes of ARDS in this patient, nor is increased intracranial pressure. His possible predisposition to ARDS could be secondary to bacteremia, and, again, with a drug user one must consider the possibility of endocarditis. He may have had disseminated intravascular coagulation, and his pneumonia may have been due to a virus such as influenza. There was no evidence for a varicella pneumonia, which is associated with a characteristic rash.

ARDS has been reported with miliary tuberculosis, which is one of my chief concerns in this patient. Miliary or disseminated tuberculosis is recognized much more often now in the older age groups, whereas in the past it was described primarily in children. Alcohol abuse is a common underlying disorder in disseminated tuberculosis, and this patient was an abuser of alcohol. However, he did not present with the kind of prodrome that you would expect with disseminated tuberculosis, although he had weakness, weight loss, and fever for several weeks. Many patients with disseminated tuberculosis will have the condition for many weeks or even months; in many, there is no prior history of tuberculosis. The physical findings are variable with this type of tuberculosis—lymphadenopathy in about 15% to 30% of patients, hepatomegaly in about 30%, and splenomegaly in about 10%. The liver-spleen scan showed splenomegaly, which was not otherwise explained. The hematologic findings in disseminated tuberculosis are also variable: most patients have a normal white blood cell count, but there can be leukemoid reactions or leukopenia as well as thrombocytopenia or pancytopenia. The name "miliary tuberculosis" originates from the fact that the typical chest film shows shadows reminiscent of tiny millet seeds, not the interstitial pattern that we saw in this patient's films.

Two studies in the 1970s[2,3] described patients with miliary tuberculosis and a typical type of pulmonary infiltration. Ten to twenty percent of these patients had concomitant pleural effusion, which may be the case in this patient, although the findings in the lower right side of the chest may be the result of the earlier motor vehicle accident. It is difficult to ignore the possibility of a recent pleural reaction given the presence of a progressive diffuse interstitial type of pneumonia. We have no report on this patient concerning the results of a purified protein derivative test for tuberculosis; however, a negative test certainly would not have ruled out the diagnosis of disseminated tuberculosis. Miliary tuberculosis is an uncommon cause of ARDS, although there are several reports in the literature indicating such an association[4,5]; these occurred most often in patients with alcoholism who demonstrated reticular-nodular and alveolar infiltrates on chest films. Many of these patients were negative for purified protein derivative on admission to the hospital. Interestingly, in seven of the eight patients described, disseminated intravascular coagulation developed during their clinical courses. Again, although we tend to think of patients with tuberculosis as being anemic, the hematocrit values of these patients with both miliary tuberculosis and ARDS ranged from 39% to 45%. In our patient, the initial hematocrit value was 39%; however, he had received transfusions earlier, so that we do not know what his true baseline hemoglobin and red cell count levels would have been. We cannot rule out the diagnosis of tuberculosis in this patient, and we need to be intensely aware of this diagnostic possibility, since the condition is treatable.

I must mention the possibility of endocarditis, particularly in a drug abuser who has a febrile illness. No heart murmur was heard in this patient, but that is true in about 10% of patients who have right-sided endocarditis. If this patient had such a condition, the findings on the chest roentgenogram would suggest bilateral septic pulmonary emboli. A patient with endocarditis may have congestive heart failure, but there is no evidence that this patient was in congestive failure. Thrombocytopenia is found in about 10% of patients with endocarditis; leukopenia, however, is relatively uncommon and when present is usually associated with significant splenomegaly. Again, most patients with endocarditis are anemic, which was not true here.

The organisms that cause endocarditis in drug users are usually different from those in the non-user population; there are also regional differences. On the East Coast, *Staphylococcus aureus* is probably the most common organism, whereas *Pseudomonas* is prevalent in Detroit, and *Serratia* is found on the West Coast. *Candida* and *Staphylococcus epidermidis* can also be associated with endocarditis in these patients. One could postulate that the patient's thrombocytopenia, splenomegaly, and fever were the result of endocarditis, and that ultimately ARDS developed secondary to sepsis. But we still remain uncertain.

I do not think this patient had desquamative interstitial pneumonitis, which is a more chronic process. Neither collagen vascular disease nor vasculitis appear to be likely possibilities. Certain viral pneumonias could be consistent with the final clinical picture, but again the patient did not have the rash that usually accompanies measles or varicella; influenza remains a possibility. Adenoviruses occasionally cause a diffuse roentgenologic picture such as is seen in this patient's lungs.

Finally, the main point of this case may be the presence of AIDS and the pulmonary processes that may accompany that syndrome.[6,7] The Centers for Disease Control defines AIDS as a disease that is at least moderately predictive of a defect in cell-mediated immunity that occurs in the absence of a known cause for diminished resistance. This is a clinical diagnosis. The fact that this patient was on a regimen of high-dose prednisone perhaps keeps us from labeling his condition as AIDS. Complications of AIDS include Kaposi's sarcoma, pneumocystis pneumonia, and other opportunistic infections. What aspects of AIDS did this patient present? First, there is no definitive laboratory test to diagnose AIDS; however, there are findings that are relatively consistent in patients in whom classic AIDS develops. The white blood cell count often indicates a lymphopenia; this patient's was borderline, with an absolute lymphocyte count of about 1,000/mm^3 on his final admission. He had thrombocytopenia, which has not been characteristic of AIDS; perhaps the syndrome of autoimmune thrombocytopenia will become better recognized as more patients with AIDS are seen. No definitive immunologic study was carried out on this patient to determine his T-lymphocyte status. Patients with AIDS sustain an absolute decrease in the number of helper T-lymphocytes and, as a consequence, have an inverted helper-to-suppressor ratio. This inversion is not confined to patients with AIDS, since it can occur in viral infections—for example, with cytomegalovirus infection. Other in vivo and in vitro immunologic tests might have been done had there been time on his final admission. Patients with AIDS may have elevated levels of serum gamma globulin, although they seem to have a problem handling new antigens. Finally, in terms of AIDS, I was curious to know whether the patient had ecchymoses secondary to thrombocytopenia, or could those lesions have been the early manifestations of Kaposi's sarcoma? Kaposi lesions have been described primarily in homosexual patients, but a few cases have been reported in drug abusers and in Haitians.

This patient did not appear to have the familiar prodrome of AIDS—that is, weeks and usually months of nonspecific malaise, diarrhea, fever, and profound weight loss of up to 60 or 70 pounds. He lost 10 pounds during a period of a relatively acute illness. He did not have generalized lymphadenopathy, although this is not necessary for the diagnosis of AIDS. The final episode of interstitial pneumonia is consistent with the diagnosis of AIDS. A variety of organisms can cause this type of pneumonia. It is impossible to identify the causative agent by looking at the chest roentgenogram. Most patients with diffuse interstitial pneumonia have dyspnea that is usually progressive, together with a dry non-productive cough. Infections are generally found, and pneumocystis pneumonia is the most prevalent infectious disease seen with AIDS. Pneumocystis is a small protozoan that causes diffuse interstitial pneumonia in humans and does not involve other organ systems, as far as we know. Pleural effusions are not seen; that is why I was concerned as to whether the pleural involvement in this patient was at all related to his current illness. Pneumocystis pneumonia in other patient groups is often noted to develop during periods of reduced steroid therapy. Such patients probably have the organism in their lungs already, and when steroid therapy is reduced, the pneumonic reaction appears. It is unusual for an otherwise immunologically normal patient, such as an asthmatic patient receiving steroid therapy, to have pneumocystis pneumonia. The suppressive effects of steroid therapy are insufficient to allow pneumocystis infection to progress in such patients.

How *Pneumocystis* is acquired by human subjects is not known. It is found among animals in nature. Passage from person to person has not been shown, although it is suspected because of the occurrence of small-scale epidemics. If this patient had AIDS, *Pneumocystis* would be the most likely organism to cause this clinical and radiologic picture. Again, I would like to point out that pleural reactions with effusion are not part of an infection with *Pneumocystis*.

The other characteristic infection that must be considered is infestation with *Strongyloides*. We know little about this patient's travels and personal contacts, although *Strongyloides* has been found in the United States. This organism will often lie dormant in the intestine until the advent of steroid therapy, following which—often many months later—a hyperinfection syndrome may develop. Such patients usually have marked intestinal distention, and they often have gastrointestinal bleeding and polymicrobic sepsis, which apparently this patient did not have. The organisms ultimately reach the lungs. This condition should be considered, particularly when a patient is to be started on a steroid regimen and when he or she comes from an area that is endemic for *Strongyloides*. It has been suggested that such patients be treated with thiobendazole for several days because the identification of *Strongyloides* may be difficult, and several stool examinations may not be adequate.

Diffuse toxoplasmosis of the lungs has been reported but is quite rare. Fungal infections that invade the lungs are numerous; those most likely to produce this disseminated picture are cryptococcosis and histoplasmosis. Again, this emphasizes the importance of knowing about the patient's travels. In patients with AIDS, cryptococcosis has been described in the lungs, usually as part of a more disseminated infection, which can involve the meninges, the bone marrow, and a variety of other organ systems. A serum cryptococcal antigen test would have been helpful in this patient, since the test result tends to be positive in disseminated cryptococcal disease. Histoplasmosis infection should be considered if this patient had been in or traveled through the endemic areas of this country. One does not have to travel very far to be in contact with that infective organism. Histoplasmosis can cause this type of pulmonary picture, and many of the patients with histoplasmosis have hepatosplenomegaly, lymphadenopathy, and pancytopenia. Thrombocytopenia is also described. These possibilities highlight the type of historic information needed to make a more complete differential diagnosis.

The virus that is most often associated with these clinical and radiologic findings in the lungs is the influenza virus. Yet this organism would not account for all of the patient's symptoms. Herpes simplex virus has been noted in patients with AIDS, but more often it causes a chronic mucocutaneous type of lesion in the genital or perianal regions rather than a diffuse pneumonia. Cytomegalovirus is a common infecting organism among the general population. Disseminated cytomegalovirus infection is quite common in patients with AIDS. It is a large DNA virus that is found in many bodily secretions including saliva, cervical secretions, and blood. It can produce a variety of disorders including hepatitis, gastrointestinal changes, retinitis, encephalitis, and hematologic abnormalities such as thrombocytopenia. The most serious consequence of this infection is a diffuse type of interstitial pneumonia, which is similar to that seen in this patient, and which has been a serious problem in transplant recipients.

Bacterial infections occasionally may cause an interstitial pneumonia such as we see here, most often a Gram-negative bacteremia associated with ARDS. In addition to *Mycobacterium tuberculosis*, *Mycobacterium avium-intracellulare*, an atypical form, can be a serious problem in patients with AIDS, especially among Haitian patients. It can cause disseminated disease, has been isolated from the blood (which is unusual for mycobacterial infections), and has been found in lymph nodes and other organ systems.

These are the main causes of diffuse interstitial pneumonia in patients with AIDS and, in fact, in the general population. Recently, instances of Legionnaires' disease have also been described in patients with AIDS.

Despite the number of atypical characteristics in this patient, I am fairly sure that he had AIDS with pneumocystis infection. In addition, I suspect that the patient had disseminated tuberculosis—because of the pleural reaction and the splenomegaly—and that ARDS later developed. Perhaps the pleural reaction and the splenomegaly were red herrings and were related to other conditions. If they were, then I think the final pulmonary findings were due to pneumocystis pneumonia. Many patients with AIDS who have a pneumocystis infection also have cytomegalovirus infection.

CLINICAL DIAGNOSES

Diffuse interstitial pneumonia, most probably due to *Pneumocystis carinii*, that progressed to adult respiratory distress syndrome, which was fatal.

Probable acquired immunodeficiency syndrome, secondary to chronic intravenous drug abuse.

Suspected disseminated tuberculosis, possibly due to *Mycobacterium avium-intracellulare*.

Probable disseminated cytomegalovirus infection.

PATHOLOGY PRESENTATION

Stanley Lipper, MD (Director, Department of Laboratories, Queens Hospital Center): This autopsy was performed by the Medical Examiner, so that I did not observe the gross findings personally or select the tissue for sectioning.

At autopsy the elevation of the right diaphragm seemed to be related to previous trauma, with a large surgical scar over the right side of the chest. Also noted were deeply pigmented linear scars in the antecubital fossae, on the forearms, and on the dorsa of the hands, with thick-walled underlying veins.

The pleural cavities contained approximately 200 cc of dark yellow fluid. The lungs together weighed 1,200 g, the normal weight being 800 g; the trachea and bronchi were filled with a thin frothy liquid. The lung tissue was irregularly mottled with dark red firm areas scattered throughout, from which a small amount of viscous frothy fluid could be obtained. The spleen was congested, weighing 320 g.

From the principal tissue sections available one can draw a fairly complete picture of the main pathologic findings. The patient had pneumonia caused by *Pneumocystis carinii*; toxoplasmosis was evident in the lungs, heart, spleen, and brain; cytomegalovirus infection was present in the lungs, and to a much less degree in the spleen. I am unable to offer any comment on the status of the lymph nodes, thymus, bone marrow, or the gastrointestinal tract.

In a fairly representative section of the lung, you can observe the extensive exudate distending all the alveolar spaces, with some thickening of the interstitial tissue of the lung (Fig 2). However, the inflammatory reaction and response in the lung are limited to a few aggregates of lymphocytes and occasional plasma cells. At higher magnification, one can appreciate that this alveolar exudate is bubbly in appearance and contains alveolar macrophages. Macrophages containing hemosiderin have accumulated in the alveolar spaces. This is little evidence for interstitial

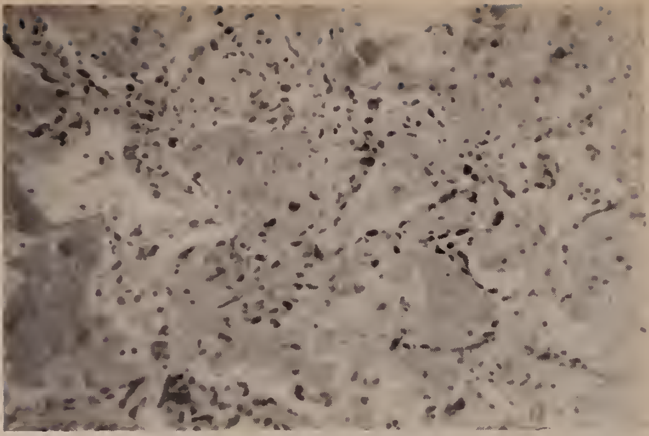


FIGURE 2. Section of lung showing massive intra-alveolar exudate. (hematoxylin and eosin stain; original magnification × 64)

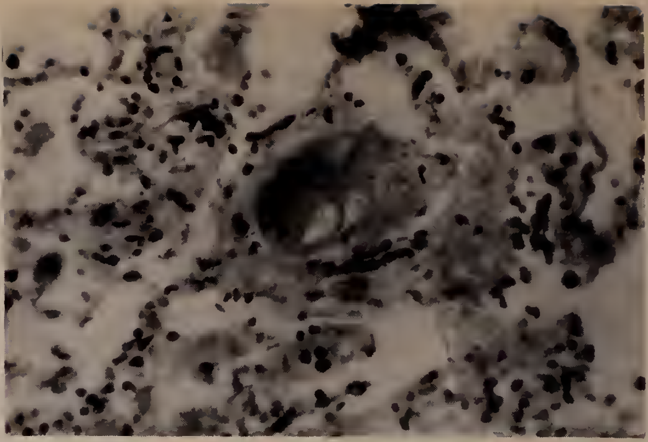


FIGURE 4. Lung tissue showing foreign body giant cell containing refractile material. (hematoxylin and eosin stain; original magnification × 160)

pneumonitis. In addition, intra-alveolar structures can be identified as cytomegalovirus suspended within the exudate, with the resulting juxtaposition of two infections—one being due to *Pneumocystis* and the other caused by cytomegalovirus. The silver stain of the exudate shows the characteristic appearance of *Pneumocystis carinii* cysts: oval to round, with a slightly excavated appearance, rather like a red blood cell viewed on edge (Fig 3). Infrequent, although visible in the lungs, are toxoplasma pseudocysts, which we will see in better detail in other organs. An incidental finding was the presence of foreign body giant cells, attached to the walls of the alveoli and containing refractile material, very likely representing a reaction to intravenous contaminants introduced during self-administered drug injections (Fig 4).

Sections of the myocardium demonstrate the presence of toxoplasma pseudocysts within the myocardial fibers; higher magnification revealed the multiple tiny dots that represent trophozooites within the pseudocyst (Fig 5). The pseudocyst ruptures and destroys much of the myofiber (Fig 6); however, there is little reaction to the presence of these organisms in the myocardium, either due to a diminished immune response or, more likely, to a subclinical chronic infection.

The spleen showed a paucity of white pulp areas and few lymphoid follicles. The red pulp areas were congested and contained few cells, with some lymphocytes and plasma cells, indicating a depleted spleen. Within the spleen only rare cytomegalovirus inclusion bodies were visible.

Although the brain showed no external features of note, it was peppered with tiny petechial hemorrhages and collections of microglial cells, which are brain macrophages. Surrounding some vessels were nodular collections of microglial cells, with associated toxoplasma pseudocysts (Fig 7).

In summary, we have a case of fulminating *Pneumocystis carinii* pneumonia with a diffuse intra-alveolar exudate, and associated findings of cytomegalovirus and toxoplasma infection. We have to equate the findings with the history of corticosteroid therapy in this patient. On this basis, a purist would eliminate the definitive diagnosis of AIDS. We have enough evidence to conclude that the patient had severely compromised immune function. The patient is an extreme example of infection with the microorganisms commonly seen in patients with AIDS.[8,9]

FINAL DIAGNOSES

Probable acquired immunodeficiency syndrome (AIDS),

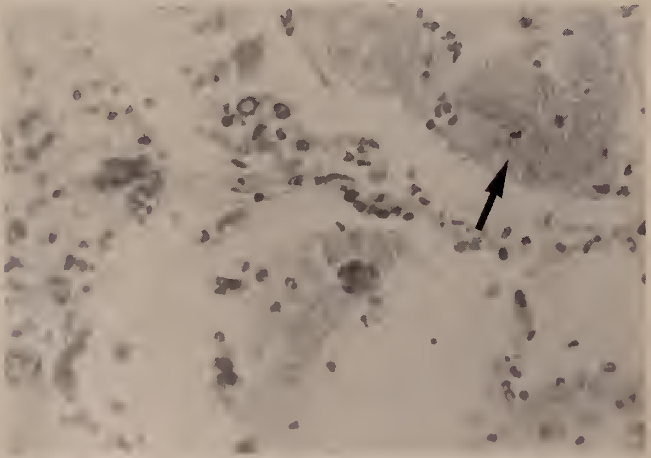


FIGURE 3. Silver stain of lung, showing pneumocystis organisms (arrow). (original magnification × 160)

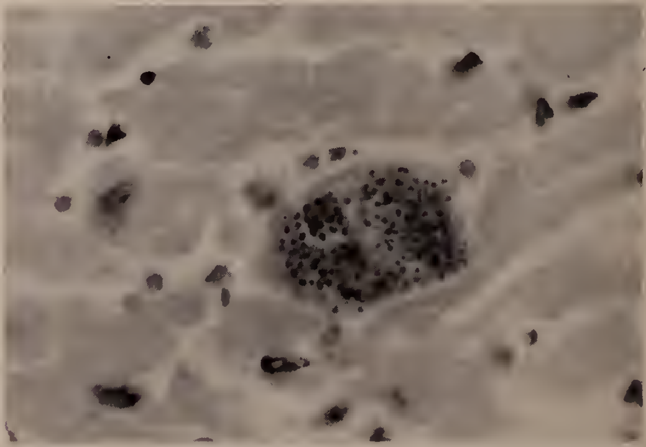


FIGURE 5. Toxoplasma pseudocyst containing trophozooites within myocardial fiber. (hematoxylin and eosin stain; original magnification × 340)

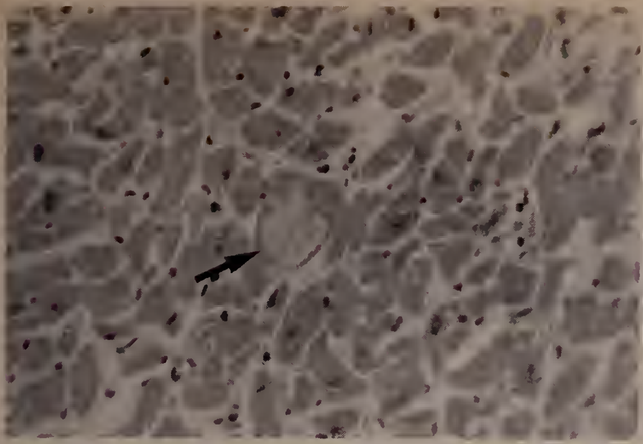


FIGURE 6. Myocardial fiber partially ruptured by toxoplasma pseudocyst (arrow). (hematoxylin and eosin stain; original magnification X 160)

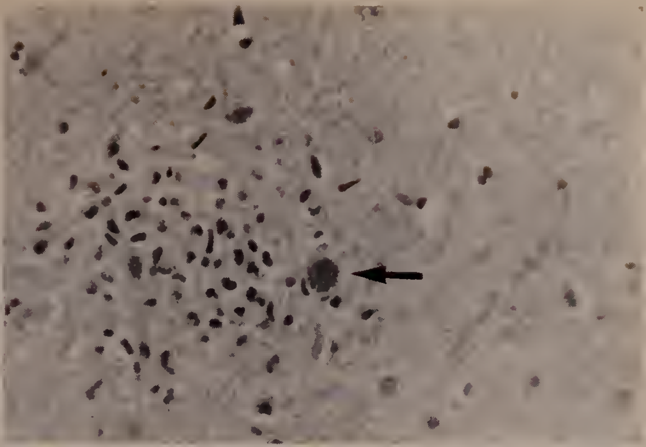


FIGURE 7. Microglial nodule within brain, with adjacent toxoplasma pseudocyst (arrow). (hematoxylin and eosin stain; original magnification X 160)

secondary to prolonged intravenous drug abuse.

Multiple opportunistic infections secondary to AIDS, including *Pneumocystis carinii* involvement of the lungs, toxoplasma infection involving the lungs, heart, spleen, and brain, and cytomegalovirus infection of the lungs and spleen.

Foreign-body giant cell reaction in the lungs, secondary to intravenous injection of drug contaminants.

Pleural reaction in the lower right side of the chest, secondary to previous trauma.

REFERENCES

1. Morris L, Distenfeld A, Amorosi E, Karpatkin S: Autoimmune thrombocytopenic purpura in homosexual men. *Ann Intern Med* 1982; 96:714–717.
2. Munt PW: Miliary tuberculosis in the chemotherapy era: with a clinical review in 69 American adults. *Medicine* 1971; 51:139–155.
3. Gelb AF, Leffler C, Brewin A, Mascatello V, Lyons HA: Miliary tuberculosis. *Am Rev Resp Dis* 1973; 108:1327–1333.
4. Huseby JS, Hudson LD: Miliary tuberculosis and adult respiratory distress syndrome. *Ann Intern Med* 1976; 85:609–611.
5. Murray HW, Tuazon CU, Kirmani N, Sheagren JN: The adult respiratory distress syndrome associated with miliary tuberculosis. *Chest* 1978; 73:37–43.
6. Fauci AS, Macher AM, Longo DL, et al: Acquired immunodeficiency syndrome: epidemiologic, clinical, immunologic and therapeutic consideration. *Ann Intern Med* 1984; 100:92–106.
7. Selik RM, Haverkos HW, Curran JW: Acquired immune deficiency syndrome (AIDS) trends in the United States, 1978–1982. *Am J Med* 1984; 76:493–500.
8. Reichert CM, O'Leary TJ, Levens DL, Simrell CR, Macher AM: Autopsy pathology in the acquired immune deficiency syndrome. *Am J Pathol* 1983; 112:357–382.
9. Horowitz SL, Bentson JR, Benson F, Davos I, Pressman B, Gottlieb MS: CNS toxoplasmosis in acquired immunodeficiency syndrome. *Arch Neurol* 1983; 40:649–652.

PRAISE FOR THE PHYSICIAN'S OFFICE

"We must encourage greater financing for out-patient care and try to cut back on the more expensive hospital utilizations as much as possible—particularly with regard to our aging population.

The doctor's office, with its beautiful simplicity, is the least expensive medical care delivery system- . . . . No hospital, no HMO or free-standing clinic, with all their duplicative personnel and paper work, can possibly compete dollar-wise with the low-cost productivity of the physician's office. We know it, and we should make certain that those in government also are made aware of it."

—from the inaugural address
of Allison B. Landolt, MD
President, Medical Society
of the State of New York
October 17, 1984

Factitious hypoglycemia associated with eosinophilic leukemoid reaction

Alan T. Lefor, MD, Myron Miller, MD

In 1927, Falcon-Lesses[1] observed complete disappearance of glucose from blood samples of patients with myelogenous and lymphocytic leukemia. Subsequent studies on blood from leukemic patients had led to the conclusion that the low serum glucose level is secondary to in vitro glycolysis by leukocytes.[2-7] Sodium fluoride can inhibit this process but only when present in very large amounts.[2,4,5] Leukocyte glycolysis can occur in blood samples obtained from healthy subjects,[6] but there are few reports in the literature of factitious hypoglycemia associated with leukemoid reactions,[3,6] particularly of an eosinophilic type.

Case Report

A 62-year-old man was transferred from another hospital to State University Hospital because of eosinophilia (white blood cell count, 131,000/mm^3 with 90% eosinophils) and hypoglycemia (blood glucose concentration, 26 mg/dL).

The patient had fatigue, dyspnea, decreased exercise tolerance, and weight loss of about 20 pounds during four months before being admitted to the first hospital for treatment of these symptoms. Five and a half years prior to this admission, he had undergone resection of a left retroperitoneal liposarcoma at another hospital.

Physical examination on admission revealed a thin man with a temperature of 38.1°C, a palpable mass in the left upper quadrant of the abdomen, and a liver edge palpable 3 cm below the right costal margin.

Initial laboratory studies included a white blood cell count of 133,700/mm^3 with a differential count of 72% eosinophils, 5% neutrophils, 16% band forms, 3% lymphocytes, 1% basophils, and 3% monocytes. The platelets appeared normal on the peripheral blood smear. The blood glucose level was 57 mg/dL. The insulin concentration was 4 μU/mL. Liver function tests were within normal limits except for an alkaline phosphatase level of 42 U/dL (normal, 13-20 U/dL).

Diagnostic studies included a normal liver-spleen radionuclide (^{99}Tc) scan and an abdominal computed tomographic scan that revealed a large retroperitoneal mass in the left upper quadrant.

Random blood glucose concentrations over the next several days were 84, 89, 10, and 27 mg/dL, during which time the patient was receiving an infusion of 10% dextrose in water at the rate of 100 mL/hr. The patient denied any symptoms of hypoglycemia throughout this time.

Blood samples obtained from the patient and from a control subject were divided and placed into three tubes. The first tube contained no additives and was brought to the laboratory for processing immediately after collection. A second tube was kept at room temperature for four hours before separating the cells from the serum. The third tube contained sodium fluoride and was kept at room temperature for four hours before being brought to the laboratory for measurement of serum glucose concentration.

The following day a heparinized sample of blood was obtained from the patient, and the erythrocytes were promptly removed by centrifugation, leaving only the plasma and the buffy-coat layer. These were gently mixed, and plasma glucose determinations were performed on the sample every hour for five hours. The same procedure was performed simultaneously on a sample of heparinized blood from a patient with chronic lymphocytic leukemia (a 58-year-old man; white blood cell count, 128,000/mm^3 with 2% neutrophils and 98% lymphocytes). All experiments were carried out at room temperature (68°F).

As shown in Table I, when blood from the patient was promptly centrifuged and the serum glucose content measured immediately, the glucose concentration was 132 mg/dL. Allowing the blood to stand at room temperature for four hours before it was processed resulted in a marked reduction in the glucose level to 43 mg/dL. When sodium fluoride (concentration, 2.5 mg/mL) was present in the collection tube, the blood glucose level did not fall with prolonged standing. Blood from a healthy subject allowed to stand at room temperature for four hours showed a decrease in glucose concentration from 80 to 61 mg/dL.

Serial measurement of glucose in plasma samples containing the white blood cells demonstrated that a major decline in glucose concentration was evident after one hour (Fig 1). The glucose level continued to fall sharply over the next two hours in a linear fashion, so that at the end of three hours, glucose concentration had decreased from 142 mg/dL to

TABLE I. Effect of Time and Presence of Sodium Fluoride on Whole Blood Glucose Determination in the Patient and in a Healthy Subject

Tube	Additive	Time	Glucose, mg/dL Patient	Glucose, mg/dL Healthy Subject
1	None	Immediate	132	80
2	None	4 hr	43	61
3	Sodium fluoride	4 hr	136	71

From the Department of Medicine, SUNY-Upstate Medical Center and the Veterans Administration Medical Center, Syracuse, New York.

Address correspondence to Dr Miller, Professor, Department of Medicine, Veterans Administration Medical Center, 800 Irving Avenue, Syracuse, New York 13210.

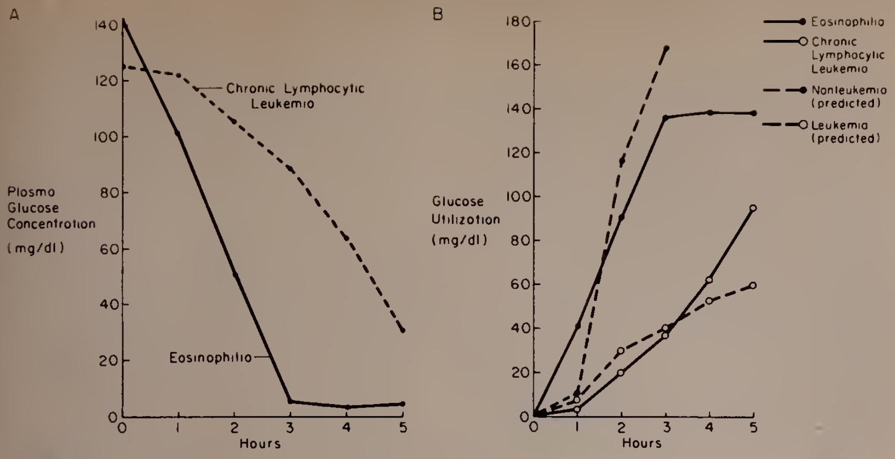


FIGURE 1. A, effect of time at room temperature on glucose concentration of plasma samples obtained from the patient with eosinophilia and a patient with chronic lymphocytic leukemia. The plasma contained the buffy-coat layer of cells following centrifugation and removal of the erythrocytes. B, glucose utilization in the plasma of the patient with eosinophilia and in the plasma of a patient with chronic lymphocytic leukemia (solid lines). The broken lines indicate predicted glucose utilization by normal leukocytes and by leukemic leukocytes for cell numbers equivalent to those present in the two patients (calculated from the equations of Rawnsley and Bowman[6]).

5 mg/dL. Plasma obtained from a patient with chronic lymphocytic leukemia who had essentially the same total leukocyte count showed a slower rate of decline in glucose concentration: a glucose level of 30 mg/dL after five hours.

DISCUSSION

Because this patient had a prior history of retroperitoneal liposarcoma, a recognized cause of true hypoglycemia,[8] the report that his blood glucose concentration was low led his physicians to suspect that he suffered from serious hypoglycemia. However, a careful review failed to disclose features characteristic of symptomatic hypoglycemia. The demonstration of a normal glucose level in a promptly processed specimen and a low glucose concentration in a blood sample for which analysis was delayed led us to conclude that the hypoglycemia was factitious and due to an in vitro process that could be inhibited by addition of sodium fluoride. To further eliminate the possibility of systemic hypoglycemia we discontinued the 10% dextrose infusion and found that, although the patient received nothing orally for eight hours, blood collected in sodium fluoride tubes (2.5 mg/mL) had glucose concentrations at zero, two, four, six, and eight hours of 95, 87, 87, 85 and 83 mg/dL, respectively.

Glucose utilization by leukocytes is plotted against time in the manner of Rawnsley and Bowman (Fig 1).[6] The data for both this patient and for the patient with chronic lymphocytic leukemia agree with previous work indicating a much higher rate of glucose utilization in nonleukemic blood compared to leukemic blood.[3,6] The results also demonstrate a more rapid in vitro utilization of glucose by eosinophils than by lymphocytes. Previous studies of blood from leukemic patients have led to the same conclusion.[1,6]

The cause of eosinophilia in this patient did not appear to be due to leukemia. Two weeks after resection of the retroperitoneal tumor—found to be a liposarcoma on histologic analysis—the leukocyte count dropped to 46,900/mm^3 with a differential count of 9% band forms, 29% neutrophils, 57% eosinophils, 3% lymphocytes, and 2% monocytes. We believe, therefore, that the elevated eosinophil count was most likely a leukemoid reaction secondary to the recurrent retroperitoneal liposarcoma, rather than a primary process. The percentage of eosinophils continued to decline to 37% five weeks after surgery. The death of the patient 10 weeks after resection of the tumor precluded further evaluation to determine the cause of the eosinophilia.

Factitious hypoglycemia due to in vitro glycolysis by leukocytes is rarely considered as a cause of a low blood glucose level. In patients with marked leukocytosis and hypoglycemia who have no hypoglycemic symptoms, the possibility of factitious hypoglycemia should be considered. By collecting blood from these patients in tubes containing sodium fluoride to inhibit in vitro glycolysis and by promptly processing the samples, the diagnosis of factitious hypoglycemia can readily be established.

REFERENCES

1. Falcon-Lesses M: Glycolysis in normal and leukemic blood. *Arch Intern Med* 1927; 39:412-420.
2. Field J, Williams H: Artifactual hypoglycemia associated with leukemia. *N Engl J Med* 1961; 265: 946-948.
3. Goodenow TJ, Malarkey W: Leukocytosis and artifactual hypoglycemia. *JAMA* 1977; 237:1961-1962.
4. Hanrahan J, Sax S, Gillo A: Factitious hypoglycemia in patients with leukemia. *Am J Clin Pathol* 1963; 40:43-45.
5. Messeloff C, Stolz C, Schoenfeld M: Factitious hypoglycemia in chronic myelogenous leukemia. *NY State J Med* 1964; 64:551-552.
6. Rawnsley H, Bowman H: Autoglycolysis in leukemic and nonleukemic blood. *Am J Med Sci* 1965; 249:203-210.
7. Salomon J: Spurious hypoglycemia and hyperkalemia in myelomonocytic leukemia. *Am J Med Sci* 1974; 267:359-362.
8. Scully R (ed): Case 5-1981: Case records of the Massachusetts General Hospital. *N Engl J Med* 1981; 304:282-290.

SUMMER RESEARCH ASSISTANT POSITION AT THE *JOURNAL*

Applications are invited from medical students (including those who will be entering medical school in the fall) for the position of research assistant at the *New York State Journal of Medicine*. This 10-week paid position is an opportunity for a student to participate in the editorial process of a peer reviewed scientific journal and to develop an original project for possible publication. Inquiries should be directed to the Editor. Applications should consist of a curriculum vitae and a covering letter. Deadline: April 1.

Retroperitoneal cystic lymphangioma

VINAY K. KAPOOR, MS, SUKANTO K. DAS, MS, ASHOK K. MUKHOPADHYAY, MD,
LALIT K. SHARMA, MS

Lymphangiomas are cystic and cavernous tumors that arise from lymphatic vessels and resemble on histologic analysis the more common cystic hygroma of the neck found in children.[1] Smooth muscle is present in the walls of a true cystic lymphangioma, differentiating it from an acquired lymphocyst.[2] Cystic lymphangiomas of the abdomen are rare and are found in the retroperitoneum. We report on a patient with a large retroperitoneal cystic lymphangioma.

CASE REPORT

A 31-year-old man sought treatment for an asymptomatic lump in the abdomen that had progressively increased in size for fifteen months. There were no symptoms to suggest obstruction of the gastrointestinal or urinary tracts. He was a well-built and fairly well-nourished young man. General physical examination was within normal limits. The abdomen was full, and a lump could be seen occupying almost all the abdominal quadrants. The lump had irregular margins and a bosselated surface. Its consistency was variegated—some areas were soft and fluctuant, whereas others were firm. The lump was nontender and not mobile from side to side. Percussion over the lump elicited a dull note. The chest was clear to auscultation. A clinical diagnosis of retroperitoneal tumor was made.

Routine investigations disclosed no abnormalities. A roentgenogram of the abdomen revealed a large soft tissue shadow in the right and central regions of the abdomen, displacing the bowel loops to the left. A chest film was within normal limits. An intravenous urogram showed the right kidney displaced upward by the mass; the right ureter was displaced medially, and an impression could be seen on the upper surface of the urinary bladder. Ultrasonographic examination showed a large retroperitoneal tumor extending from the under surface of the liver to the pelvic cavity, containing multiple anechoic areas with well-defined septa intervening.

Laparotomy was performed through a long right paramedian incision. A large multiloculated cystic tumor was present in the retroperitoneal space, occupying almost all the quadrants of the abdominal cavity and displacing the small bowel loops to the left upper quadrant of the abdomen. Larger cysts were punctured, and fluid was aspirated to decrease the size of the tumor and facilitate exposure. The whole tumor was removed intact. The abdominal viscera were normal. The abdomen was closed with a drainage tube in place. The patient had an uneventful recovery and the wound healed well.

The tumor measured 38 × 14 × 7 cm in size and weighed about 2.2 kg; no definite capsule was identified. The surface of the tumor was bosselated. Multiple translucent cysts of varying sizes were present; some were unilocular, others were multilocular. The cysts contained serous fluid, and no solid areas could be identified (Fig 1).

Histologic examination revealed irregular spaces containing light pink proteinaceous fluid and lined by flattened epithelial cells. The walls of the cysts consisted of fibrocollagenous tissue, and strips of smooth muscle could be seen (Fig 2).

DISCUSSION

Scattered reports of retroperitoneal cystic lymphangiomas are in the literature—both as isolated case reports[3,4] and as small collective series. Farrel and Grube[5] reported eight cases of retroperitoneal cystic lymphangiomas in the period from 1951 to 1961. Carpenter et al[6] reported 25 cases of multilocular cysts of the peritoneum found between 1907 and 1981, of which only 17 were cystic lymphangiomas. The authors divided them into two categories—cystic lymphangiomas and cystic mesotheliomas. These cysts have identical macroscopic appearances but can be differentiated on the basis of their light microscopic features; their clinical characteristics and courses differ.

Cystic lymphangiomas are lined with endothelial cells that have no microvilli; their walls are composed of smooth muscle. The cysts contain clear serous and chylous fluid. They are more common in men, and a significant number are found in children and young adults. Recurrence after complete excision is uncommon.

Cystic mesotheliomas are lined with mesothelial cells that have luminal microvilli; their walls do not contain smooth muscle. The cysts contain clear fluid. They are more common in adult women and tend to recur after excision—with multiple operations often required.

Both cystic lymphangiomas and cystic mesotheliomas are benign tumors—malignant change does not occur.

Cystic lymphangiomas of the abdomen are found in the retroperitoneal space and in the omentum and mesentery, which are forward extensions of the same space. The symptoms are bizarre, and the signs are nonspecific. A correct diagnosis is rarely made before surgery. The cysts may be detected during a routine physical examination, during surgery for another disease, or on postmortem examination. The patient may seek treatment for an asymptomatic abdominal lump. Obstruction of gastrointestinal, urinary, and biliary passages, hemorrhage in the cyst cavity, secondary infection, rupture of the cyst, and torsion are common complications. There are cases of cystic lymphangiomas of the abdomen associated with hydro-

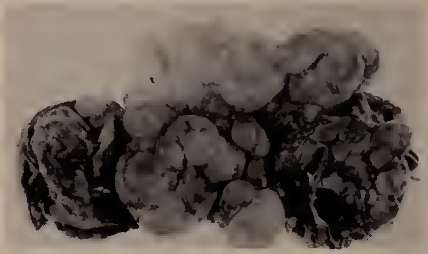


FIGURE 1. Retroperitoneal cystic lymphangioma—multiple cysts containing serous fluid.

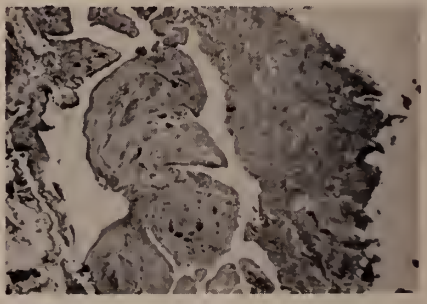


FIGURE 2. Part of the cyst wall showing smooth muscles. (hematoxylin and eosin stain, original magnification × 1600.)

From the Department of Surgery (Mr Kapoor, Mr Das and Mr Sharma), and the Department of Pathology (Dr Mukhopadhyay), All-India Institute of Medical Sciences, New Delhi, India.

Address correspondence to Mr Sharma, Associate Professor, Department of Surgery, All-India Institute of Medical Sciences, New Delhi-110029, India.

nephrosis[7] and cases that appear as pseudopancreatic cysts.[8] Treatment is by complete excision, which may necessitate bowel resection. Recurrence after complete excision is rare. Aspiration of large cysts before surgery facilitates exposure and excision without an increased risk of recurrence.[1]

REFERENCES

1. Peralta Jr MM, Brown HW: Retroperitoneal cystic lymphangioma. *Int Surg* 1971; 55:113-118.
2. Henzel JH, Pories WJ, Burget DE, Smith JL: Intra-abdominal lymphangiomata. *Arch Surg* 1966; 93:304-308.
3. Rauch RF: Retroperitoneal lymphangioma. *Arch Surg* 1959; 78:45.
4. Raszkowski HJ, Rehbock DJ, Cooper FG: Mesenteric and retroperitoneal lymphangioma. *Am J Surg* 1959; 97:363-367.
5. Farrel WJ, Grube P: Intra-abdominal cystic lymphangiomas. *Am J Surg* 1964; 108:790-793.
6. Carpenter HA, Lancaster JR, Lee RA: Multilocular cysts of the peritoneum. *Mayo Clin Proc* 1982; 57:634-638.
7. Harrow BR: Retroperitoneal lymphatic cyst (cystic lymphangioma). *J Urol* 1957; 77:82-89.
8. Sturim HS, Kouchoukos NT: Intra-abdominal cystic lymphangioma presenting as a pancreatic pseudocyst. *Amer J Surg* 1965; 109:807-809.

Fibroma of the lung

HELEN Y. KINAS, MD, ROGER L. GARCIA, MD, EMMA LUGO, MD

Fibromas of the lung are rare benign tumors of the lower respiratory tract that originate most often from the wall of the bronchi. They may appear as small intraluminal polypoid masses or, as in this patient, they may grow outside the bronchus and into surrounding parenchyma. These fibromas must be differentiated from benign tumors that originate from visceral pleura—these are called localized fibrous mesotheliomas of pleura or submesothelial fibromas. They appear to arise from submesothelial mesenchyme and have well-defined clinical and morphologic features.[1]

We report on a patient with fibroma of the lung, describing the roentgenographic and pathologic findings.

CASE REPORT

A 54-year-old man, who was seen in the emergency room with a chief complaint of right upper quadrant pain, was admitted because of renal colic with hematuria. A routine chest roentgenogram showed a sharply circumscribed, homogeneous round mass in the left mid-lung field (Fig 1). The patient denied any history of smoking or respiratory difficulties. Physical examination was unremarkable except for tenderness in the right upper quadrant.

Two bronchoscopies were performed that demonstrated compression of the lingular bronchial mucosa. Bronchial washings, brushings, and biopsy were also performed; they were negative for malignancy.

The chest film demonstrated a mass that measured approximately 5 cm in diameter and occupied the left mid-lung field. There was no evidence of calcification or cavitation. Inferior focal atelectasis was noted (Fig 1).

A computerized axial tomographic (CAT) scan of the chest was obtained; it showed bilateral pleural effusions and a large solid mass, with no evidence of calcification or cavitation. The medial portion of the mass was adjacent to the left hilum and major bronchi (Fig 2). A gallium scan showed slight uptake in the area of the lesion but no mediastinal involvement.

A lung biopsy yielded unsatisfactory material for diagnosis. The patient underwent a left upper lobectomy. During surgery, the mass was seen to involve the medial base of the lingular lobe. It did not involve the pleural surface. The mass was round, with expansile borders, and measured 5 cm in diameter. It was located near the hilum of the lobe. The bronchi and blood vessels were displaced and compressed slightly, but they were not invaded by the tumor. There was no connection between the tumor and the visceral pleura. Recognizable lung parenchyma separated the pleura from the tumor along the entire periphery of the mass. The cut surfaces were slightly bulging, white to light pink, partly nodular, and partly fasciculated.

On histologic examination the tumor was found to contain interlacing bundles of collagen fibers with varying numbers of cells. The cells had the morphologic and staining characteristics of fibroblasts and lacked signs of differentiation to any other cell type. No cellular atypia, mitotic activity, or other indicators of malignancy were present. The tumor appeared to be intimately associated with a bronchus near the hilum and, therefore, to have arisen from it and to have expanded extrinsically, displacing the surrounding parenchyma.

DISCUSSION

Fibromas are by definition benign tumors of fibroblasts. They are mesenchymal tumors commonly found in the superficial soft tissues, although they can occur anywhere in the body.

Fibroma of the lung is a rare tumor. It may arise from the trachea, bronchi, pleura, or parenchymal lung tissue. Lung

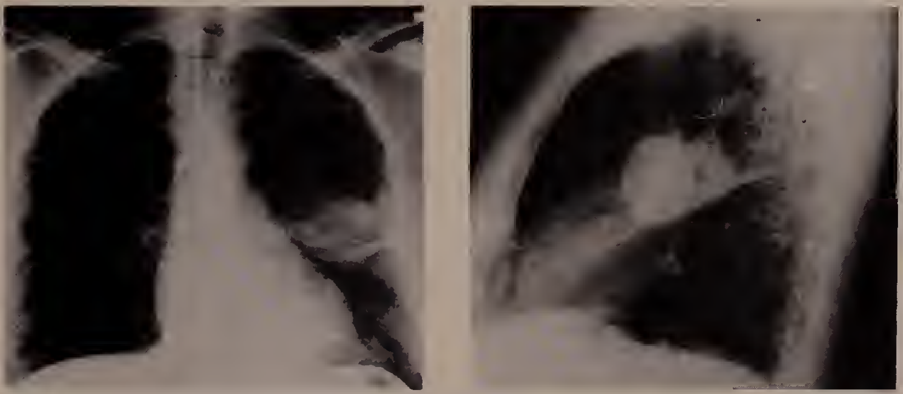


FIGURE 1. Left, posteroanterior view demonstrating a large, well-circumscribed mass in the left mid-lung field with inferior focal atelectasis; Right, lateral projection locates the mass in the upper lobe abutting the major fissure superiorly.

From the Departments of Radiology (Drs Kinas and Lugo) and Pathology (Dr Garcia), Beth Israel Medical Center, New York.

Address correspondence to Dr Kinas, Associate Attending Radiologist, Beth Israel Medical Center, 10 Nathan D. Perlman Place, New York, NY 10003.

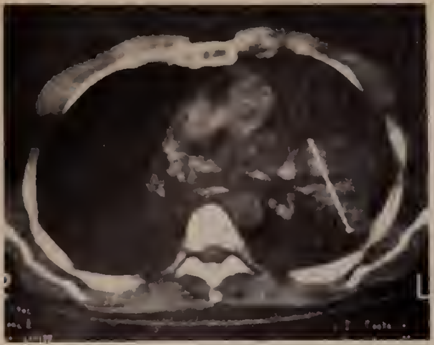


FIGURE 2. The mass is well delineated on CAT scan and measures approximately 5 cm, with no evidence of calcification or cavitation.

fibromas occur in both sexes, with a male predominance of 65%.[2] Most reported cases have been seen in Caucasians and in adults aged 24 to 84 years.

This benign tumor may be asymptomatic for several years, until it becomes large enough to cause symptoms such as chest pain, cough, dyspnea, or circulatory decompensation. Associated complications include bronchial obstruction, pulmonary osteoarthropathy, and malignant transformation to fibrosarcoma, although the latter is rare and difficult to document. Fibrinous pleurisy is common, and calcifications have been described in association with lung fibromas. Bronchial obstruction due to compression by the tumor may result in inadequate drainage and, ultimately, in bronchitis and pneumonia.[3] In pulmonary osteoarthropathy, the involved joint becomes swollen and tender prior to the appearance of chest symptoms. Following the removal of the tumor, these joint findings are relieved.

Pulmonary fibromas are divided into two types: the tracheobronchial form and the parenchymal form. The site of occurrence is evenly distributed between the two forms. The tracheobronchial form may be sessile or pedunculated.[4,5] Since this form occurs in tubular structures, symptoms typical of airway obstruction are often present before the tumor is visible on a roentgenogram. The parenchymal form usually appears as a small, well-circumscribed pulmonary mass, generally located in a lower lobe. Surgical removal is the treatment of choice, usually involving a local incision. The prognosis is excellent.[6]

Use of a CAT scan to determine the extent of the tumor mass is important. Whether the mass is surrounded by lung parenchyma or extends from the bronchi, it can be assessed by this method.

REFERENCES

1. Scharifker D, Kaneko M: Localized fibrous 'mesothelioma' of pleura (submesothelial fibroma): a clinicopathologic study of 18 cases. *Cancer* 1979; 43: 627–635.
2. Endelman R: Pulmonary fibroma—a rare benign tumor. *Am Rev Resp Dis* 1967; 96:1242.
3. Crim PD, Kiechle FL: Fibroma of the lung, case report. *J Thorac Surg*, 1952; 23:205–209.
4. Madewell JE, Feigin DS: Benign tumors of the lung. *Semin Roentgenol* 1977; 12:175–186.
5. Hochberg LA, Schachter B: Benign tumors of the bronchus and lung. *Am J Surg* 1965; 89:425–438.
6. Sauk JJ Jr, Pilego M, Anderson WK: Primary pulmonary fibroma, *Minn Med* 1972; 55:220-223.

Treatment of hemoptysis via bronchial artery embolization

MICHAEL TAFRESHI, MD, STEVEN SOBO, MD, LEON ADLER, MD, ARTHUR LISS, MD,
GEORGE PILLARI, MD

The efficacy of transcatheter vascular occlusion is well established, and the procedure has been applied successfully to the treatment of bleeding in various organ systems.[1] Selective bronchial arteriography and transcatheter bronchial artery occlusion have been effective in the management of massive hemoptysis.

A surgical plan for the treatment of massive hemoptysis is often delayed or limited, and is frequently precluded by underlying lung disease and respiratory insufficiency. In this report we describe a patient with hemoptysis secondary to tuberculosis and aspergilloma, who was effectively treated by means of transcatheter bronchial artery occlusion.

CASE REPORT

A 51-year-old woman was admitted to Queens Hospital Center because of progressively increasing hemoptysis for several hours prior to admission. Two months earlier, the patient had been admitted briefly for hemoptysis; the treatment was symptomatic, the bleeding was self-limited, and the patient was discharged after a one-week stay.

The patient complained of a persistent cough, blood-stained sputum, and weight loss of approximately 10 lb in the past two weeks. She had had tuberculosis 20 years prior to this admission. Her treatment at that time consisted of a two-year regimen of isoniazid and aminosalicylic acid therapy, and a 90-day course of streptomycin therapy. Six years after the original therapy for tuberculosis, the patient was treated for recurrent disease with the same regimen for a period of one year. At no time did the patient undergo surgical intervention for pulmonary disease.

Physical examination revealed a thin woman with considerable muscle-wasting, in acute distress from active coughing productive of bright red blood. Her blood pressure was 100/60 mm Hg; pulse rate, 120/min; and respirations were noted to be rapid. Laboratory studies were hematocrit, 32%; hemoglobin, 10 g/dL; white blood cell count, 9,800/mm^3 with a normal differential. Prothrombin time was 11.6 sec, with a control of 11.2 sec. A chest roentgenogram showed a loss of volume in the right lung with a shift of the trachea, heart, and mediastinal structures to the right. A cavitary lesion with an air-fluid level was demonstrated in the upper lobe of the right lung (Fig 1). Bronchoscopy disclosed bleeding from the bronchus in the upper lobe of the right lung. Early in the patient's hospital course, bleeding subsided considerably and hemoptysis was diminished and periodic. Results of pulmonary function studies were as follows: vital capacity, 45% of expected value; forced expiratory volume, 32% of normal. A titer for *Aspergillus* was 1:82, and repeated sputum examinations were negative for acid-fast bacilli.

On the 16th hospital day massive hemoptysis developed associated with hypotension and a drop in the hematocrit value from 32% to 20%. The patient was given a transfusion of four units of packed red cells. The patient was brought to the radiology department for bronchial arteriography. At this time she was relatively stable yet actively coughing. Her

---

From the Departments of Radiology, Queens Hospital Center Affiliation, Long Island Jewish-Hillside Medical Center, Jamaica (Drs Tafreshi, Sobo, Adler, and Liss), and Long Island Jewish-Hillside Medical Center, (Dr Pillari), New Hyde Park.

Address correspondence to Dr Tafreshi, Acting Director, Department of Radiology, Queens Hospital Center, 82-68 164th St, Jamaica, NY 11432.

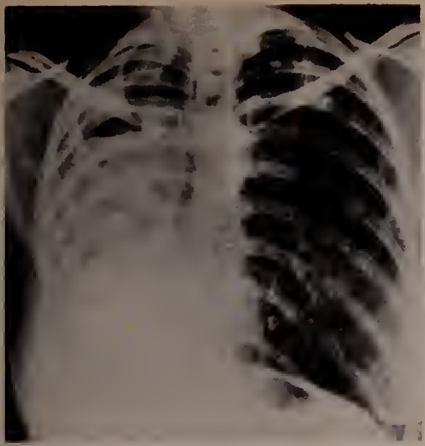


FIGURE 1. Chest film obtained on admission showing loss of volume in the right lung with a shift of the trachea, heart and mediastinal structures. Also evident is a cavitary lesion with an air-fluid level in the right upper lobe.

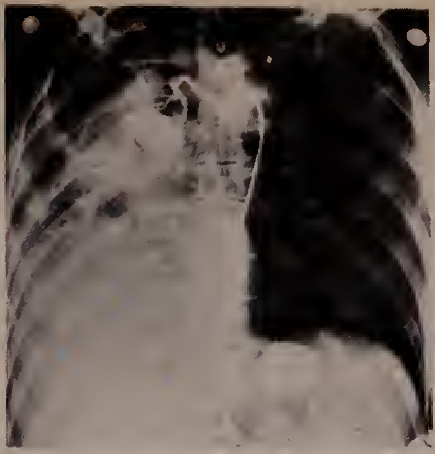


FIGURE 2. Angiogram demonstrating extravasation of the contrast material into the right upper lobe.

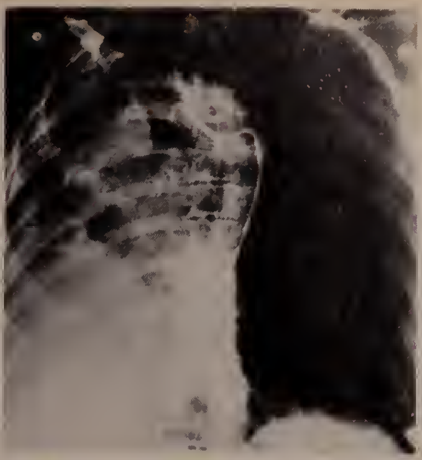


FIGURE 3. Angiogram after embolization showing occlusion of the right bronchial artery with no evidence of contrast extravasation.

cough was productive of large amounts of bright red blood during the catheterization procedure.

The bronchial artery in the upper lobe of the right lung was cannulated, and selective right bronchial arteriography was performed. Extravasation of the contrast material was noted in the right upper lobe (Fig 2). Multiple 1 to 2 mm pledgets of gelfoam were cut from a large strip of gelfoam. Embolization of the bronchial artery was performed by hand injecting approximately 25 to 30 gelfoam pledgets that were mixed with normal saline solution through the catheter placed in the affected right bronchial artery. Subsequent arteriography (Fig 3) demonstrated occlusion of the bronchial artery distal to its origin and bifurcation. Absence of extravasation was noted on the follow-up film.

Following successful embolization, the patient's condition improved dramatically, and on the 34th hospital day the patient was discharged in satisfactory condition. On subsequent follow-up at the pulmonary clinic, the patient was free of hemoptysis five months after her discharge.

Discussion

Massive hemoptysis is defined as blood loss of 600 mL or more in a 24-hour period.[1,2] Mortality in such cases is estimated at 80%.[2,3] The therapeutic effectiveness of transcatheter bronchial artery embolization is well documented and is a reasonable alternative to surgery.[1,2] Previous reports suggest a wide range of indications for embolization therapy including tuberculosis, bronchiectasis, pneumoconiosis, bronchial carcinoma, latent manifestations of pulmonary embolism, hydatid cysts, and a substantial number of patients with cystic fibrosis.[4] A limited number of patients with aspergilloma complicating tuberculosis has been described in the literature, and long-term intervals without bleeding are unusual in patients with aspergilloma. Remy et al[4] noted that the success of the transcatheter technique is limited because aspergilloma cavities have been shown to develop collateral blood supply from either the axillary or subclavian arteries. Additionally, bronchial artery-pulmonary artery anastomosis is known to occur and results in renewed bleeding at the site of the cavitary lung disease following effective occlusion of the bronchial artery circulation to the affected segment. In only four of six patients with aspergilloma who were treated during active hemoptysis was the procedure successful in controlling bronchial hemorrhage. Two patients died of their acute episode; of the remaining four patients, three had recurrent hemoptysis within two to seven months following embolization therapy. The treatment of massive hemoptysis in patients with tuberculosis is more successful. For example, of 16 patients treated for hemoptysis by means of bronchial artery embolization, 12 remained free of recurrence for more than seven months. Similar results were reported in cases of cystic fibrosis and in isolated reports of sarcoidosis, Hodgkin's disease, and bronchiectasis.[4]

Recurrent bleeding following bronchial artery embolization requires further arteriographic study in order to identify the most common causes—namely, recanalization of the occluded vessel or collateral blood supply from either the pulmonary artery circulation or the systemic vessels, such as the subclavian or axillary arteries. A thoracic aortogram, in addition to selective bronchial arteriography, is usually necessary prior to a definitive occlusion. In some cases, pulmonary arteriography may be necessary as well. Prior to embolization, the intercostal vessels and the bronchial artery branches should be carefully refused on the arteriogram to exclude the presence of communication with a spinal artery. In such instances anastomosis offers the risk of spinal artery occlusion and complications such as paralysis and transverse myelitis.[1,4–6] Identification of spinal artery anastomosis on the diagnostic arteriogram warrants that the procedure be abandoned unless the catheter can be securely placed distally in the bronchial artery branch from which the extravasated contrast dye is demonstrated.

Surgery is not a viable method of treatment in many patients with massive hemoptysis. In fact, surgery is contraindicated in patients with severe bilateral pulmonary disease, patients in whom the bleeding site is poorly localized on bronchoscopy, patients with vital capacities of less than 40% of predicted value, and patients with diminished forced expiratory volume (<40%). Some patients will refuse surgery.

In this report, bronchial artery embolization was used effectively in a patient with massive hemoptysis secondary to aspergilloma. Transcatheter embolization therapy has proved successful in the treatment of hemoptysis secondary to a range of underlying pulmonary abnormalities, most notably tuberculosis and cystic fibrosis.[7]

References

1. Athansoulis CA, Pfister RC, Greene RE, Roberson GH: *Interventional Radiology.* Philadelphia, WB Saunders Co, 1982.

2. Wholey MH, Chamorro HA, Rao G, Ford WB, Miller WH: Bronchial artery embolization for massive hemoptysis. *JAMA* 1976; 236:2501–2504.

3. Prioleau Jr WH, Vujic I, Parker EP, Voegele LD, Hairston P: Control of hemoptysis by bronchial artery embolization. *Chest* 1980; 78:878-880.

4. Remy J, Arnaud A, Fardou H, Giraud R, Voisin C: Treatment of hemoptysis by embolization of bronchial arteries. *Radiology* 1977; 122:33-37.

5. Bookstein JJ, Moser KM, Kalafer ME, Higgins CB, Davis GB, James WS: The role of bronchial arteriography and therapeutic embolization in hemoptysis. *Chest* 1977; 72:658-661.

6. Feigelson HH, Ravin HA: Transverse myelitis following selective bronchial arteriography. *Radiology* 1965; 85:663-665.

7. Stern RC, Wood RE, Boat TF, Matthews LW, Tucker AS, Doershuk CF: Treatment and prognosis of massive hemoptysis in cystic fibrosis. *Am Rev Respir Dis* 1978; 117:825-828.

8. Bredin CP, Richardson PR, King TKC, Sos TA, Smith JP: Treatment of massive haemoptysis by combined occlusion of pulmonary and bronchial arteries. (abstract) *Chest* 1977; 72:258-259.

9. MacErlean DP, Gray BJ, FitzGerald MX: Bronchial artery embolization in the control of massive haemoptysis. *Br J Radiol* 1979; 52:558-561.

Disseminated toxoplasmosis with signs and symptoms of a cerebral lesion

IRIS FLETCHER NORSTRAND, MD, PHD, KAZEM FANI, MD, FIDELIO A. JIMENEZ, MD,
JOANNA HOLLENBERG SHER, MD

As disseminated toxoplasmosis is not usually suspected in a patient with symptoms of a cerebral mass lesion, the following two patients are described in whom the initial signs were those of a right cerebral hemisphere tumor.

CASE REPORTS

Case 1. A 48-year-old furniture mover was admitted to the hospital because of the onset of bifrontal headache and dizziness one and a half months before admission and the occurrence of two focal left-sided seizures followed by a left hemiplegia on the day of admission. Except for surgery for lip cancer five months earlier, he had enjoyed excellent health. There was no history of drug addiction, hemophilia, venereal disease, head injuries, heart disease, hypertension, diabetes, or other serious illnesses. The patient was a heavy beer drinker, was separated from his wife, and lived alone.

On admission, temperature was 100°F; blood pressure, 120/80 mm Hg; pulse rate, 100 beats/min. The patient was plethoric-looking and was fully oriented, but was apathetic. Physical examination was unremarkable except for scaly papules on the extremities and excoriated papules with central ulceration on both earlobes. There was flaccid left hemiplegia with mild left supranuclear facial weakness, absent abdominal reflexes, and a more active left knee and ankle jerk. There were no pathologic reflexes.

Findings from routine laboratory studies were within normal limits except for a white blood cell count of 1,500/mm^3 and a platelet count of 79,000/mm^3. There were 54% polymorphonuclear leukocytes, 32% lymphocytes, 9% monocytes, and 5% eosinophils. The red blood cell count was 4,500,000/mm^3, with a hemoglobin level of 13.9 g/dL; the hematocrit value was 40.7%. Immunofluorescence testing disclosed antinuclear antibody. Two chest roentgenograms—on admission and three weeks before death—showed no active pulmonary disease. A computed tomographic (CT) scan was performed with and without contrast (Fig 1). A cerebral angiogram was inconclusive, although it was suggestive of a lesion in the right cerebral hemisphere. A bone marrow biopsy specimen showed hypocellular marrow. Despite the CT scan findings of a solitary right cerebral hemisphere lesion with surrounding edema, craniotomy was not considered because it was thought that the patient had a systemic disease with central nervous system involvement. Toxoplasmosis was not specifically considered.

Except for a transient two-day regression of left hemiplegia a month after admission, his condition deteriorated and a low-grade intermittent fever developed. He experienced another focal left-sided seizure with recurrent left-sided Todd's paralysis and increasing lethargy. Subsequently cyanosis, conjugate deviation of the head and eyes to the right, stertorous irregular respirations, and deep obtundation developed. Despite administration of steroids and antibiotics, the patient died one and a half months after admission.

At autopsy, the brain weighed 1,625 g. Lesions of toxoplasmosis were found in the brain (Fig 2) and heart. Although generalized lymphadenopathy and hepatosplenomegaly were found, no *Toxoplasma* were noted in these organs.

Case 2. A 54-year-old man was admitted for evaluation of right frontotemporal headache and attacks of cramping of the left lower extremity, followed by left hemiparesis that began one and half weeks before admission.

The patient had been addicted to heroin 14 years previously, but he denied substance abuse after detoxification. For the past two years the patient had been followed in a clinic because of unexplained anemia, leukopenia, and rubbery bilateral axillary and inguinal lymph nodes. A bone marrow biopsy specimen obtained one year previously showed only hypocellularity. The patient had recently been admitted to a hospital because of cough, night sweats, and a sore throat. A chest roentgenogram showed bilateral interstitial infiltrates at the bases of both lungs. Results of serologic studies for cytomegalovirus, toxoplasmosis, and Epstein-Barr virus were negative. After treatment with erythromycin, there was gradual clinical improvement, although the chest roentgenogram remained unchanged for several weeks.

On admission, his temperature was 102°F; pulse rate, 104 beats/min; blood pressure, 120/80 mm Hg. Sensorium and higher intellectual functions were intact. Physical examination was unremarkable except for scarring of the veins of both forearms. Neurologic examination disclosed left hemiparesis and diminished left abdominal reflexes; no other reflex abnormalities were noted.

Abnormal findings from laboratory studies were as follows: white blood cell count, 5,900/mm^3; red blood cell count, 3,870,000/mm^3; hemoglobin, 10.9 g/dL; hematocrit, 32.9%; mean corpuscular volume, 84/μm^3; mean corpuscular hemoglobin, 28.17 pg/cell; mean corpuscular hemoglobin concentration, 33.1%; platelets, 310,000/mm^3; alkaline phosphatase, 163 mU/mL. A chest roentgenogram taken on admission showed chronic obstructive pulmonary disease without evidence of active infiltration. A CT scan performed after admission showed obliteration of the right lateral ventricle, with large hypodense areas involving both cerebral hemispheres; there was much greater involvement on the right. There were no abnormal areas of enhancement. There was bilateral obliteration of the cerebral sulci. A follow-up cerebral CT scan was taken two weeks later (Fig 3). An abdominal CT scan showed prominence of the head and body of the pancreas, with no retroperitoneal lymphadenopathy. Analysis of cerebrospinal fluid disclosed normal pressure, three lymphocytes, and a total protein value of 33

From the Departments of Neurology (Dr Norstrand) and Pathology (Drs Fani and Jimenez), Brooklyn Veterans Administration Medical Center, and Department of Pathology (Dr Sher), Downstate Medical Center, New York.

Address correspondence to Dr Norstrand, Assistant Chief, Department of Neurology, Brooklyn VA Medical Center, 800 Poly Place, Brooklyn, NY 11209.

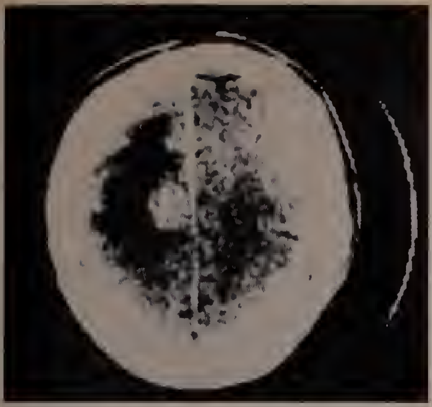


FIGURE 1. Computed tomographic scan of patient 1, with and without contrast, shows solitary low-density lesion high in convexity of right parietal lobe, with uniform enhancement in center of lesion and marked surrounding edema of entire hemisphere, indicating possible metastatic tumor.

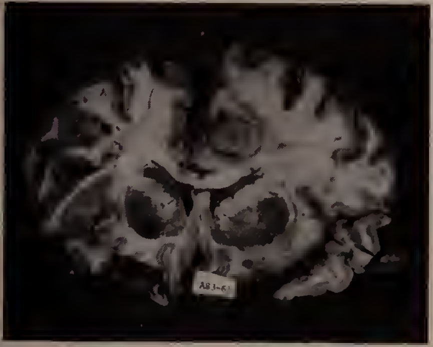


FIGURE 2. Coronal sections through brain of patient 1 show large areas of necrosis and edema throughout right cerebral hemisphere. There are multiple lesions, with the largest measuring 7 cm in diameter and compressing right cingulate gyrus.

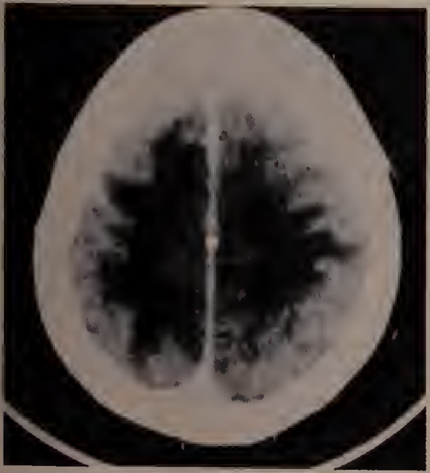


FIGURE 3. Follow-up computed tomographic scan of patient 2 shows greater involvement without enhancing lesions.

mg/dL; culture of the fluid was negative. The following positive titers in the cerebrospinal fluid were noted: *Toxoplasma gondii* (indirect hemagglutination, 1:64); *Entamoeba histolytica* (indirect hemagglutination, 1:32), and *Legionella pneumophila* (immunofluorescence antibody, 1:64). Positive serologic titers were as follows: influenza type A, 1:32; parainfluenza virus, 1:16; herpes virus, 1:32; *T gondii* (indirect hemagglutination, 1:64); (immunofluorescence antibody, 1:16); *Mycoplasma*, 1:64; *Chlamydia* (*Bedsonia* group), 1:16; *L pneumophila*, 1:64; and Epstein-Barr virus (immunofluorescence antibody, 1:10).

Except for transient return of function to the left extremities for two days at the end of the first week following admission, his condition deteriorated. There was progressive clouding of the sensorium, development of delusions of persecution and suicidal ideation, noisy, assaultive behavior, urinary incontinence, and occasional focal and generalized seizures. Several days before death, the patient become dyspneic and sluggish. A chest roentgenogram showed an infiltrate in the lower lobe of the left lung; lung biopsy revealed *Pneumocystis carinii.* Despite treatment with intravenous trimethoprim and sulfamethoxazole, intubation, and supportive therapy, the patient died one and a half months after admission.

The autopsy showed bronchopneumonia with abscess formation. Gross examination of the brain showed multiple necrotizing granulomas, varying from 0.4 cm to 3.0 cm in greatest diameter in all lobes of the cerebrum and in the left basal ganglia, subthalamus, brainstem, and cerebellum. The lesions had necrotic centers, with infiltrates of lymphocytes and macrophages, as well as prominent vasculitis. Cysts containing *Toxoplasma* organisms and free trophozoites were numerous in the inflammatory exudate.

DISCUSSION

Toxoplasmosis initially seen as a brain tumor or brain abscess has been reported in the literature.[1-7] These studies report that only in older patients do the microgranulomas reach a large size and begin to soften to give rise to signs that mimic a cerebral tumor or abscess, as occurred in both patients. Recently, Vilaseca et al[8] reported a well-demarcated spherical brain abscess in the right frontoparietal area, discovered at surgery, in a 35-year-old homosexual man with fever, headache, lymphadenopathy, and skin lesions caused by Kaposi's sarcoma. The patient died four days after craniotomy. Hymes et al,[9] in a report of eight cases of Kaposi's sarcoma in homosexual men, described one patient with a right frontal lobe mass. No necropsy was done.

In both these patients, the initial clinical findings consisted of right frontal headaches, focal left-sided seizures, and a left hemiparesis, suggesting a right frontal mass lesion. In case 2, the CT scan showed a diffuse process, with greater involvement of the right hemisphere. Since this patient, with *P carinii* and previous lymphadenopathy, mild leukopenia, and anemia, clearly represented a case of acquired immunodeficiency syndrome (AIDS), and the titer of *T gondii* in both spinal fluid and serum was 1:64, disseminated toxoplasmosis or other opportunistic infection was strongly suspected. Central nervous system toxoplasmosis in patients with AIDS is being reported in the literature with increasing frequency.[10] Although the CT scan was less helpful in case 1—showing only involvement of the right cerebral hemisphere—it was postulated that the patient had a generalized systemic disease, with central nervous system involvement and immunosuppression. By definition, case 1 fulfilled the criteria for a patient with AIDS because of the leukopenia and thrombocytopenia due to unknown causes. Further evidence of immunosuppression was the recent treatment for lip cancer. Although no biopsy was performed, the skin lesions could have been caused by Kaposi's sarcoma. In both cases, despite evidence of cerebral edema on CT scans, steroids were of no benefit. Both patients exhibited transient improvement before their deaths.

The possibility of toxoplasmosis should be considered in any patient initially seen with the clinical picture of a mass lesion.

REFERENCES

1. Tognetti F, Galassi E, Gaist G: Neurological toxoplasmosis presenting as a brain tumor: Case report. *J Neurosurg* 1982; 56:716-721.
2. Schulhof LA, Russell JR: Intracerebral toxoplasmosis presenting as a mass lesion. *Surg Neurol* 1975; 4:9-11.
3. Hervei S, Simon K: Congenital toxoplasmosis mimicking a cerebral tumor: special aspects in serodiagnostics of congenital töxoplasmosis. *Monatsschr Kinderheilkd* 1979; 127:43-47.
4. Bobowski SJ, Reed WG: Toxoplasmosis in an adult, presenting as a space-occupying cerebral lesion. *Arch Pathol* 1958; 65:460-464.
5. McLeod R, Berry PF, Marshall WH Jr, Hunt SA, Ryning FW, Remington JS: Toxoplasmosis presenting as a brain abscess: Diagnosis by computerized tomography and cytology of aspirated purulent material. *Am J Med* 1979; 67:711-714.
6. Bamford CR: Toxoplasmosis mimicking a brain abscess in an adult with treated scleroderma. *Neurology* 1975; 25:343-345.
7. Pitchenik AE, Fischl MA, Walls KW: Evaluation of cerebral-mass lesions in acquired immunodeficiency syndrome. *N Engl J Med* 1983; 308:1099.
8. Vilaseca J, Arnau JM, Bacardi R, Mieras C, Serrano A, Navarro C: Kaposi's sarcoma and *Toxoplasma gondii* brain abscess in a Spanish homosexual. *Lancet* 1982; 1:572.
9. Hymes KB, Cheung T, Greene JB, et al: Kaposi's sarcoma in homosexual men: A report of eight cases. *Lancet* 1981; 2:598-600.
10. Wong B, Gold JWM, Lange M, Fried R, et al: Central nervous system toxoplasmosis in patients with AIDS. *NY Clin Res* 1983; 31:379A.

AN APPRECIATION

Our referees

The cornerstone of a peer review journal is its refereeing process. In 1984 the individuals listed below served as referees for manuscripts submitted to the Journal. *Their continuing contribution is acknowledged with deep appreciation. We invite other individuals who would like to become involved in the editorial process to write to the* Journal.

David P. Adams, Gainesville, FL
Horacio J. Adrogue, MD, Houston, TX
Raymond Alexanian, MD, Houston, TX
Constantine S. Anast, MD, Boston, MA
George J. Annas, JD, Boston, MA
William Arnold, MD, New York, NY
Steven G. Atcheson, MD, Reno, NV
Harold L. Atkins, MD, Stony Brook, NY
Peter Auld, MD, New York, NY
Robert Austrian, MD, Philadelphia, PA
Demetrius H. Bagley, MD, Philadelphia, PA
Daniel G. Baker, MD, St. Davids, PA
Emil J. Balthazar, MD, New York, NY
Lynn H. W. Banowsky, MD, San Antonio, TX
Leon Barnes, MD, Pittsburgh, PA
William Barnes, MD, New York, NY
Andrew Bartilucci, PhD, Jamaica, NY
Jorge L. Benach, PhD, Stony Brook, NY
Kurt Benirschke, MD, La Jolla, CA
Bruce Edward Berger, MD, San Francisco, CA
Michael E. Berlow, MD, Syracuse, NY
Henry W. Blackburn, Jr, MD, Minneapolis, MN
Christopher R. Blagg, MD, Seattle, WA
Randall Bloomfield, MD, Brooklyn, NY
Max Borten, MD, Brookline, MA
Robert D. Brandstetter, MD, New Rochelle, NY
David Bruns, MD, Charlottesville, VA
Richard J. Calame, MD, Brooklyn, NY
Guy D. Campbell, MD, Jackson, MS
William A. Causey, Jackson, MS
A. J. Chandraeskhar, MD, Claredon, IL
P. Joan Chesney, MD, Madison, WI
Charles L. Christian, MD, New York, NY
Ralph V. Clayman, MD, Minneapolis, MN
Joseph Cleary, MD, New York, NY
Stephen D. Clements, MD, Atlanta, GA
Michael B. Cohen, MD, New Orleans, LA
Paul S. Cohen, MD, Syracuse, NY
Philip Cole, MD, DrPH, Birmingham, AL
John E. Conte, Jr, MD, San Francisco, CA
Gene Coppa, MD, New York, NY
Thomas J. Craig, MD, New City, NY
John A. Crocco, MD, New York, NY
Joseph Davis, MD, Miami, FL
Thomas J. Degnan, MD, Manhasset, NY
Bennett Marsh Derby, MD, New York, NY
Frank DeStefano, MD, Atlanta, GA
Marcus A. DeWood, MD, Spokane, WA
Herbert Diamond, MD, New Hyde Park, NY
Lewis C. Drusin, MD, New York, NY
Nicholas Reed Dunnick, MD, Chapel Hill, NC
Nicholas G. Economides, MD, Memphis, TN
Garabed Eknoyan, MD, Houston, TX
John T. Ellis, MD, New York, NY
Bery Engebretsen, MD, Des Moines, IA
Franz Enzinger, MD, Washington, DC
John M. Esdaile, MD, New Haven, CT
Joseph Feldman, DrPH, Brooklyn, NY
Austin Fink, MD, Brooklyn, NY
Raymond Fink, PhD, Valhalla, NY
Bruce D. Fisher, MD, Plainfield, NJ
Thomas B. Fitzpatrick, MD, Boston, MA
Marshall S. Flam, MD, Fresno, CA
John T. Flynn, MD, New York, NY
Samuel J. Fomon, MD, Iowa City, IA
Daniel Foster, MD, Dallas, TX
Roger S. Foster, MD, Burlington, VT
Arthur Fox, MD, New York, NY
Eli Friedman, MD, Brooklyn, NY
Rachel Fruchter, PhD, Brooklyn, NY
Charles D. Gerson, MD, New York, NY
Patricia Giardina, MD, New York, NY
Boyd W. Goetzman, MD, Davis, CA
Philip J. Goldstein, MD, Brooklyn, NY
Myles Gombert, MD, Brooklyn, NY
Alexander Gotta, MD, Brooklyn, NY
William J. Graham, MD, Oklahoma City, OK
John R. Graybill, MD, San Antonio, TX
Margaret T. Grossi, MD, New York, NY
David Allen Hafler, MD, Boston, MA
W. Dallas Hall, MD, Atlanta, GA
Joel I. Hamburger, MD, Southfield, MI
Milton B. Handelsman, MD, New York, NY
Edward V. Hannigan, MD, Galveston, TX

Tomas N. Heimann, MD, Port Chester, NY
Terry Hensle, MD, New York, NY
Margaret W. Hilgartner, MD, New York, NY
Alan D. Hilgenberg, MD, Boston, MA
Margaret Mary Hoehn, MD, Denver, CO
Robert W. Hollenhorst, MD, Brainerd, MN
Lewis B. Holmes, MD, Boston, MA
Mark Homonoff, MD, New York, NY
Thomas Houston, MD, Rome, GA
Max Howard, MD, New York, NY
Jeffrey L. Huffman, MD, New York, NY
A. S Iskandrian, MD, Philadelphia, PA
Bobbie Jacobson, MB, BS, London
Bernard M. Jaffe, MD, Brooklyn, NY
Fidelio A. Jimenez, MD, Brooklyn, NY
Peter R. Jochimsen, MD, Iowa City, IA
Robert L. Johnson, MD, Newark, NJ
James R. Jones, MD, Piscataway, NJ
Lois G. Jovanovic, MD, New York, NY
Mamoru Kaneko, MD, New York, NY
Richard E. Kanner, MD, Salt Lake City, UT
Silloo Kapadia, MD, Pittsburgh, PA
Robert Katz, MD, New York, NY
Mary Kaufman, MD, Brooklyn, NY
Florence Kavaler, MD, Brooklyn, NY
Philip L. Kaye, MD, Flushing, NY
Thomas P. Kearns, MD, Rochester, MN
Margaret Anne Keller, MD, Torrance, CA
Merrill S. Kies, MD, Chicago, IL
R. Michael King, MD, Rochester, MN
Irvin Klein, MD, New York, NY
Lloyd Klein, MD, Philadelphia, PA
David A. Kliot, MD, Brooklyn, NY
Vernon Knight, MD, Houston, TX
Henry Krakauer, MD, PhD, Bethesda, MD
Charles Kramer, Esq. New York, NY
Howard P. Krieger, MD, New York, NY
Paul D. Krieger, MD, New Milford, CT
Itzhak Kronzon, MD, New York, NY
Leslie A. Kuhn, MD, New York, NY
Donald Henry Kuiper, MD, Lansing, MI
Joseph F. Kuzma, MD, Milwaukee, WI
Robert A. Kyle, MD, Rochester, MN
Neil A. Lachant, MD, Torrance, CA
F. E. Lajam, MD, Manhasset, NY
J. Thomas LaMont, MD, Boston, MA
Robert Landesman, MD, New York, NY
Lewis Landsberg, MD, Boston, MA
Norman Latov, MD, New York, NY
Gary Lee Lattimer, MD, Williamsport, PA
Chung K. K. Lee, MD, Minneapolis, MN
Daniel L. Levin, MD, Dallas, TX
Lawrence D. Lilien, MD, Wilmette, IL
Robert D. Lindberg, MD, Houston, TX
Arthur S. Localio, MD, New York, NY
James Lomax, MD, Brooklyn, NY
Robert T. L. Long, MD, Florence, AL
Bernard Lown, MD, Brookline, MA
Joshua Lynfield, MD, New York, NY
Richard Macchia, MD, Brooklyn, NY
Walter C. MacDonald, MD, Vancouver, BC
William C. MacLean, Jr MD, Columbus, OH
Henry Mannix, MD, West Hartford, CT
Barry J. Maron, MD, Bethesda, MD
John S. Marr, MD, New York, NY
James Mazzara, MD, New York, NY
John H. McGovern, MD, New York, NY
Richard H. Meade III, MD, Arlington, MA
Yashbir Mehta, MD, Addison, IL
Ralph Mendez, MD, Los Angeles, CA
Daniel C. Merrill, MD, Martinez, CA
Donna Mildvon, MD, New York, NY
James Morrissey, JD, New York, NY
Donald Moy, JD, Lake Success, NY
Edward C. Muecke, MD, New York, NY
Henry Murray, MD, New York, NY
Daniel M. Musher, MD, Houston, TX
Josef Neu, MD, Milwaukee, WI
James Robert Newland, MD, Omaha, NE
James A. Nicholas, MD, New York, NY
David J. Nochimson, MD, Farmington, CT
Stephen Nordlicht, MD, New York, NY
Gary Jay Novetsky, MD, Skokie, IL
Michael J. O'Connell, MD, Rochester, MN
Robert J. O'Connor, MD, Lake Success, NY
William D. Odell, MD, Salt Lake City, UT
Man S. Oh, MD, Brooklyn, NY
Adedamola Oni, MD, Nashville, TN
David G. Ostrow, MD, Chicago, IL
Edward A. Palank, MD, Manchester, NH
Henry Pek, MD, Great Neck, NY
Harold C. Pillsbury, MD, Chapel Hill, NC
Ira A. Polisar, MD, Brooklyn, NY
Joseph Post, MD, New York, NY
Stephen R. Preblud, MD, Atlanta, GA
John T. Prior, MD, Syracuse, NY
Homayoon Rahbari, MD, Monroe, NY
John H. Ranson, MD, New York, NY
Lee Reichman, MD, Newark, NJ
Joseph J. Ricca, MD, Brooklyn, NY
Philip S. Ricke, MD, Tucson, AZ
Derry Ridgway, MD, Portland, OR
Ronald Rieder, MD, Brooklyn, NY
Thomas S. Riles, MD, New York, NY
Jean E. Rinaldo, MD, Pittsburgh, PA
Max Robinowitz, MD, Washington, DC
Arvin E. Robinson, MD, Mobile, AL
Manuel Rodstein, MD, Bronx, NY
Milton Rosenberg, MD, Lake Success, NY
Richard F. Rosenberg, MD, Great Neck, NY
David J. Rosenblum, MD, Brooklyn, NY
Richard Rothman, MD, Philadelphia, PA
Marvin Rotman, MD, Brooklyn, NY
Michael G. Sarr, MD, Baltimore, MD
John A. Schaefer, MD, New York, NY
David Charles Schecter, MD, New York, NY
Robert C. Schlant, MD, Atlanta, GA
Joseph Schluger, MD, Brooklyn, NY
Richard Schwarz, MD, Brooklyn, NY
H. William Scott, Jr, MD, Nashville, TN
Joseph W. Segura, MD, Rochester, MN
Harlan Selesnick, MD, Chicago, IL
Stephen Seligman, MD, Brooklyn, NY
Michael B. Seshul, MD, Nashville, TN
Barry Shandling, MD, Toronto, ONT
John J. Shane, MD, Springtown, PA
Charles F. Sharp, MD, Los Angeles, CA
A. H. Shatila, MD, Cleveland, OH
Earl Shelp, PhD, Houston, TX
Lawrence M. Sherman, MD, Snyder, NY

Louis M. Sherwood, MD, Bronx, NY
John Shillito, MD, Boston, MA
Frederick Sillman, MD, Brooklyn, NY
Richard Silver, MD, New York, NY
Mark Silverman, MD, Atlanta, GA
John D. Slade, MD, Trenton, NJ
David Sohn, MD, New York, NY
David Sokol, MD, Tucker, GA
Albert Stanek, MD, Brooklyn, NY
A. C Steere, Jr, MD, New Haven, CT
John S. Stehlin, Jr, MD, Houston, TX
Richard Stillman, MD, Brooklyn, NY
Charles Dennis Swerdlow, MD, Stanford, CA
Jessie L. Ternberg, MD, St. Louis, MO
Peter B. Terry, MD, Baltimore, MD
Arthur N. Tessler, MD, New York, NY
Colin G. Thomas, Jr, MD, Chapel Hill, NC
Byron M. Thomashow, MD, New York, NY
Norman W. Thompson, MD, Ann Arbor, MI
Russell Thomsen, MD, Washington, DC
Bjorn Thorbjarnarson, MD, New York, NY
Robert W. Tofte, MD, St. Paul, MN
Jonathan B. Towne, MD, Milwaukee, WI
John L. Tully, MD, Cambridge, MA
Rodrigo Urizar, MD, Albany, NY
William Valenti, MD, Rochester, NY
Joseph F. Valicenti, MD, Charleston, SC
Alje Vennema, MD, New York, NY
Nicholas J. Vianna, MD, Albany, NY
Robert Alan Vigersky, MD, Washington, DC
Ann Elaine Walts, MD, Los Angeles, CA
W. Richard Webb, MD, South San Francisco, CA
Michael R. Weitekamp, MD, Hershey, PA
Berttina B. Wentworth, PhD, Lansing, MI
Peter H. Wiernik, MD, Bronx, NY
Richard Winterbauer, MD, Seattle, MD
William L. Wood, Jr, Esq, New York, NY
Stanley B. Wolfe, MD, Southfield, MI
Arthur H. Wolintz, MD, Brooklyn, NY
Mahfouz H. Zaki, MD, DrPH, Hauppauge, NY
David Zakin, MD, New York, NY
Herbert R. Zatzkin, MD, John Island, SC
Horst P. Zincke, MD, Rochester, MN
Arthur Zitrin, MD, New York, NY
Andrew J. Zweifler, MD, Ann Arbor, MI

Reforming Medicine: Lessons of the Last Quarter Century

Edited by Victor W. Sidel and Ruth Sidel. 311 pp. New York, Pantheon Books, 1984. $9.95 (paperback)

This review could start with the question "Why does medicine and the American health care system need to be reformed?" Many professionals would say that the system works well, and that most Americans get good care. On the other hand, the reformers could point to the disparate health status between the poor and the well-off, the uncontrolled growth of technology utilization, the rapid inflationary spiral of health care costs, the woes of the elderly, the rural versus urban inequities, and the lower longevity in the United States in comparison with other countries. In their introduction, the Sidels note that "there was a feeling that much of the caring was disappearing from health care, that what should be a humane service for everyone in need was as racist, sexist, ageist, distant, and unfeeling as other U.S. institutions and perhaps in some ways even more so."

Ralph Waldo Emerson wrote, "What is a man born from but to be a reformer, a remaker of what has been made, a denouncer of lies, a restorer of truth and good?" Including the Sidels, 19 reformers from the front-line trenches recount lessons from their turbulent efforts of the 1960s and 1970s, as they sought to reform medicine in America. Despite Emerson's use of the word "man," many of the reformers are women, and they fought the battle for the women's health movement. Their individual contributions exhibit the intimacy of expression of those who have endured the joys and sorrows of actually being there when the events were taking place. These authors clearly show that they are writing from the depths of their ideological convictions.

In the section on health care institutions, Geiger discusses community health centers as instruments of social change. Whereas he agrees that health care has improved, he concludes that the essential structure of the nation's health care system is unchanged, remaining centered around hospitals, oriented toward technology, and focused on tertiary care. Young follows with his description of urban hospital development in Chicago, pointing out the inequity, the low performance, and the emphasis on high technology. He states that after two decades of hospital expansion, the conclusions are "pessimistic, if not absolutely negative." Bandaids for the old and poor provided through Medicare and Medicaid are the core of Brown's analysis. There can be no doubt that these two governmental programs significantly improved access to health care services for the old and the poor. However, Brown does note that the greatest limitation of the reforms was the separation of medical care for the poor from the program for the elderly. Political support accrued for Medicare, while Medicaid's political base eroded under the welfare stigma.

Occupational health and safety and the women's health movements illustrate efforts aimed at special groups. Coye, Smith, and Mazzocchi delineate the lessons of the 1970s and the struggles with the Occupational Safety and Health Administration. Economics becomes a critical factor as the benefits of occupational health and safety reforms are weighed against dollar cost. Abortion, diethylstilbesterol use, and experimentation on women are among the areas covered by Rodriguez-Trias. She comments that the greatest challenge by the women's movement was in women's demands for control over their own bodies.

Reforms in the area of medical education, affirmative action, the National Health Service Corps and new health professionals are discussed. Huet-Cox takes a look at "new wine in old wine skins," as she progresses from the Flexner report to the curriculum changes in medical education in the 1960s. What was reformed? There were enlarged enrollments, more minority and low-income students, expanded training in family practice and generally more exposure to relevant health care topics. Affirmative action, as analyzed by Strelnick and Young, affirms the power of academic institutions to go their own way despite attempts to impose a public agenda on them. Furthermore, they note that class as well as ethnicity must be considered in effecting reforms. Mullan enriches this section with his first-hand experience with the National Health Service Corps. He comments that "it is impossible to escape the conclusion that the health care of geographically or economically marginal populations is tied to the economy of the nation." Physician's assistants and nurse practitioners are the new health professionals described by Backup and Molinaro. As a challenge to the establishment, these mid-level health practitioners have raised the spectre of "a sorcerer's apprentice turned loose."

Hatch and Eng discuss community participation and control, and Sirott and Waitzkin exhort the virtues of holism and self-care. Both of these segments take up issues that cut to the base of professional dominance of health care services. Both also relate to reforms that are still being pursued.

Finally, the Sidels take us toward the 21st century. After stressing the formidable resistance of the system to change, they posit three categories of reform: (1) activities resisted so successfully that they never really took hold; (2) activities implemented but later co-opted or absorbed into the fundamental structure of the system; and (3) activities briefly successful but later destroyed through underfunding or lack of commitment. Each of the examples cited in the book are placed into the appropriate category by the Sidels.

Do these groupings bode well for future health care reformers? This book has to be read for the answer. A flavor and feeling of the intensity levels of the authors cannot be adequately transmitted in a book review. You may disagree with their ideals and their activities, but you will realize that this collection of authors has something to say that is worth discussing among all those in the health care system. Of import to the reader is the fact that each of the articles does give a broad historical overview of a problem and is backed up by references.

By gathering together a group of health care activists to recall their struggles to effect change, the Sidels have enriched our knowledge of how the system works or doesn't work. This book is recommended for all who would like to learn about the inner dynamics of the major events that took place within our health care system during the past 25 years.

ALLEN D. SPIEGEL, PhD
Professor
Department of Preventive Medicine
and Community Health
State University of New York
Downstate Medical Center
New York City 11203

FROM THE LIBRARY

MEDICINE UNDER SIEGE

By the word 'medicine' in 'Medicine Under Siege,' is meant the medical profession. For ages the medical profession has been permitted to follow unhindered its humane occupation of caring for the sick, giving little heed to commercial and economic problems which were so foreign to its principles and to its traditions. Of late, however, the world seems to have been given over to commercialism, and attempts are constantly being made to drag medicine from the humanitarian position of a sympathetic, hope-inspiring, sound medical adviser and to lower it to the level of a purely materialistic, commercial occupation, and would soon have it listed with the class of manual labor. This may seem to be a pessimistic and extreme view of the situation but we think if one will devote a little study to the encroachments that are being made in the field of the practice of medicine, to the attitude assumed by some of the great leaders of industry as expressed in articles in popular lay magazines, to the dangers of misguided and misapplied philanthropy, to the extension of the work of health officers in the domain of the private physician, to the enactment by legislatures of arbitrary and unnecessary laws that place additional burdens on and restrict the independent action of the physician, we believe that one cannot fail to be impressed by the fact that the siege of medicine is on.

M. L. HARRIS, MD
President of the American Medical Association (1929–1930)
(*NY State J Med* 1930; 30:823)

OPERATIVE PERINATOLOGY: INVASIVE OBSTETRIC TECHNIQUES

Edited by Leslie Iffy, MD and David Charles, MD. 1,084 pp, illustrated. New York, Macmillan Publishing Co, 1984. $110.00 (hardcover)

This book is a monumental attempt to combine large amounts of basic and clinical knowledge into a single volume. The two editors, Iffy and Charles, are assisted by six associates and two junior editors. The purpose of the book is to detail the operative and invasive procedures involved in modern obstetrics. The book is divided into 12 sections, which include applied anatomy, asepsis and infection control, recognition and management of reproductive abnormalities, medical and surgical complications, abnormal delivery, perinatal infection, special techniques for labor, neonatal complications, genital malignancies, and social perspectives. There are more than a hundred contributors; some sections are written by as many as four authors. The subject of operative perinatology or invasive obstetrical techniques would have comprised sufficient material for one book. However, the editors include obstetric knowledge that extends far beyond this field of study. They include information on anatomy, infection control, mastitis, perinatal infection, genital malignancies, educational issues, social problems, and malpractice. More than half of the material in this textbook could have been excluded if the authors had held strictly to the title.

The book advocates the use of x-ray pelvimetry for special purposes, including breech presentation, labor with unengaged head, previous difficult delivery, and abnormal pelvic configurations. Some large universities in the United States no longer use pelvimetry because of the risks of x-ray exposure and the use of clinical criteria for judging pelvic adequacy. The authors state that patient anxiety and fear may be eliminated by performing mammograms. Both techniques reduce the opportunity for error, but mistakes are far fewer if both methods are used. Even with both of these methods, one of the most difficult diagnoses is breast malignancy in a lactating mother.

Sabbagha, in an outstanding chapter on ultrasound, indicates difficulties in the diagnosis of ectopic pregnancy. In early pregnancy, a uterus without a gestational sac may indicate an ectopic pregnancy. An abnormal pregnancy or an early missed abortion without intrauterine visualization may lead to diagnostic problems.

In the section on ectopic pregnancy, Diamond writes, "the factor responsible for the early diagnosis of ectopic pregnancy is the development of the beta subunit hCG determined by radioreceptorassay which can detect pregnancy two weeks after conception or at the time of the first missed period." This sentence quoted from Chapter 25 confuses two distinct laboratory determinations. The receptor assay recognizes the entire hCG molecule and the biological activity of the hCG hormone. Radioimmunoassay for the beta subunit of hCG recognizes a specific portion of the molecule. These tests recognize different portions of the molecule.

At the time the book was written there was great enthusiasm about fetal therapy for hydrocephalus via amniocentesis for the removal of cerebrospinal fluid from the fetal ventricle. Since 1983, experience with this group of abnormalities has been discouraging, and several sonographers and obstetricians have avoided amniocentesis as a treatment for hydrocephalus.

Version of the fetus from the breech or transverse position has become popular since the advent of continuous monitoring and tocolytic agents. Hansen provides good descriptions of the procedures and indicates contraindications and possible causes for abnormal presentation. The incidence and types of complications are omitted.

Cohen summarizes electronic and biochemical fetal monitoring. The illustrations and descriptive text cover the field in capsule form, ideally suited for residents and medical students. Historic anecdotes are intertwined with the latest interpretations of recorded fetal electronic variations.

Cesarean section is reviewed by Burchell. The rapid increase in the frequency of this operation is upsetting to the author, especially as the frequency approaches 25% of all deliveries. Extraperitoneal cesarean operations are described in detail, but the author does not indicate the frequency of their use. This operation is confined to history; at The New York Hospital-Cornell Medical Center no such procedure has been performed in more than 20 years.

Iffy and Charles have accumulated a huge amount of clinical material concerning operative perinatology. Perhaps a more limited subject matter with emphasis on details would make the book more suitable for use by an obstetrician. The breadth of topics with limited range may be satisfactory for medical students and residents. Books such as *Operative Obstetrics* by Douglas and Stromme, Read's *Controversy in Obstetrics and Gynecology*, and *Complications in Obstetrics and Gynecology* by Schaefer and Graber would be more appropriate references for the practicing specialist. In the future, Iffy and Charles might divide this book into two sections: the first dealing with anatomy, physiology in obstetrics, infections, equipment, and historical notes; and the second volume including only operative perinatology.

ROBERT LANDESMAN, MD
The New York Hospital-
Cornell Medical Center
New York City 10021

PRINCIPLES OF SPORTS MEDICINE

Edited by W. Norman Scott, MD, Barton Nisonson, MD, James A. Nicholas, MD. 433 pp, illustrated. Baltimore, Williams & Wilkins, 1984. $65.00 (hardcover).

In this book the editors have undertaken a monumental task: they want "to bridge the hiatus between physicians and interested paramedicals, professional therapists, trainer, coach, parent, and athlete." Their ultimate goal is "a well informed individual who understands preventive and therapeutic sports medicine."

The editors assembled a qualified group of authors to convey the principles of sports medicine. The authors present current medical research as well as controversial theories that relate to their fields of study. The text is not intended as a handbook for the treatment of injuries or as a text on exercise physiology. Rather it reviews the anatomic, physiologic, pathologic, and rehabilitative aspects of athletics.

The text is divided into three parts: Physiology in Sports, Recognition and Treatment of Injuries, and Preventive Sports Medicine. The well-organized chapters within these three sections contain numerous graphics, x-rays, diagrams, and anatomic drawings and illustrations. They are readable and well referenced, especially the chapter on pediatric sports medicine.

The bulk of the text is devoted to the recognition and treatment of injuries. However, unlike many texts on athletic injuries, this book does not describe in detail the surgical procedures involved in treating the injuries. Instead, the editors focus on the anatomy and biomechanics of the human body and the effects of sports injuries to various bodily parts.

Two chapters left this reviewer confused as to their place in the text. One deals with football equipment and the other with safety in skiing. Perhaps a discussion on equipment in general would have been more appropriate, with emphasis on the collision sports such as football, lacrosse, hockey, and rugby. A chapter on safety in all sports might have emphasized equipment, regulations, playing fields, facilities, playing conditions, and weather conditions.

Another bothersome error is in the chapter entitled "Conditioning and Rehabilitation." The authors state in the beginning of the chapter that "muscle biopsy and histochemical techniques will be reviewed in terms of predicting athletic ability, changes with injury, and immobilization and implications for research." Yet such a review could not be found in the chapter, and there was no reference to it in any other chapter in the book. An "overview of specific rehabilitation programs dealing with the shoulder, knee, and ankle" could not be found in the chapter either.

Although there are some shortcomings, the editors have achieved their goal. Despite the definite slant toward orthopedics, the book does bring together many ideas in an organized fashion, which helps one understand the full spectrum of sports medicine. The book should be a valuable reference to family physicians, internists, and pediatricians who treat patients active in athletic endeavors.

JOHN P. SHEEHY, MD
Community Hospital at Glen Cove
Glen Cove, NY 11542

THE LIVES OF LESIONS. CHRONOLOGY IN DERMATOPATHOLOGY

By A. Bernard Ackerman, MD and Anna Ragaz, MD. 253 pp, illustrated. New York, Masson Publishing USA, Inc, 1984. $75.00 (hardcover)

This book was derived from a series of lectures given by Dr Ackerman at the New York University Medical Center. He is a professor in the Department of Dermatology at that institution, where for the past decade he has been responsible for the training of dermatology residents and dermatopathology fellows. He is known for his innovative thinking regarding the pathology of skin diseases. Dr Ragaz, also at New York University, collaborated with Dr Ackerman on both the lecture series and the compilation of the book.

Many skin diseases are evolutionary in nature, that is, they undergo a process of change from early to late stage lesions. Clinical textbooks on dermatology routinely describe this process, but dermatopathology texts have not followed suit. Rather, the histology is usually depicted as static. This book is unique in its attempt to portray the histologic evolution of skin diseases.

The layout of the book is excellent. Each section follows a pattern: three clinical color photographs that depict early, fully developed, and late stage lesions; a one-page clinical and histopathologic description of the natural course of the skin disease; a one-page summary of the salient histologic features of each of the three stages; and lastly, several pages of low and high power microscopic views of the three stages as well as subtle variations.

Undoubtedly the finest feature of the book is the superb quality of the black and white photomicrographs. Not one appears to be overexposed or underexposed or even slightly out of focus. Even more importantly, the photos illustrate salient points. The text and labeling of the photomicrographs are clear and concise. The 10-page glossary is quite useful, since even experienced dermatologists may have some trouble with the terms used by dermatopathologists.

The book is designed for use by dermatologists and dermatopathologists. Other physicians will probably find standard texts more useful. This text, which describes in detail only 25 skin diseases, is really meant as a supplement to a standard text on dermatopathology. Not all skin lesions evolve, and many therefore are not included. However, there are some omissions of important lesions that clearly do evolve, such as those of acne vulgaris.

In short, the book is a classic in its field. It is "must" reading for dermatologists and dermatopathologists.

WILFRED MINKIN, MD
White Plains Hospital Medical Center
White Plains, NY 10601

A PRIVATE PRACTICE

By Patrick Reilly, MD. 346 pp. New York, Macmillan Publishing Co, 1984. $15.95 (hardcover)

This is a true account of a successful 36-year-old pediatrician who became addicted to tranquilizers and sleeping pills. Patrick Reilly (a pseudonym) details in the first five chapters his slow but inexorable dependence on Valium, Equagesic, Talwin, Compazine, amphetamines, and alcohol for overwhelming anxiety attacks. He vividly portrays how he furtively obtained and stockpiled his medications by falsifying prescriptions or stealing the drugs from his own patients. Then, in the solitude of his office, he managed to conceal his Mephistophelian ritual from his wife and colleagues. We read how he repeatedly deceived his wife and friends concerning his missed dinners, forgotten engagements, mood swings, and episodes of anxiety, attributing them to job stress. We cringe when we note his mismanagement of meningococcal meningitis as a viral syndrome, miscalculation of a fatal dose of digitalis for a three-month-old child, and the ordering of penicillin for an allergic child. Reilly's dishonesty extended to self-deception: "I may not have functioned at a hundred percent . . . but I'm damn good." Even after he decided to admit himself into a drug rehabilitation center and not to be dishonest again, Reilly lied to his wife in the same breath. He said that x-rays had caused his sterility, whereas he secretly had had a vasectomy in order to avoid the responsibility of children.

The remaining 20 chapters recount his difficult rehabilitation process, punctuated by a withdrawal seizure, profound anxiety and depression that required medication, and recalcitrant denial. It is clear from the many poignant messages that cry out from these pages that physicians with drug problems can by virtue of their training be their "own worst enemies." Compassionate, detached concern can easily translate into a superficial feeling of control, a sense of aloofness, and a myriad of excuses for not admitting that there is an addiction problem. In fact, the physician-narrator gained insight into his addiction only in the final hours of his three-week hospitalization. Throughout the intervening days at the treatment center he recounted the emotional abyss of his terrifying struggle through the use of flashbacks and interactions with the other patients of his group. The language is tough; the pain can be felt by the reader. We follow Dr Reilly as he rediscovered his sexuality and self-esteem—a path marked by premature ejaculation, an affair, and suicidal ideation.

Perhaps my only objection to this book is that the denouement is based on presumption. Dr Reilly is not discharged from the center, but rather "recycled" for another three weeks of rehabilitation in order to consolidate his therapy. The reader is left to make his or her own conclusions concerning the narrator's fate, although by all measures, Reilly's prognosis is excellent.

We, as physicians, regularly ask our patients to "let go" and follow our advice. Unless we are patients with a serious illness, like Dr Patrick Reilly, being honest with ourselves and "letting go" is a curious philosophical exercise with little practical meaning. *A Private Practice* is recommended for a healthy dose of reality testing for those who would like the next best thing.

RICHARD BIRRER, MD
State University of New York
Downstate Medical Center
New York City 11203

DIABETES: THE GLUCOGRAF METHOD OF NORMALIZING BLOOD SUGAR

By Richard K. Bernstein, MD. 294 pp, illustrated. California, Jeremy P. Tarcher, Inc, 1981. $8.95 (softcover)

Although this book is entitled *Diabetes*, it is primarily concerned with the strict maintenance of normal blood glucose levels in patients with insulin-dependent diabetes mellitus. Its consideration of adult-onset, noninsulin-dependent diabetes is minimal.

The author, now over 50 years of age, has had diabetes since he was 12½ years old. He suffered all the psychological and physical inconveniences of uncontrolled diabetes until he adopted the method of strict glycemic control described in this book. Originally trained as a management engineer, he now has an MD degree and treats diabetic patients.

The author's previous engineering training is evident in the methodical and often mathematical approach he has taken in attaining normal blood sugar levels. This approach and the precise manner of keeping accurate and complete daily records that he recommends are given the name "the Glucograf Method," mentioned in the title of the book.

To illustrate his "tight management approach," he advises that the insulin therapy for the person with insulin-dependent diabetes be started with ultralente insulin. To equal the dose of insulin usually secreted by a fasting, nonexercising person in a 24-hour period, one-tenth of a unit per kilogram weight is given before breakfast and again before supper. To prevent a postprandial blood sugar increase, one-tenth of a unit of regular insulin per kilogram of weight is given before each meal. Along with these insulin requirements, a low carbohydrate (17% to 25%), high protein (45%) diet is advised, with normal amounts of fat (30% to 38%, mostly polyunsaturated). Preprandial and postprandial blood sugar levels must be determined daily to ascertain that the desired physiologic blood glucose levels are attained. The directions for changes in insulin administration and diet are explicitly indicated to correct for any fluctuations in blood sugar levels.

For patients and physicians who are dedicated to maintaining constant normal blood sugar levels, without using a pump, this book is one of the best guides. It not only presents the necessary knowledge for the patient in an explicit yet simplified manner, but also elaborates on the many difficulties and complications encountered when attempting to comply with even a less rigorous regimen. Most treatises on diabetes tend to neglect such practical aspects as when to inject insulin to prevent hypoglycemia, what to do about "snacking," and how to manage control when exercising or when one has an infection with loss of appetite.

The surgeon could well follow the operative program for patients with diabetes. The survey of equipment used by patients is well presented—cost effectiveness is emphasized. The suggestions are valuable for physicians, nurses, and health-care workers who care for diabetic patients.

We are entering an era in which "tight control" is being accepted by the medical profession as necessary in the treatment of insulin-dependent juvenile diabetes. Although methods other than those described in this book are being used, the information presented here is valuable to those involved in the treatment of such patients.

MILTON B. HANDELSMAN, MD
The Long Island College Hospital
New York City 11201

TECHNOLOGICAL BASIS OF RADIATION THERAPY: PRACTICAL CLINICAL APPLICATIONS

Edited by Seymour H. Levitt, MD, Norah duV. Tapley, MD. 336 pp, illustrated. Philadelphia, Lea & Febiger, 1984. $45.00 (hardcover)

The authors have produced an excellent book that is much needed by radiation therapists, technicians, and all those involved in employing radiation therapy. The authors are radiation therapists with much experience in the field, and their choice of contributors is further evidence of their expertise. The subject matter of the book is well chosen and covers portions of the body that commonly receive radiation therapy. Especially remarkable are the chapters "Treatment Aids for External Beam Radiotherapy," "Complex Field Arrangements: Field Shaping and Separation of Adjoining Fields," "Dosimetry of Irregularly Shaped Fields," "Brain Tumor Localization: Determination of Extent and Volume for Radiation Treatment," "Gynecologic Cancers: Pelvic Examination and Treatment," and "Treatment Techniques Depending on Tumor Extent in Cancer of the Breast.

Interstitial techniques are described with precision and proper documentation. Mention is also made of an imaginative technique using templates that attempts to develop more homogeneous irradiation to some parts of the body. The chapter on lymphomatous disease is also excellent. It describes in detail the diagnosis of a tumor and the determination of its extent as well as the techniques used in treatment. This book belongs on the shelves of residents, radiation therapists, physicians, and radiation therapy technologists.

JOSEPH L. BOHORQUEZ, MD
The Albany Medical College
Albany, NY 12208

Medical Directory Errata

Corrections of editorial errors in biographic material in the *Medical Directory of New York State* Volume LIX 1984–85 are listed below.

These corrections are set in a matching format to be inserted into your MEDICAL DIRECTORY OF NEW YORK STATE

★ **ALTCHEK, Victor S,** 125-06 101 Ave. Richmond Hill. 11419. Tel 718-849-2900. NY MED 42. D-S. SAC. FACS. Asst Attd Sur Beth Israel. Attd Sur La Guardia. Also Manhattan.

★ **BARONE, Luciano Vito,** RFD 4. Box 48. Warwick. 10990. Tel 914-986-2058. CORNELL 78. D-P. Staff Ped St Anthony.

★ **BERGER, Marvin,** 10 Nathan D. Perlman Pl. Manhattan. 10003. Tel 212-420-2092. CHGO Med 61. D-IM(CVD). SJM-4. FCCP. FACC. FACP. NY Cardiol. Attd Phy Med Beth Israel. Asso Chief Cardiol. Beth Israel. (AMA)

★ **BERGER, William Matthew,** 91 Rugby Rd. Brooklyn. 11226. Tel 718-287-3333. TEMPLE 62. D-OL. FICS. Am Ac Otolar. PAMA. NY Otol. Vis Otolar Kings Co. Asso Otolar Interfaith. Attd Otolar Univ. Asso Otolar L.I.C. Asso Otolar Man E&ET. (AMA)

BRANDEIS, Steven Zachary, 30 Park Ave. Manhattan. 10016. Tel 212-696-5411. NYU 75. D-S. D-CRS. SA. Am Colon & Rect Sur. Attd Sur NY Inf-Beekman Down. Attd Sur Bell.

★ **BRASLOW, Nelson Mark,** 169 Washington Ave. Kingston. 12401. Tel 914-331-8830. HARVARD 75. D-IM(PD). Amer Thor. Amer Int Med. NYS Int Med. Dir Respir Dis Kingston. (AMA)

★ **BROOK, David W,** 55 E 87 St. Manhattan. 10128. Tel 212-831-3388. YALE 61. D-PN(P). SI. Am Psych. Asso Attd Psych Mt Sinai. Asso Attd Psych City Elmhurst.

★ **CASAZZA, Sharen,** 19 South St. Washingtonville. 10992. Tel 914-496-3656. PENN 75. D-P. Am Ac Ped. Staff Ped Arden Hill. (AMA)

★ **COBURN, Morton Baron,** 222-15 Union Tpke. Flushing. 11364. Tel 718-776-0655. MIDDLESEX 39. SFM-13. FCCP. PAMA. Am Ac Otolar. NY Rhino-Otolar. Asst Otolar Cabrini Med Ctr. Chief Otolar Parsons. Chief Otolar Deepdale. Staff Otolar Hillcrest.

★ **COHEN, Herbert Irving,** 25 Central Park W. Manhattan. 10023. Tel 212-541-9033. Roosevelt Hosp Manhattan 10019. Tel 212-554-7192. JOHNS HOPKINS 49. Am Ac Alg. Chief Ped Alg St Lukes-Roosevelt. Att Ped St Lukes-Roosevelt. Also Amagansett. (AMA)

★ **COVEY, Alan H,** 445 Main St. Box 1128. Center Moriches. 11934. Tel 516-878-1043. Tel 516-288-3620. YALE 54. D-IM. SJM-4. FACP. FACA. Am Int Med. NYS Int Med. Phy Int Med Cent Suffolk. (AMA)

★ **DITCHEK, Alan.** 137 Girard St. Brooklyn. 11235. Tel 718-615-1197. AUTONOMA GUADALAJARA 81. (AMA)

★ **DRUCKER, Barry Lee,** 58-47 Francis Lewis Blvd. Bayside. 11364. Tel 718-423-1500. 68-52 Fresh Pond Rd. Ridgewood 11385. Tel 718-497-4455. KIRKSVILLE Col Osteo 71. D-O. E. Am Ac Oph. Attd Oph Beth Israel. Attd Oph Deepdale. Attd Oph Parsons. Attd Oph Hillcrest.

★ **EBIN, Leonard N,** 521 Park Ave. Manhattan. 10021. Tel 212-744-6200. VA 51. D-R. D-IM. D-NUM. SD. FACG. Nuclear Med. Radiol NA. Ac Med NY. Ac Gastroent. Asso Radiol Met. Sup Roent Coler Meml. (AMA)

★ **FAYER. Michael C,** 3 E 74 St. Manhattan. 10021. Tel 212-744-7331. MIDDLESEX 45. L. Asst O&G Beth Israel. (AMA)

FISCHEL, Robert Elliot, 5 W 86 St. Manhattan. 10024. Tel. 212-799-8818. P&S NY 74. D-PN(P). Am Psych. Am Psychoan Clin Asst Psych Mt Sinai. Asst Attd Psych St Lukes-Roosevelt.

★ **HISLER, Stuart Eric,** 130-56 Lefferts Blvd. South Ozone Park. 11420. Tel 718-843-2233. AUTONOMA GUADALAJARA 77. J. Am Int Med. Attd Int Med Booth Meml. Attd Int Med Parkway. Attd Fam Pract Jamaica.

★ **KAPLAN, Rosalind P,** 199 Weaver St. Scarsdale. 10583. Tel 914-472-6447. MD 64. D-O. SE. Am Ac Oph. Am Med Wom. Asst Oph Yonkers Genl. Asst Oph Union. Asst Oph White Plains. Also Manhattan.

★ **LAVYNE, Michael Hugh,** 525 E 68 St. Manhattan. 10021. Tel 212-472-8398. CORNELL 72. D-NS. SM-9. FACS. Am Neur Sur. Attd Neurosur New York. (AMA)

★ **LINDEN, Peter James,** 465 Park Ave. Manhattan. 10022. Tel 212-752-4888. ROCH 69. D-PS. SM-7. Am Pl & Recon Sur. Asst Plast Sur NY E&E Inf. Asst Plast Sur St Clares. Asst Plast Sur Cabrini Med Ctr. Asst Plast Sur NY Inf-Beekman. (AMA)

★ **LOBBATO, Vincent J,** 222 E 19 St. Manhattan. 10009. Tel 212-475-0190. UNIV GUERRERO 78. D-S. A. NYS Sur. Attd Gen Sur Cabrini Med Ctr. (AMA)

★ **MEISTER, Bertram,** 984 N Broadway. Yonkers. 10701. Tel 914-965-2121. LAUSANNE 55. SH. Am Ac Derm. Am Ger. NYS Derm. Chief & Attd Derm Yonkers Genl. Attd Derm St Johns Riverside. Asst Derm Bx Munic. Attd Derm Blythedale. (AMA)

★ **NAUMBURG, Jr., George W,** 15 E 91 St. Manhattan. 10128. Tel 212-534-7222. YALE 45. D-PN(P). SI-2 Am Psych. Am Psychoan. Am Adolesc Psych. Asso Adolesc Psych Mt Sinai. (AMA)

★ **PARRISH, Louis,** 148 E 84 St. Manhattan. 10028. Tel 212-737-3636. LA 50. SI-2. Am Psych. Am Ac FP.

★ **ROSMAN, Lawrence David,** 109-33 71 Rd-Suite 2E. Forest Hills. 11375. Tel 718-263-3718. NYU 75. D-IM(END). FACP. Endocr. Clin Soc NY Diabet. Attd Int Med Booth Meml. Attd Endocr Booth Meml. Attd Endocr Hillcrest. Attd Phy Endocr Parkway. Also Manhattan.

★ **SCHREIBER, David,** 854 E Broadway. Long Beach. 11561. Tel 516-431-6789. LRCP&S GLASGOW 36. D-PM(OM). SM-10. FACPM. Am Occ Med. Am Pub Hlth. (AMA)

★ **SETTEMBRINI, Leonard V,** Mount Vernon NY. The appointment Attd Oph Mt Vernon Hosp was inadvertently omitted from the Hosp Staff List section of the Medical Directory.

★ **SHAPIRO, Harvey Nathan,** 4 Crescent Rd. Great Neck. 11021. Tel 516-487-1680. NYU 48. D-IM. SJ. Clin Soc NY Diabet. NYS Int Med. Staff Int Med LIJHMC. Sr Asst Int Med No Shore Univ.

★ **SOBEL, Sidney H,** Rochester NY. Was incorrectly listed in the Hosp Staff List section of the Medical Directory. The correct listing should read—Chief Radiation-Oncologist Genesee Hosp.

★ **STANTON, Sherry Frances,** 4300 Hylan Blvd. Staten Island. 10312. Tel 718-356-3608. See Manhattan.

★ **STRAM, Richard Allan,** Plaza Dr & Vestal Pkwy. Binghamton. 13903. Tel 607-798-9356. NY MED 78. B. Attd Orth Sur United Hlth Service. Attd Orth Sur Binghamton Psych. Attd Orth Sur Lourdes. (AMA)

★ **WINEBURG, Elliot N,** 30 Central Park South. Manhattan. 10019. Tel 212-308-1368. ZURICH 56. D-PN(P). SI-2. Am Psych. Am Clin Hypno. Asst Psych Mt. Siani. Attd Psych Parkway. Also Queens. (AMA)

★ **WOLIN, Charles Allen,** 280 Mamaroneck Ave. White Plains. 10605. Tel 914-949-0202. BASEL 60. SJ. Asst Phy White Plains. Asso Phy St Agnes.

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Edwin Jerome Adlerman, MD, Bronxville. Died January 22, 1984; age 67. He was a 1940 alumnus of Georgetown University School of Medicine. Dr Adlerman was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Urology. His memberships included the American Urological Association, the American Academy of Compensation Medicine, Inc, the American Occupational Medical Association, the New York County Medical Society and the Medical Society of the State of New York.

John T. Alexander, MD, Huntington. Died October 7, 1984; age 65. He was a 1949 graduate of Harvard Medical School. Dr Alexander was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

William D. Arnold, MD, New York. Died October 4, 1984; age 59. He was a 1948 alumnus of Cornell University Medical College. Dr Arnold was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. His memberships included the Academy of Medicine, the American Academy of Orthopaedic Surgeons, the American Association for the Surgery of Trauma, the New York County Medical Society and the Medical Society of the State of New York.

Clifton H. Berlinghof, MD, Binghamton. Died October 16, 1984; age 86. Dr Berlinghof was a 1925 alumnus of Johns Hopkins University School of Medicine. He was a Fellow of the American College of Physicians, and a member of the New York State Society of Internal Medicine, the Broome County Medical Society and the Medical Society of the State of New York.

David B. Blumenfeld, MD, New York. Died October 6, 1984; age 86. Dr Blumenfeld was a 1923 alumnus of the State University of New York Downstate Medical Center. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Jacob Maurice Bloom, MD, New York. Died August 22, 1984; age 90. Dr Bloom was a 1920 graduate of New York University College of Medicine. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Robert Abbe Chait, MD, Rockville Centre. Died September 29, 1984; age 75. He was a 1935 graduate of the Université de Paris Faculté de Médecine. Dr Chait was a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology, the American Academy of Allergy, the Nassau County Medical Society and the Medical Society of the State of New York.

Simeon O. Chlenoff, MD, New York. Died May 7, 1984; age 89. He was a 1944 graduate of the Universität Bern Medizinische Fakultät, Bern. Dr Chlenoff was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the New York Pathological Society, the American Psychiatric Association, the Academy of Psychoanalysis, the New York County Medical Society and the Medical Society of the State of New York.

John Gordon Cole, MD, New York. Died February 6, 1984; age 76. He was a 1932 alumnus of the University of Minnesota Medical School. Dr Cole was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Ophthalmology. His memberships included the Academy of Medicine, the New York Ophthalmology Society, the American Academy of Ophthalmology and Otolaryngology, the American Ophthalmological Society, the New York County Medical Society and the Medical Society of the State of New York.

Henry S. F. Cooper, MD, New York. Died September 10, 1984; age 89. Dr Cooper was a 1924 alumnus of Harvard Medical School. He was a member of the Academy of Medicine, the New York County Medical Society and the Medical Society of the State of New York.

George Dacks, MD, Livonia. Died April 7, 1984; age 81. Dr Dacks was a 1934 graduate of the University of Rochester School of Medicine and Dentistry. He was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Joseph Theodore Eagan, MD, Oneonta. Died September 7, 1984; age 88. Dr Eagan was a 1921 alumnus of Yale University School of Medicine. He was a member of the American Thoracic Society, the Medical Society of the County of Otsego and the Medical Society of the State of New York.

Max Ellenberg, MD, New York. Died August 18, 1984; age 73. He was a 1935 alumnus of New York University College of Medicine. Dr Ellenberg was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the Clinical Society of the New York Diabetes Association, the New York County Medical Society and the Medical Society of the State of New York.

Solomon Salmen Fiderer, MD, Brooklyn. Died October 1, 1984; age 91. He was a 1925 alumnus of the Medizinische Fakultät der Universität, Wien. Dr Fiderer was a member of the American Academy of Family Physicians, the Medical

Society of the County of Kings and the Medical Society of the State of New York.

Morris H. Goldenberg, MD, Tamarac, Florida. Died September 28, 1984; age 80. He was a 1929 graduate of George Washington University School of Medicine. Dr Goldenberg was a Fellow of the American College of Radiology and a Diplomate of the American Board of Radiology. His memberships included the Radiological Society of New York State, the New York Roentgen Society, the American Public Health Association, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Rudolph Victor Gorsch, MD, Vernon, Connecticut. Died October 13, 1984; age 96. Dr Gorsch was a 1918 alumnus of Columbia University College of Physicians and Surgeons. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

James Henry Gray, Jr, MD, Ransomville. Died September 23, 1984; age 74. He was a 1934 graduate of the University of Buffalo School of Medicine. Dr Gray was a member of the Medical Society of the County of Niagara and the Medical Society of the State of New York.

Joseph Guccione, MD, Ridgewood. Died September 26, 1984; age 58. Dr Guccione was a 1959 graduate of the Università degli Studi di Roma, Facoltà di Medicina e Chirurgia, Roma. He was a member of the Academy of Medicine, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Gordon M. Hemmett, MD, Rochester. Died September 15, 1984; age 74. He was a 1938 graduate of McGill University Faculty of Medicine, Montreal. Dr Hemmett was a Fellow of the American College of Preventive Medicine and a Diplomate of the American Board of Preventive Medicine, Inc. His memberships included the Academy of Medicine, the American Academy of Occupational Medicine, the American Occupational Medical Association, the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Henry Hillel, MD, Syracuse. Died September 20, 1984; age 89. Dr Hillel was a 1921 alumnus of Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin. He was a Qualified Fellow of the International College of Surgeons, and a member of the Onondaga County Medical Society and the Medical Society of the State of New York.

William J. Keefe, MD, Wilmington, Delaware. Died June 3, 1984; age 94. Dr Keefe was a 1918 graduate of McGill University Faculty of Medicine, Montreal. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Morris Kimberg, MD, Lake Worth, Florida. Died October 6, 1984; age 92. Dr Kimberg was a 1915 alumnus of New York University College of Medicine. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Hans Kuehn, MD, New York. Died June 13, 1984; age 83. Dr Kuehn was a 1926 alumnus of the Medizinische Fakultät der Universität, Wien. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Hans Kulka, MD, Wurtsboro. Died September 13, 1984; age 70. Dr Kulka was a 1942 alumnus of the Universität Zurich Medizinische Fakultät, Zurich. He was a member of the Medical Society of the County of Orange and the Medical Society of the State of New York.

Rene Pierre Lacombe, MD, New York. Died March 3, 1984; age 84. Dr Lacombe was a 1936 alumnus of the Université de Paris Faculté de Médecine. His memberships included the Academy of Medicine, the American Psychoanalytic Association, the American Psychiatric Association, the New York County Medical Society and the Medical Society of the State of New York.

Richard F. Mascola, MD, Jamaica. Died October 14, 1984; age 76. Dr Mascola was a 1933 alumnus of the University of Vermont College of Medicine. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Joseph Bryant Mathewson, MD, Ithaca. Died September 18, 1984; age 83. He was a 1926 alumnus of Cornell University Medical College. Dr Mathewson was a member of the Medical Society of the County of Tompkins and the Medical Society of the State of New York.

Luigi Mazza, MD, Rome, Italy. Died May 23, 1984; age 55. He was a 1951 alumnus of the Università degli Studi di Roma, Facoltà di Medicina e Chirurgia, Roma. Dr Mazza was a Fellow of the American College of Anesthesiologists and a Diplomate of the American Board of Anesthesiology. His memberships included the American Society of Anesthesiologists, Inc, the New York County Medical Society and the Medical Society of the State of New York.

Edward P. McDonald, MD, Albany. Died September 21, 1984; age 82. He was a 1926 graduate of the Jefferson Medical College of Philadelphia. Dr McDonald was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Medical Society of the County of Albany and the Medical Society of the State of New York.

Milton Mendelson, MD, Central Valley. Died September 16, 1984; age 81. He was a 1930 alumnus of the State University of New York Upstate Medical Center. Dr Mendelson was a member of the Medical Society of the County of Orange and the Medical Society of the State of New York.

Armand Major Patella, MD, Brooklyn. Died May 6, 1984; age 72. Dr Patella was a 1940 graduate of the State University of New York Downstate Medical Center. He was a member of the American Academy of Family Practice, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Geremia Rizzuti, MD, Glen Cove. Died October 6, 1984; age 85. Dr Rizzuti was a 1923 graduate of the Università di Napoli Facoltà di Medicina e Chirurgia, Napoli, and was a member of the Clinical Society of the New York Diabetes Association, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Domenico L. Romano, MD, New York. Died August 29, 1984; age 85. He was a 1927 graduate of the Università degli Studi di Bologna, Facoltà di Medicina e Chirurgia, Bologna. Dr Romano was a Diplomate of the American Board of Dermatology, Inc. He was a member of the American Occupational Medical Association, the New York County Medical Society and the Medical Society of the State of New York.

Livia Halasz Rosenfeld, MD, New York. Died August 16, 1984; age 75. Dr Rosenfeld was a 1933 graduate of the Medizinische Fakultät der Universität, Wien. She was a member of the American Fertility Society, the New York County Medical Society and the Medical Society of the State of New York.

Mitchell Jay Rubinow, MD, Miami Beach, Florida. Died September 9, 1984; age 83. He was a 1926 graduate of the State University of New York Downstate Medical Center. Dr Rubinow was a member of the American Academy of Family Physicians, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Jerome M. Seides, MD, Nanuet. Died September 9, 1984; age 65. He was a 1943 alumnus of New York University College of Medicine. Dr Seides was a Fellow of the American Psychiatric Association and a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the American Psychoanalytic Association, the Medical Society of the County of Rockland and the Medical Society of the State of New York.

Avram N. Selinger, MD, Cliffside Park, New Jersey. Died August 1, 1984; age 70. Dr Selinger was a 1944 alumnus of Universitatea din Lasi Facultatea de Medicina, Lasi. He was a member of the New York Cardiological Society, the New York County Medical Society and the Medical Society of the State of New York.

David Raymond Shapiro, MD, Newburgh. Died June 26, 1984; age 63. Dr Shapiro was a 1945 graduate of McGill University Faculty of Medicine, Montreal. He was a Fellow of the American College of Anesthesiologists, and a member of the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Orange and the Medical Society of the State of New York.

Dean Davidge Smith, MD, Naples, Florida. Died April 12, 1984; age 79. He was a 1934 graduate of Johns Hopkins University School of Medicine. Dr Smith was a member of the Broome County Medical Society and the Medical Society of the State of New York.

James D. Van Doren, MD, Syracuse. Died August 1, 1984; age 57. He was a 1952 graduate of Cornell University Medical College. Dr Van Doren was a Diplomate of the American Board of Obstetrics and Gynecology, and a member of the Onondaga County Medical Society and the Medical Society of the State of New York.

Leo G. Weishaar, MD, Nyack. Died August 17, 1984; age 93. Dr Weishaar was a 1915 graduate of New York University College of Medicine. He was a member of the Medical Society of the County of Rockland and the Medical Society of the State of New York.

Lanpher Chas Weston, MD, Massena. Died September 22, 1984; age 69. He was a 1943 graduate of the State University of New York Upstate Medical Center. Dr Weston was a Qualified Fellow of the International College of Surgeons, and a member of the Medical Society of the County of St. Lawrence and the Medical Society of the State of New York.

George Albert Zak, MD, New York. Died September 2, 1984; age 71. He was a 1937 alumnus of the Medizinische Fakultät der Universität, Wien. Dr Zak was a Fellow of the American College of Chest Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the American Thoracic Society, the New York County Medical Society and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089–1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), an abbreviated title of no greater than 40 spaces for use as a running foot, and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 2,500 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 750 and 1,000 words. Other kinds of original essays should not exceed 2,000 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear on the text. When there are six or fewer authors, all should be listed; where there are seven or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

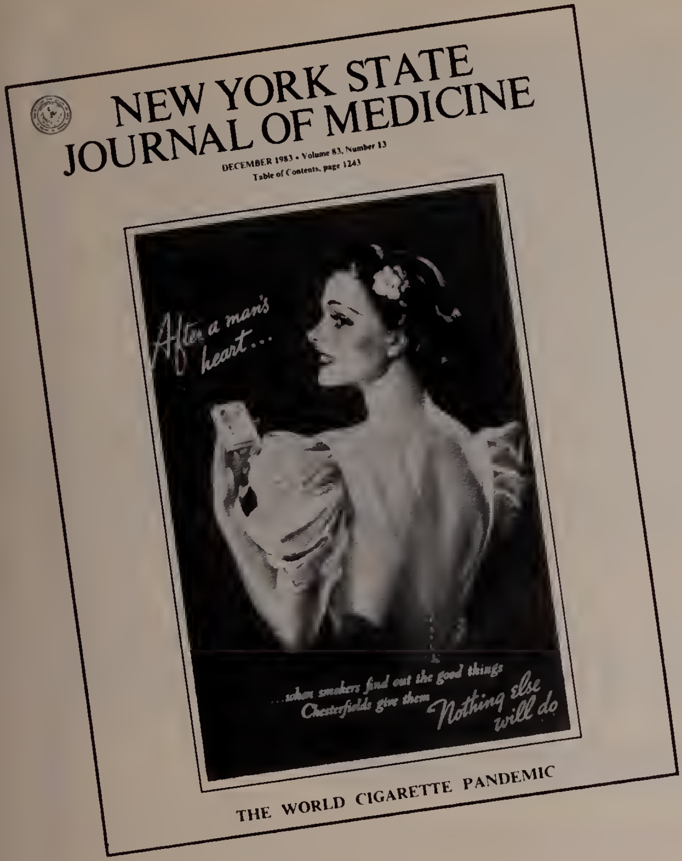
1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. McGuigan JE. Peptic ulcer. In: Petersdorf RG, Adams RD, Braunwald E, Isselbacher KJ, Martin JB, Wilson JD, eds. ***Harrison's Principles of Internal Medicine.*** New York, McGraw-Hill, 1983, pp 1697–1712.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Alan Blum, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

The definitive analysis of the world cigarette pandemic


NEW YORK STATE JOURNAL OF MEDICINE

DECEMBER 1983 • Volume 83, Number 13

Table of Contents, page 1243

THE WORLD CIGARETTE PANDEMIC

After a man's heart...
...when smokers find out the good things Chesterfields give them
Nothing else will do

"STIMULATING, AUTHORITATIVE, and timely review for every physician and medical student."

JESSE L. STEINFELD, M.D.
Surgeon General, U.S.
Public Health Service
1969-1973

"ENOUGH STORY IDEAS HERE TO CHOKE A CAMEL and maybe the entire tobacco industry as well . . . Fifty-odd articles, by experts from the U.S. and abroad, examine the tobacco problem in the most concrete of clinical, political, economic, legal, and ethical terms, while dozens of samples drawn from five decades of cigarette advertising trace the marketing history of the weed from early claims of medical endorsement to current pitches to women, children, and the third world."

COLUMBIA JOURNALISM REVIEW

An extra press run has enabled the NEW YORK STATE JOURNAL OF MEDICINE to offer single or bulk copies of its recent issue on the world cigarette pandemic. As the official publication of the Medical Society of the State of New York since 1900, the *Journal* is one of the foremost independent peer-reviewed general medical journals in the country. Providing timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news, the *Journal* makes important reading—and a welcome gift—to physicians and students in the United States or abroad. A free copy of the December issue will be included with each subscription.

Quantity	**Per Copy***
Up to 10	$5.00
11-25	$4.50
26-100	$4.00
over 100	$3.50
over 1,000	$3.00

ORDER FORM

Name______________________________

Address______________________________

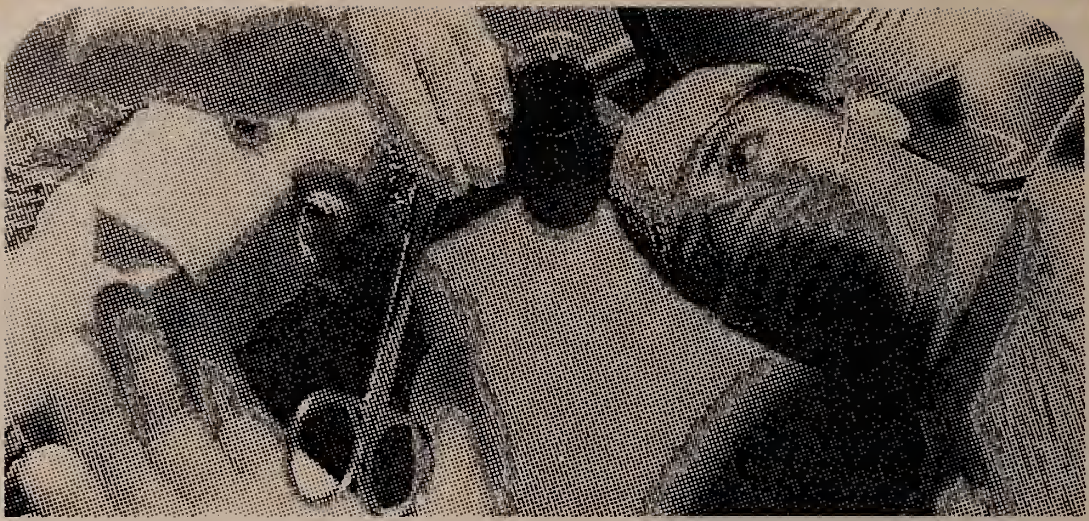
City/State/Zip______________________________

Please send________copies of December 1983 issue of the Journal.

Enter my subscription to the Journal. ☐ $25.00 U.S. & Canada; ☐ $30.00 Foreign

Total amount enclosed $__________ ***Note:** Domestic postage included. Please add New York state & local sales tax where applicable, or sales tax exemption certificate. Foreign postage additional.

Mail to: Circulation Department, **New York State Journal of Medicine**
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.



A special practice for specialists

If you're a surgeon or OB/GYN or other medical specialist, the Air Force may have a special practice for you.

What makes it special? You'll enjoy an excellent pay and benefits package. Your regular working hours will allow you to spend more time with your family. You'll receive 30 days of vacation with pay each year. And you will work with modern equipment and some of the most highly trained professionals in the world, serving your country and your patients. Now that's special!

Find out just how special your practice can be.

Contact: Major Elaine F. Laub (212) 264-8663

AIR FORCE

A great way of life.

FOR PHYSICIANS AND SURGEONS WITH A PART-TIME EXPOSURE ONLY

Now offering Medical Malpractice Insurance on an ***occurrence basis*** at drastically reduced rates thru Frontier Insurance Co., Monticello, NY.

With the tail on claims made policies, you might lose your shirt.

Call or write for further information and application.

MEDICAL
MQ
QUADRANGLE

Medical Quadrangle, Inc.
P. O. Box 456
318 Bedford Road
Bedford Hills, New York 10507
914 666-0170
212 829-3654
"Specializing in Medical Liability Insurance"

Info: Extended Programs in Medical Education, Room 569-U, University of California, San Francisco, CA 94143. Tel: (415) 666-4251.

Feb 14–16. **Annual Oncology Review.** 20 Cat 1 credits. Los Angeles. Info: UCLA Extension Department of Health Sciences, 10995 Le Conte Ave, Rm 614, Los Angeles, CA 90024. Tel: (213) 825-1901.

COLORADO

Feb 2–9. **Diagnostic Radiology.** 21 Cat 1 credits. Aspen. Info: Extended Programs in Medical Education, Room 569-U, University of California, San Francisco, CA 94143. Tel: (415) 666-4251.

Feb 2–9. **Eleventh Annual Conference in Anesthesiology.** *Feb 23–Mar 2.* **Tenth Annual Symposium in Intensive Care.** *Feb 23–Mar 2.* **Principles of Practice Management.** Vail. Info: Professional Seminars, PO Box 012318, Miami, FL 33101. Tel: (305) 547-6411.

FLORIDA

Feb 7–9. **The Nineteenth Annual Symposium on Cosmetic Surgery.** Miami. Info: Thelma MacGregor, Cedars Medical Center, 1400 NW 12th Ave, Miami, FL 33136.

Feb 7–10. **The Fort Lauderdale Conference on Pediatrics.** 20 Cat 1 credits. Fort Lauderdale. Info: Rayna Saville, CME Coordinator, Children's Hospital of Buffalo, 219 Bryant St, Buffalo, NY 14222. Tel: (716) 878-7630.

Feb 11–15. **Advances in Gene Technology: Molecular Biology of the Immune System.** Miami. Info: University of Miami, PO Box 016129, Miami, FL 33101.

Feb 17–22. **Current Problems in Cardiovascular Diseases.** 21 Cat 1 credits. Lake Buena Vista. Info: Postgraduate Courses, Mayo Clinic-Mayo Foundation, Rochester, MN 55905.

Feb 18–20. **February Course in Arrhythmias.** Fort Lauderdale. Info: Dr. Jules Constant, Cardiac Study Fund, Box 114, Hiler Branch, Buffalo, NY 14223. Tel: (716) 836-5172.

Feb 21–24. **The Twelfth Pediatric Dermatology Seminar.** Info: Guinter Kahn, MD, 16800 NW 2nd Ave, Ste 401, Miami, FL 33169. Tel: 1-800-327-6334.

LOUISIANA

Feb 14–16. **New Concepts in Management of Cardiovascular Diseases.** New Orleans. Info: American College of Cardiology, Extramural Programs Department, 9111 Old Georgetown Road, Bethesda, MD 20814. Tel: (301) 897-5400, Ext. 230.

MARYLAND

Feb 4–6. **Rehabilitation of the Coronary Patient.** *Feb 11–13.* **Cardiology Update for Intensive Care Unit Nurses.** *Feb 20–22.* **Thromboembolic and Atherosclerotic Disease—An Update on**

PRE-REGISTER NOW

MEDICLINICS

POSTGRADUATE MEDICAL REFRESHER COURSE

FORT LAUDERDALE, FLORIDA

MARCH 4 - MARCH 15, 1985

PRACTICAL UPDATE FOR PRIMARY CARE PHYSICIANS

50 CATEGORY 1 CREDIT HOURS

LIMITED 25 CREDIT ONE WEEK COURSE AVAILABLE

This program has been reviewed and is acceptable for 50 Prescribed Hours by the American Academy of Family Physicians. As an organization accredited for continuing medical education, the Florida Academy of Family Physicians designates this program as meeting the criteria for 50 credit hours in Category 1 of the Physician's Recognition Award of the American Medical Association. This program also is acceptable for 50 Mandatory hours by the Florida Medical Association.

PRE-REGISTRATION — $450.00

(UNTIL FEBRUARY 15, 1985)

FOR INFORMATION CONTACT: MEDICLINICS
2917 South Ocean Blvd., Suite 905
Highland Beach, Florida 33431
(305) 272-8973

EXCELLENT FACULTY, PEAK OF WINTER SEASON

CHOICE OF 300 HOTELS & MOTELS

Pathogenesis, Prevention and Treatment.

Feb 25–27. **Cardiovascular Surgery: Coronary—Extending the Procedure; Valvular—Matching the Patient, Challenging Management Problems.** Bethesda. Info: American College of Cardiology, Learning Center Registrar, 9111 Old Georgetown Rd, Bethesda, MD 20814. Tel: (301) 897-5400, Ext 241.

NEVADA

Feb 1–3. **Primary Care in Sports Medicine for General Practitioners.** 13 Cat 1 credits. Las Vegas. Info: Vickie Zomar Beckwith, RN, National Athletic Health Institute, 575 E. Hardy St, Inglewood, CA 90301. Tel: (213) 674-1600.

NEW JERSEY

Feb 28–Mar 1. **Critical Issues in Psychiatry.** Atlantic City. Info: Gene O' Neill, 45 Louella Ct, Wayne, PA 19087. Tel: (609) 854-4081 or (215) 688-7582.

TEXAS

Feb 26–Mar 1. **The 38th Annual Symposium on Fundamental Cancer Research.** Houston. Info: Office of Conference Services, Box 131, MD Anderson Hospital and Tumor Institute, 6723 Bertner Ave, Houston, TX 77030. Tel: (713) 792-2222.

UTAH

Feb 2–9. **Clinical Topics in Internal Medicine.** Snowbird. Info: Office of CME Scott and White, Temple, TX 76508. Tel: (817) 774-2350.

Feb 3–8. **New Clinical Applications in Cardiac Imaging and Therapeutic Interventions.** Park City. Info: American College of Cardiology, Extramural Programs Department, 9111 Old Georgetown Rd, Bethesda, MD 20814. Tel: (301) 897-5400, Ext 320.

Feb 16–23. **Office Based Sports Medicine.** 27½ Cat 1 credits. Park City. Info: Extended Programs in Medical Education, School of Medicine, Rm 569-U, University of California, San Francisco, CA 94143. Tel: (415) 666-4251.

Feb 23–Mar 2. **Fifth Annual Pulmonary and Critical Care Medicine: A Practical Approach.** Park City. Info: Extended Programs in Medical Education, Room 569-U, University of California, San Francisco, CA 94143. Tel: (415) 666-4251.

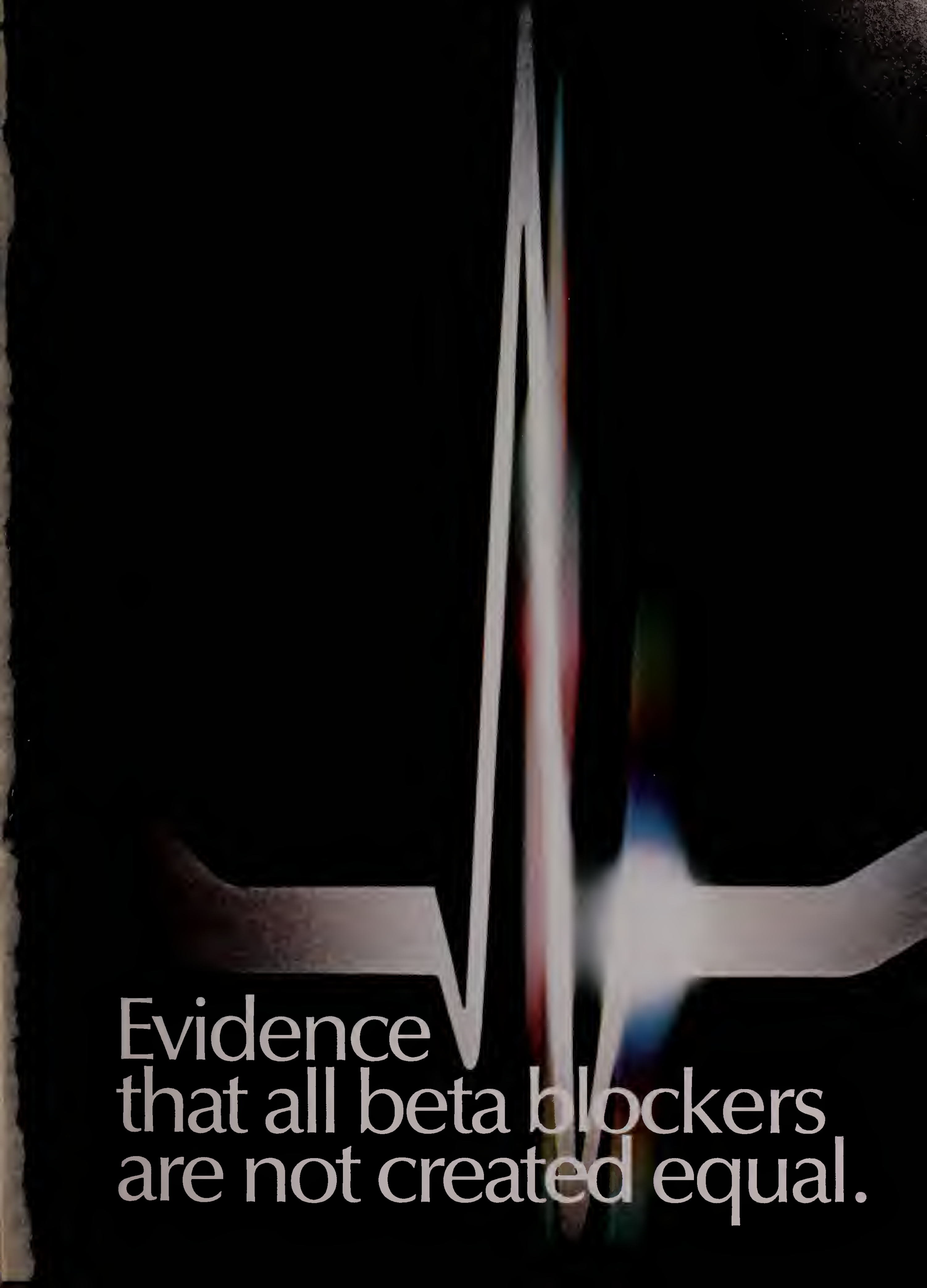
VERMONT

Feb 18–22. **Perspectives in Critical Care Medicine.** Jeffersonville. Info: Ellis Hospital, Critical Care Center, 1101 Nott St, Schenectady, NY 12308. Tel: (518) 382-4135.

AROUND THE WORLD

PUERTO RICO

Feb 5–8. **Advances in Noninvasive Diagnosis and Therapy—1985.** Humacao. Info: American College of Cardiology. Extramural Programs Department. 9111 Old Georgetown Rd, Bethesda MD 20814. Tel: (301) 897-5400, Ext 230.


Evidence
that all beta blockers
are not created equal.

New studies uncover the potassium effects of beta-2 blockade

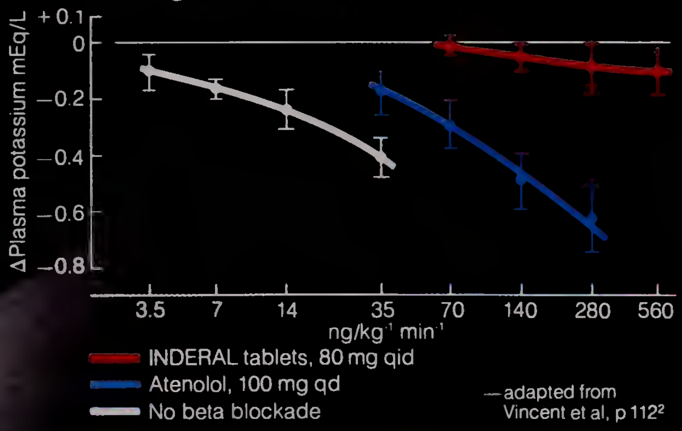
Clinical pharmacology data from the *New England Journal of Medicine:*

"...when normal young men are given infusions of epinephrine at levels such as those that circulate in patients with myocardial infarction, their serum potassium concentrations fall by about 0.8 mmol per liter. Hypokalemia is prevented by...beta-2 blockade."[1]

INDERAL (propranolol HCl) prevented beta-2 mediated hypokalemia

In a pharmacological study comparing INDERAL with atenolol, 10 hypertensive patients were infused with the nonselective beta agonist, isoproterenol, which also stimulates beta-2 mediated hypokalemia. At doses high enough to overcome beta-1 mediated heart rate reductions, the hypokalemia caused by isoproterenol was blunted by INDERAL, but not by atenolol.[2]

INDERAL compared with atenolol: change in plasma potassium after beta-agonist infusion[2]



—adapted from Vincent et al, p 112[2]

For beta-1/beta-2 blockade

INDERAL®

(PROPRANOLOL HCl) TABLETS

Please see last page for brief summary of prescribing information.

Epinephrine-induced hypokalemia can intensify diuretic-induced hypokalemia

From the *Lancet:*

"Although both diuretics and adrenaline are known to cause hypokalaemia, we believe that this is the first demonstration that these two factors can act in an additive manner. Routine monitoring of serum potassium in patients on thiazide diuretics may underestimate the risks of hypokalaemia."[3]

Epinephrine drives potassium into cells—an effect that has been shown to be under beta-2 receptor control[4]; thiazides promote excretion of potassium. When these actions occur together, hypokalemia can intensify significantly.[3]

Epinephrine-induced hypokalemia can cause ECG abnormalities typical of other forms of hypokalemia

In a study of the effects of epinephrine-induced hypokalemia on the electrocardiogram, it was shown that levels of plasma epinephrine similar to those observed during myocardial infarction can produce changes in ventricular repolarization, which are reflected in T-wave flattening and QT prolongation in normal subjects.[5]

In clinical pharmacology studies of hypertensive and normal patients, epinephrine-induced hypokalemia was prevented by beta-2 blockade.[2,4]

For beta-1/beta-2 blockade

INDERAL®

(PROPRANOLOL HCl) TABLETS

Please see last page for brief summary of prescribing information.

Right from the start in hypertension…

Once-daily INDERAL LA (propranolol HCl) for smooth blood pressure control without the potassium problems of diuretics

Patients with "uncomplicated" hypertension may develop ventricular arrhythmias in the presence of hypokalemia.[6] Once-daily INDERAL LA maintains smooth blood pressure reductions without a negative effect on serum potassium and provides patients with broad cardiovascular benefits–all in a convenient daily dose.

Like conventional INDERAL tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.

And for lifetime benefits in foundation treatment of angina

Once-daily INDERAL LA provides 24-hour control of angina symptoms–plus the cardiovascular benefits of the world's leading beta blocker. And unlike calcium channel blockers, INDERAL LA, alone or with a nitrate, is recommended for first-line treatment of stable angina.

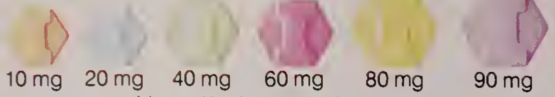
Simply start new patients on 80 mg INDERAL LA once daily. Dosage may be increased to 160 mg once daily to achieve maximal control.

Once-daily
INDERAL LA
(PROPRANOLOL HCl) LONG-ACTING CAPSULES
In HYPERTENSION
and ANGINA

The appearance of these capsules is a registered trademark of Ayerst Laboratories
Please see last page for brief summary of prescribing information

INDERAL®

(PROPRANOLOL HCl) TABLETS

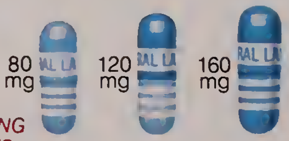


The appearance of these tablets is a registered trademark of Ayerst Laboratories

Once-daily

INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES



The appearance of these capsules is a registered trademark of Ayerst Laboratories

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULARS.)*

INDERAL® (propranolol hydrochloride) **Tablets and Injectable**
INDERAL® LA (propranolol hydrochloride) **Long Acting Capsules**

INDICATIONS AND USAGE—INDERAL Tablets

Hypertension: INDERAL (propranolol hydrochloride) is indicated in the management of hypertension It may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL is indicated for the long-term management of patients with angina pectoris

Cardiac Arrhythmias:

1.) Supraventricular arrhythmias

a) Paroxysmal atrial tachycardias, particularly those arrhythmias induced by catecholamines or digitalis or associated with the Wolff-Parkinson-White syndrome (See W-P-W under WARNINGS.) b) Persistent sinus tachycardia which is noncompensatory and impairs the well-being of the patient. c) Tachycardias and arrhythmias due to thyrotoxicosis when causing distress or increased hazard and when immediate effect is necessary as adjunctive, short term (2-4 weeks) therapy May be used with, but not in place of, specific therapy (See Thyrotoxicosis under WARNINGS.) d) Persistent atrial extrasystoles which impair the well-being of the patient and do not respond to conventional measures. e) Atrial flutter and fibrillation when ventricular rate cannot be controlled by digitalis alone, or when digitalis is contraindicated

2) Ventricular tachycardias

Ventricular arrhythmias do not respond to propranolol as predictably as do the supraventricular arrhythmias. a)Ventricular tachycardias. With the exception of those induced by catecholamines or digitalis, INDERAL is not the drug of first choice In critical situations when cardioversion technics or other drugs are not indicated or are not effective, INDERAL may be considered If, after consideration of the risks involved, INDERAL is used, it should be given intravenously in low dosage and very slowly (See DOSAGE AND ADMINISTRATION) *Care in the administration of* INDERAL *with constant electrocardiographic monitoring is essential* as *the failing heart requires* some *sympathetic drive for maintenance of myocardial tone.* b) Persistent premature ventricular extrasystoles which do not respond to conventional measures and impair the well-being of the patient

3.) Tachyarrhythmias of digitalis intoxication

If digitalis-induced tachyarrhythmias persist following discontinuance of digitalis and correction of electrolyte abnormalities, they are usually reversible with *oral* INDERAL. Severe bradycardia may occur (See OVERDOSAGE.) Intravenous propranolol hydrochloride is reserved for life-threatening arrhythmias Temporary maintenance with oral therapy may be indicated. (See DOSAGE AND ADMINISTRATION in the package circulars.)

4) Resistant tachyarrhythmias due to excessive catecholamine action during anesthesia

Tachyarrhythmias due to excessive catecholamine action during anesthesia may sometimes arise because of release of endogenous catecholamines or administration of catecholamines. When usual measures fail in such arrhythmias, INDERAL may be given intravenously to abolish them. All general inhalation anesthetics produce some degree of myocardial depression. Therefore, when INDERAL (propranolol hydrochloride) is used to treat arrhythmias during anesthesia, it should be used with extreme caution and constant ECG and central venous pressure monitoring. (See WARNINGS.)

Myocardial Infarction: INDERAL is indicated to reduce cardiovascular mortality in patients who have survived the acute phase of myocardial infarction and are clinically stable.

Migraine: INDERAL is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta receptor stimulation Clinical improvement may be temporary

Pheochromocytoma: After primary treatment with an alpha-adrenergic blocking agent has been instituted, INDERAL may be useful as *adjunctive* therapy if the control of tachycardia becomes necessary before or during surgery

It is hazardous to use INDERAL unless alpha-adrenergic blocking drugs are already in use, since this would predispose to serious blood pressure elevation. Blocking only the peripheral dilator (beta) action of epinephrine leaves its constrictor (alpha) action unopposed

In the event of hemorrhage or shock, there is a disadvantage in having both beta and alpha blockade since the combination prevents the increase in heart rate and peripheral vasoconstriction needed to maintain blood pressure.

With inoperable or metastatic pheochromocytoma, INDERAL may be useful as an adjunct to the management of symptoms due to excessive beta receptor stimulation

INDICATIONS AND USAGE—INDERAL LA Long Acting Capsules

Hypertension: INDERAL LA (propranolol hydrochloride) is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL and INDERAL LA are contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block; 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL (propranolol hydrochloride) therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)— PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL, like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g., dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA· Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin.

THYROTOXICOSIS· Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General:* Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL is not indicated for the treatment of hypertensive emergencies.

Beta adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure

Clinical Laboratory Tests: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy: Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers: INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular: bradycardia; congestive heart failure; intensification of AV block; hypotension; paresthesia of hands; thrombocytopenic purpura; arterial insufficiency, usually of the Raynaud type.

Central Nervous System: Lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances; hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics.

Gastrointestinal: nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory: bronchospasm.

Hematologic: agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous: alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

REFERENCES.

1. Epstein FH, Rosa RM Adrenergic control of serum potassium *N Engl J Med* 1983;309:1450-1451. **2.** Vincent HH, Boomsma F, Man in't Veld AJ, et al: Effects of selective and nonselective β-agonists on plasma potassium and norepinephrine. *J Cardiovasc Pharmacol* 1984;6:107-114. **3.** Struthers AD, Whitesmith R, Reid JL: Prior thiazide diuretic treatment increases adrenaline-induced hypokalaemia. *Lancet* 1983;1:1358-1361. **4.** Brown MJ, Brown DC, Murphy MB: Hypokalemia from beta$_2$-receptor stimulation by circulating epinephrine. *N Engl J Med* 1983;309:1414-1419. **5.** Struthers AD, Reid JL, Whitesmith R, et al: Effect of intravenous adrenaline on electrocardiogram, blood pressure, and serum potassium. *Br Heart J* 1983;49:90-93 **6.** Holland OB, Nixon JV, Kuhnert L: Diuretic-induced ventricular ectopic activity. *Am J Med* 1981;70:762-768.

9410/1184

Ayerst®
AYERST LABORATORIES
New York, N.Y. 10017

Copyright © 1984 AYERST LABORATORIES
Division of AMERICAN HOME PRODUCTS CORPORATION

Motrin®
ibuprofen, Upjohn
600 mg Tablets

More convenient for your patients.

Upjohn

© 1984 The Upjohn Company

The Upjohn Company • Kalamazoo, Michigan 49001 USA

J-4044 January 1984

NEW LAW CUTS TAX BREAKS FOR LUXURY CARS

If the car you want costs more than $16,667.00 don't spend your money, use ours—we still pass the investment tax credit to you.

If you're in the 50% tax bracket no longer can you save half the depreciation on anything over $16,677.00 for the first 3 years:

HOWEVER,

When you lease you can continue to deduct your monthly rental payments in excess of $16,000.00 *provided* you conform to the limits and rules set by the I.R.S. regarding usage. The leasee must use the vehicle more than 50% of the time for his business or profession.

SO DOESN'T IT MAKE SENSE TO LEASE NOW!

ALL MAKES & MODELS AVAILABLE.

CALL CAROL LEASING INC.

(516) 222-9041

ROOSEVELT RACEWAY, WESTBURY, NY 11590

Index to Advertisers

Ayerst Laboratories
. . . . 1A,2A,3A,4A,21A,22A,23A,
. . . . 24A, 25A,26A,27A,28A
Blue Cross/Blue Shield of Greater N.Y. . . . 15A
Classified Advertising 9A,14A,30A,32A,33A,36A
Eastman Kodak 5A,6A,7A
Eli Lilly and Co. . . . . 2nd Cover
Marion Laboratories 37A,38A
Mediclinics 20A
Medical Quadrangle 19A
A.H. Robbins 11A
Roche Laboratories
. . . . 34A,35A,36A,3rd Cover,4th Cover
Stuart Pharmaceuticals 31A,32A
U.S. Air Force 18A
Upjohn Company 29A
Wyeth Laboratories 12A,13A,14A

MISCELLANEOUS

MAGNIFICENT PORTRAITS. We are well known in the medical community for our beautiful portraits of doctors and their families. Our work is outstanding in oils or photography, from a single sitting or any available photograph. For free color brochure, with details of a special offer and complimentary gift certificate, call or write Parle Portraits, 100 Lasalle Street, NYC 10027, (212) 663-7361.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate, with no points, fees or charges of any kind. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

MEDSTAR: THE CUSTOMIZED COMPUTER SYSTEM for medical office management. Designed by a physician and his staff. Completely customized for each practice. Affordable, Reliable, Easy to use. Leasing available. Medstar, 363 Route 111, Smithtown, NY 11787. (516) 360-1422.

ACUPUNCTURE REVIEW FOR FIRST NATIONAL CERTIFICATION and New Jersey Certification Examinations. February 23–March 3, 1985, conducted by Dr. Ralph Alan Dale in New Jersey. Course approved toward Certification by New York Boards for Medicine and Dentistry. Contact Nancy Whelan, R.P.T., 4216 D Palm Bay Circle, West Palm Beach, Florida 33406 or call (305) 683-0815.

ARCHITECTURAL DESIGN AND CONSTRUCTION: Designers, builders of custom professional offices. Consulting services available. Whether you're opening a new office, relocating, remodeling or expanding an existing office in New York City area and adjacent New Jersey counties. Donald Young, Inc. Tel: (718) 948-5191.

HAVING A PROBLEM WITH ALCOHOL AND/OR DRUGS. Special AA Meeting for Doctors April 11th to April 15th, 1985, Govenor Morris Inn, Morristown, N.J. For information write: Secretary, 86 Miller Road, Morristown, N.J. 07906. Anonymity Preserved!

DIAGNOSTIC VIDEO IMAGER & FILM RECORDER. Never used. Built by Matrix Inst. Microprocessor, high resolution monitor, remote control. Mint. Value $15,000. Will take best offer. Mr. Pereira, 461 Jefferson Place, Mineola, NY 11501 or (212) 575-5800 Ext. 314.

Angina comes in many forms...

So does SORBITRATE®

(ISOSORBIDE DINITRATE)

Unsurpassed flexibility in nitrate therapy.

© 1985 ICI AMERICAS INC.

See following page for brief summary of prescribing information.

SORBITRATE®
(ISOSORBIDE DINITRATE)

Please consult full prescribing information before use. A summary follows:

INDICATIONS AND USAGE: SORBITRATE (isosorbide dinitrate) is indicated for the treatment and prevention of angina pectoris. All dosage forms of isosorbide dinitrate may be used prophylactically to decrease frequency and severity of anginal attacks and can be expected to decrease the need for sublingual nitroglycerin.

The sublingual and chewable forms of the drug are indicated for acute prophylaxis of angina pectoris when taken a few minutes before situations likely to provoke anginal attacks. Because of a slower onset of effect, the oral forms of isosorbide dinitrate are not indicated for acute prophylaxis.

CONTRAINDICATIONS: SORBITRATE is contraindicated in patients who have shown purported hypersensitivity or idiosyncrasy to it or other nitrates or nitrites. Epinephrine and related compounds are ineffective in reversing the severe hypotensive events associated with overdose and are contraindicated in this situation.

WARNINGS: The benefits of SORBITRATE during the early days of an acute myocardial infarction have not been established. If one elects to use organic nitrates in early infarction, hemodynamic monitoring and frequent clinical assessment should be used because of the potential deleterious effects of hypotension.

PRECAUTIONS: General: Severe hypotensive response, particularly with upright posture, may occur with even small doses of SORBITRATE. The drug should therefore be used with caution in subjects who may have blood volume depletion from diuretic therapy or in subjects who have low systolic blood pressure (eg, below 90 mmHg). Paradoxical bradycardia and increased angina pectoris may accompany nitrate-induced hypotension. Nitrate therapy may aggravate the angina caused by hypertrophic cardiomyopathy.

Marked symptomatic, orthostatic hypotension has been reported when calcium channel blockers and organic nitrates were used in combination. Dose adjustment of either class of agents may be necessary.

Tolerance to this drug and cross-tolerance to other nitrates and nitrites may occur. Tolerance to the vascular and antianginal effects of isosorbide dinitrate or nitroglycerin has been demonstrated in clinical trials, experience through occupational exposure, and in isolated tissue experiments in the laboratory. The importance of tolerance to the appropriate use of isosorbide dinitrate in the management of patients with angina pectoris has not been determined. However, one clinical trial using treadmill exercise tolerance (as an end point) found an 8-hour duration of action of oral isosorbide dinitrate following the first dose (after a 2-week placebo washout) and only a 2-hour duration of effect of the same dose after 1 week of repetitive dosing at conventional dosing intervals. On the other hand, several trials have been able to differentiate isosorbide dinitrate from placebo after 4 weeks of therapy and, in open trials, an effect seems detectable for as long as several months.

Tolerance clearly occurs in industrial workers continuously exposed to nitroglycerin. Moreover, physical dependence also occurs since chest pain, acute myocardial infarction, and even sudden death have occurred during temporary withdrawal of nitroglycerin from the workers. In clinical trials in angina patients, there are reports of anginal attacks being more easily provoked and of rebound in the hemodynamic effects soon after nitrate withdrawal. The relative importance of these observations to the routine, clinical use of isosorbide dinitrate is not known. However, it seems prudent to gradually withdraw patients from isosorbide dinitrate when the therapy is being terminated, rather than stopping the drug abruptly.

Information for Patients: Headache may occur during initial therapy with SORBITRATE. Headache is usually relieved by the use of standard headache remedies or by lowering the dose and tends to disappear after the first week or two of use.

Drug Interactions: Alcohol may enhance any marked sensitivity to the hypotensive effect of nitrates.

Isosorbide dinitrate acts directly on vascular smooth muscle, therefore, any other agent that depends on vascular smooth muscle as the final common path can be expected to have decreased or increased effect depending on the agent.

Carcinogenesis, Mutagenesis, Impairment of Fertility: No long-term studies in animals have been performed to evaluate the carcinogenic potential of this drug. A modified two-litter reproduction study in rats fed isosorbide dinitrate at 25 or 100 mg/kg/day did not reveal any effects on fertility or gestation or any remarkable gross pathology in any parent or offspring fed isosorbide dinitrate as compared with rats fed a basal-controlled diet.

Pregnancy Category C: Isosorbide dinitrate has been shown to cause a dose-related increase in embryotoxicity (increase in mummified pups) in rabbits at oral doses 35 and 150 times the maximum recommended human daily dose. There are no adequate and well-controlled studies in pregnant women. SORBITRATE should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when SORBITRATE is administered to a nursing woman.

Pediatric Use: The safety and effectiveness of SORBITRATE in children has not been established.

ADVERSE REACTIONS: Adverse reactions, particularly headache and hypotension, are dose-related. In clinical trials at various doses, the following have been observed.

Headache is the most common (reported incidence varies widely, apparently being dose-related, with an average occurrence of about 25%) adverse reaction and may be severe and persistent. Cutaneous vasodilation with flushing may occur. Transient episodes of dizziness and weakness, as well as other signs of cerebral ischemia associated with postural hypotension, may occasionally develop (the incidence of reported symptomatic hypotension ranges from 2% to 36%). An occasional individual will exhibit marked sensitivity to the hypotensive effects of nitrates and severe responses (nausea, vomiting, weakness, restlessness, pallor, perspiration, and collapse) may occur even with the usual therapeutic dose. Drug rash and/or exfoliative dermatitis may occasionally occur. Nausea and vomiting appear to be uncommon. Case reports of clinically significant methemoglobinemia are rare at conventional doses of organic nitrates. The formation of methemoglobin is dose-related and, in the case of genetic abnormalities of hemoglobin that favor methemoglobin formation, even conventional doses of organic nitrate could produce harmful concentrations of methemoglobin.

DOSAGE AND ADMINISTRATION: For the treatment of angina pectoris, the usual starting dose for sublingual SORBITRATE is 2.5 to 5 mg; for chewable tablets, 5 mg, for oral (swallowed) tablets, 5 to 20 mg; and for controlled-release forms, 40 mg.

SORBITRATE should be titrated upward until angina is relieved or side effects limit the dose. In ambulatory patients, the magnitude of the incremental dose increase should be guided by measurements of standing blood pressure.

The initial dosage of sublingual or chewable SORBITRATE for prophylactic therapy in angina pectoris patients is generally 5 or 10 mg every 2 to 3 hours. Adequate controlled clinical studies demonstrating the effectiveness of chronic maintenance therapy with these dosage forms have not been reported.

SORBITRATE in oral doses of 10 to 40 mg given every 6 hours or in oral controlled-release doses of 40 to 80 mg given every 8 to 12 hours is generally recommended. The extent to which development of tolerance should modify the dosage program has not been defined. *The oral controlled-release forms of isosorbide dinitrate should not be chewed.*

DOSAGE FORMS AVAILABLE: Sublingual Tablets (2.5, 5, 10 mg), Chewable Tablets (5, 10 mg), Oral Tablets (5, 10, 20, 30, 40 mg), Sustained Action Tablets (40 mg).

STUART PHARMACEUTICALS
Division of ICI Americas Inc.
Wilmington, DE 19897

STR-2282

MISCELLANEOUS—CONT'D

DOCTOR, YOU CAN'T BEAT the quality or the price! Holter Monitor Scanning Service. Physician owned, trained and supervised. No contracts to sign. We can arrange for lease/purchase of Holter equipment. Why are you paying more and getting less? Now using UP Service for faster turn around time. DCG Interpretation (313) 879-8860.

TAX ATTORNEY AND PENSION PLAN Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professional incorporation—No insurance required—references upon request—Jack Wachstock, PC, 3000 Marcus Ave., Lake Success, N.Y. 11530 (516) 437-4848.

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

ARCHITECTURE & PLANNING, Comprehensive services from analysis of alternative office locations through design and construction. Experienced in both new construction and renovation. Lewis & Gould (212) 807-6588.

1985 CME CRUISE/CONFERENCES on Selected Medical Topics—Caribbean, Mexican, Hawaiian, Alaskan, Mediterranean. 7-14 days year-round. Approved for 20-24 CME Cat. 1 credits (AMA/PRA) & AAFP prescribed credit. Distinguished professors. Fly Roundtrip Free on Caribbean, Mexican & Alaskan Cruises. Excellent group fares on finest ships. Registration Limited. Pre-scheduled in compliance with present IRS requiremnets. Information: International Conferences, 189 Lodge Ave., Huntington Station, NY 11746. (516) 549-0869.

PATIENT NEWSLETTER: Show patients you care by keeping in touch! Send them our quarterly health promotion newsletter. As low as $1 per year. Customized with your name, address and optional office column. Mailing service. Free sample. RX: LIVE WELL, 6809 Santa Maria Lane, Dallas, Texas 75214-2847, or call (214) 821-5218.

***Wanted by Serious Student and Collector:* Japanese Samurai Swords and Related Pieces.**
Please write or call:
Herman Wallinga, M.D.
Division Head Nuclear Medicine
The Genesee Hospital
224 Alexander St.
Rochester, NY 14607
(716) 263-6080

REAL ESTATE FOR SALE OR RENT

PSYCHIATRISTS OFFICE AVAILABLE in heart of Forest Hills. We have a large, unfurnished wood-panelled office in a professional suite. Excellent for individuals, families and groups. We are off Queens Blvd. with convenient transportation and parking. No smoking. Call Edith Eisenberg ACSW (718) 263-0779.

WANTED: MEDICAL OFFICE BUILDINGS. As principals we are seeking "existing buildings"—fully tennanted by physicians, dentists and health care services. "To be built" medical facilities as a joint venture with either an institution or prominent area physician. Contact: Paul Gellert, Gelco Associates, 155 West 68th St., N.Y., N.Y. 10023; (212) 223-1130.

FOR SALE, OFFICE BUILDING in Suffolk County, Long Island, N.Y. Excellent location. Family practitioner retiring after 30 years. Two other professionals in building. Close to Hospitals. Ample parking. Address inquiries to: Dept. 376, c/o NYSJM.

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

CHELSEA VILLAGE AREA

Ideal for Physicians initiating practice, hours can be tailored to your needs. Spacious designer medical offices. Newly renovated medical building—Rent hourly, daily, monthly, annually. Receptionist, Appointment scheduling. Fully furnished consultation and treatment rooms. Heat, electric and air conditioning included. Secretarial and bookkeeping services available. Convenient to all transportation, St. Vincent's, St. Clare's, Cabrini, V. A., N.Y.U./Bellevue. Available to all specialties. X-rays and rehabilitation services on premises.

PREPARE FOR MEDICINE'S COMPETITIVE FUTURE — Potential for establishing PPO, HMO, or Capitation group.

Tel. (212) 691-2871

FOR SALE

"Be Your Own Landlord"

PARK PLACE OFFICE BUILDING

Opposite Woolworth Building
Near City Hall and World Trade Center
New York City

◆

3 stories (12,200 sf.)
Top two floors available
for immediate occupancy.
First floor leased long term—
top credit tenant.

Principals Only
Contact John Bralower, (212) 541-4337
BRICKELL ASSOCIATES
Exclusive Agents
1251 Avenue of the Americas
New York, NY 10020

MEDICAL OFFICE TO SUBLET, 150 East 61 Street, NYC · Midtown Bloomingdale area. Street entrance, centrally air-conditioned building. Furnished consultation, 2 exam, waiting and secretary area, also treatment cubicals. All part of larger specialist office. Sublet for Tuesday, Thursday all day, Monday, Wednesday, Friday mornings. (212) 753-5527 or 879-7778.

MARCO ISLAND, FLORIDA. Two-bedroom condominium, eleventh floor; on Gulf. Beautifully decorated and furnished. For lease. Call (513) 891-1263.

FOR RENT—MEDICAL OFFICE, Brooklyn, NY, furnished and fully equipped; on bus routes and near municipal parking; 920 square feet on corner. $2,500 per month. Call (718) 454-4844, evenings.

NEW HYDE PARK, L.I., vicinity of Long Island Jewish Hospital. Surgical office, modern professional building, near major parkways and public transportation. Ambulatory surgical facility on premises. Part time/full time hours available. (516) 352-7799.

SUBLEASE-FURNISHED MEDICAL OFFICE in a Medical Building in Eastchester, NY. Easily accessible to hospitals and parkways. Reasonable. Call (914) 779-8004.

FOR SALE—HOME AND OFFICE—Poughkeepsie, NY. Modern fully equipped 6 room office, separate entrance. Beautiful 4 bedroom colonial home. Excellent location near hospitals and schools, ample parking. Physician recently retired from general practice. Family physician urgently needed in area. Office also suitable for any specialty. (914) 452-5058.

ONLY DALMANE®
flurazepam HCl/Roche
IMPROVES SLEEP...

WITH
COMPLETE
SLEEP LABORATORY
PROOF[1-5]...

Copyright © 1984 by Roche Products Inc. All rights reserved

IN ALL THESE WAYS

- Rapid sleep onset[1-6]
- Effective for middle-of-the-night and early-morning awakenings[2,5]
- Undiminished efficacy for at least 28 nights[2-4]
- Patients usually awake rested and refreshed[7-9]
- Avoids rebound insomnia after discontinuation of therapy[3,5,10-12]

Caution patients about driving, operating hazardous machinery or drinking alcohol during therapy. Limit dose to 15 mg in elderly or debilitated patients. Contraindicated during pregnancy.

AND THE PREDICTABILITY THAT COMES WITH EXPERIENCE

FOR EFFECTIVE RELIEF OF INSOMNIA

DALMANE® ⓘⓥ

flurazepam HCl/Roche

STANDS APART

15-MG/30-MG
CAPSULES

See next page for references and summary of product information.

References: 1. Kales J *et al. Clin Pharmacol Ther* 12:691-697, Jul-Aug 1971 2. Kales A *et al Clin Pharmacol Ther* 18:356-363, Sep 1975 3. Kales A *et al Clin Pharmacol Ther* 19:576-583, May 1976 4. Kales A *et al: Clin Pharmacol Ther* 32:781-788, Dec 1982 5. Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc* 27:541-546, Dec 1979 6. Kales A, Kales JD: *J Clin Pharmacol* 3:140-150, Apr 1983 7. Monti JM: *Methods Find Exp Clin Pharmacol* 3:303-326, May 1981 8. Greenblatt DJ *et al: Sleep* 5 (Suppl 1):S18-S27, 1982 9. Kales A *et al Pharmacology* 26:121-137, 1983 10. Greenblatt DJ, Allen MD, Shader RI: *Clin Pharmacol Ther* 21:355-361, Mar 1977 11. Zimmerman AM: *Curr Ther Res* 13:18-22, Jan 1971 12. Amrein R *et al: Drugs Exp Clin Res* 9(1):85-99, 1983.

Dalmane® Ⓒ
flurazepam HCl/Roche

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended. Repeated therapy should only be undertaken with appropriate patient evaluation.

Contraindications: Known hypersensitivity to flurazepam HCl, pregnancy. Benzodiazepines may cause fetal damage when administered during pregnancy. Several studies suggest an increased risk of congenital malformations associated with benzodiazepine use during the first trimester. Warn patients of the potential risks to the fetus should the possibility of becoming pregnant exist while receiving flurazepam. Instruct patient to discontinue drug prior to becoming pregnant. Consider the possibility of pregnancy prior to instituting therapy.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided, with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase, and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg recommended initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

ROCHE®
Roche Products Inc
Manati, Puerto Rico 00701

Need A Temporary Physician?
During: vacations, CME's, illness, recruiting.
Want Free Time While You Practice Medicine?
Join CompHealth's Locum Tenens Physician Group.
For further information, call 412-741-3310.

CompHealth A Physician Group
WILSON ROSS, Regional Administrator
114 Centennial Ave., Sewickley, PA 15143

NEEMA EMERGENCY MEDICAL
—a professional association—

Emergency Medicine Positions—available with emergency physician group in PA, NY, NJ, VA, WVA, MD, KY and throughout New England, the Southeast, the Midwest, including all suburban, rural and metropolitan areas. Fee-for-service with minimum guarantee provided. Malpractice paid. Practice credits toward board certification. Physician department directors also desired. Please send resume to: NEEMA Emergency Medical, Suite 400, 399 Market Street, Phila., PA 19106 or phone 215-925-3511 in PA, or 800-523-0776 outside PA.

Medical Society of the State of New York
ANNUAL CME ASSEMBLY
JUNE 7-9, 1985
The New York Hilton, New York City
Combined With The Annual Meeting of
The House of Delegates
JUNE 6-9

NATIONAL EMERGENCY SERVICES, INC. (N.E.S.)

Directorships, full- and part-time emergency department opportunities are available throughout New York State and the country for primary care physicians with ER experience. Enjoy a variety of practice settings, competitive remuneration, paid malpractice insurance, and flexible scheduling. For information and application materials contact the recruitment department at (516) 365-4010, (Outside N.Y. State 1-800-645-4848) or send C.V. to N.E.S., One Hollow Lane, Suite 304, Lake Success, N.Y. 11042.

ORTHOPEDIC SURGEON

Orthopod needed full time for my clinics in Rochester and Buffalo to perform Orthopedic Disability Evaluations.

Kick the hassle of practice, hospitals, etc. Enjoy banker's hours and maintain high income.

Write with CV to H. Lewis, M.D., 3641 Quail Hollow, Bloomfield Hills, Michigan, 48013.

BALANCED CALCIUM CHANNEL BLOCKADE!

CARDIZEM®
(diltiazem HCl)
balances potent coronary vasodilation with a low incidence of side effects

Low incidence of side effects
CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

Reduces angina attack frequency*
42% to 46% decrease reported in multicenter study.[1]

Increases exercise tolerance*
In Bruce exercise test,[2] control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes ($P<.005$).

CARDIZEM®
(diltiazem HCl)

THE BALANCED CALCIUM CHANNEL BLOCKER

Please see full prescribing information on following page.

2/84

---

*Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

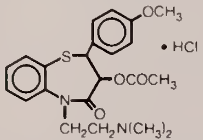
References:
1. Strauss WE, McIntyre KM, Parisi AF, et al: Safety and efficacy of diltiazem hydrochloride for the treatment of stable angina pectoris: Report of a cooperative clinical trial. Am J Cardiol 49:560-566, 1982.
2. Pool PE, Seagren SC, Bonanno JA, et al: The treatment of exercise-inducible chronic stable angina with diltiazem: Effect on treadmill exercise. Chest 78 (July suppl):234-238, 1980.

PROFESSIONAL USE INFORMATION

cardizem®
(diltiazem HCl)
30 mg and 60 mg tablets

DESCRIPTION

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one,3-(acetyloxy) -5-[2-(dimethylamino)ethyl]-2,3-dihydro-2-(4-methoxyphenyl)-, monohydrochloride,(+) -cis-. The chemical structure is:



Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform. It has a molecular weight of 450.98. Each tablet of CARDIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration.

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth muscle.

Mechanisms of Action. Although precise mechanisms of its antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways:

1. Angina Due to Coronary Artery Spasm: CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial and subendocardial. Spontaneous and ergonovine-induced coronary artery spasm are inhibited by CARDIZEM.
2. Exertional Angina: CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal and maximal exercise work loads.

In animal models, diltiazem interferes with the slow inward (depolarizing) current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Diltiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance.

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricular conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load. Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect; cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of diltiazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by diltiazem.

Intravenous diltiazem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Diltiazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, diltiazem significantly prolongs sinus cycle length (up to 50% in some cases).

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Diltiazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40%. CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 60 mg are given; a 120-mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem.

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm.** CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina).** CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS).

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia.
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing.
Cardiac Failure	Administer inotropic agents (isoproterenol, dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

The oral/LD_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort acute anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy**—CARDIZEM may be safely coadministered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

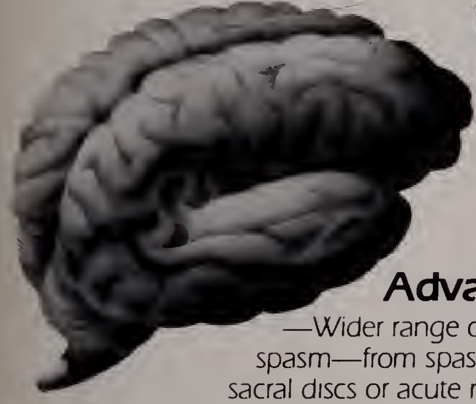
Cardizem 30-mg tablets are supplied in bottles of 100 (NDC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1771-49). Each green tablet is engraved with MARION on one side and 1771 engraved on the other. CARDIZEM 60-mg scored tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1772-49). Each yellow tablet is engraved with MARION on one side and 1772 on the other.

Issued 4/1/84

Another patient benefit product from
PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC
KANSAS CITY, MISSOURI 64137

THE SPASM/PAIN/SPASM CYCLE

In skeletal muscle spasm due to local pathology, responsive to the distinct actions of



Adjunctive
VALIUM® Ⓒ
diazepam/Roche
2-mg, 5-mg, 10-mg scored tablets

Advantages cyclobenzaprine cannot claim

—Wider range of indications as adjunctive therapy for skeletal muscle spasm—from spasm due to local pathology (*e.g.*, herniated lumbosacral discs or acute muscle strain) to spasm associated with upper motor neuron disorders (*e.g.*, cerebral palsy and paraplegia).

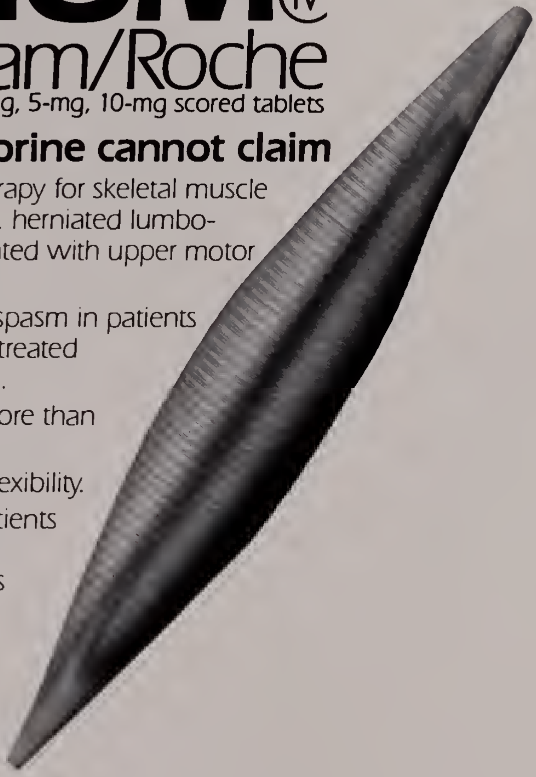
—May be used adjunctively for relieving skeletal muscle spasm in patients with hyperthyroidism, cardiac patients and patients being treated with anticholinergics or guanethidine-type antihypertensives.

—Can be administered to patients less than 15 years and more than 6 months of age.

—Scored tablets in three strengths provide greater dosage flexibility.

Since drowsiness, fatigue and ataxia sometimes occur, patients should be cautioned against driving or operating hazardous machinery and should also be advised against simultaneous ingestion of alcohol.

References: 1. Rankin EA: *Contin Educ 3*(1):46-50, Jan 1975. **2.** When muscle spasm hobbles your patient. *Patient Care 8*(11):20-37, Jun 1, 1974.

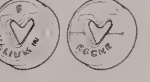


Valium* (diazepam/Roche) Ⓒ
Before prescribing, please consult complete product information, a summary of which follows:
Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic Symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal. adjunctively in skeletal muscle spasm due to reflex spasm to local pathology spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not as sole therapy)
The effectiveness of Valium in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies The physician should periodically reassess the usefulness of the drug for the individual patient
Contraindicated: Known hypersensitivity to the drug Children under 6 months of age Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy
Warnings: Not of value in psychotic patients Caution against hazardous occupations requiring complete mental alertness When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication, abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures Advise against simultaneous ingestion of alcohol and other CNS depressants Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months After extended therapy, gradually taper dosage Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence

***Usage in Pregnancy*: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

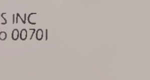
Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies Observe usual precautions in impaired renal or hepatic function Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation
The clearance of Valium and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration The clinical significance of this is unclear
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported, should these occur, discontinue drug Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy
Dosage: Individualize for maximum beneficial effect *Adults* Anxiety disorders, symptoms of anxiety, 2 to 10 mg b i d to q i d ., alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t i d or q i d as needed, adjunctively in skeletal muscle spasm, 2 to 10 mg t i.d or q i.d , adjunctively in convulsive disorders, 2 to 10 mg b i.d to q.i.d *Geriatric or debilitated patients*. 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated (See Precautions.) *Children* 1 to 2½ mg t.i.d or q i.d initially, increasing as needed and tolerated (not for use under 6 months)
How Supplied: For oral administration, round, scored tablets with a cut out "V" design—2 mg, white, 5 mg, yellow, 10 mg, blue— bottles of 100 and 500; Prescription Paks of 50, available in trays of 10. Tel-E-Dose* packages of 100, available in boxes of 4 reverse-numbered cards of 25, and in boxes containing 10 strips of 10

Imprint on tablets
2 mg—2 VALIUM* (front)
ROCHE (scored side)

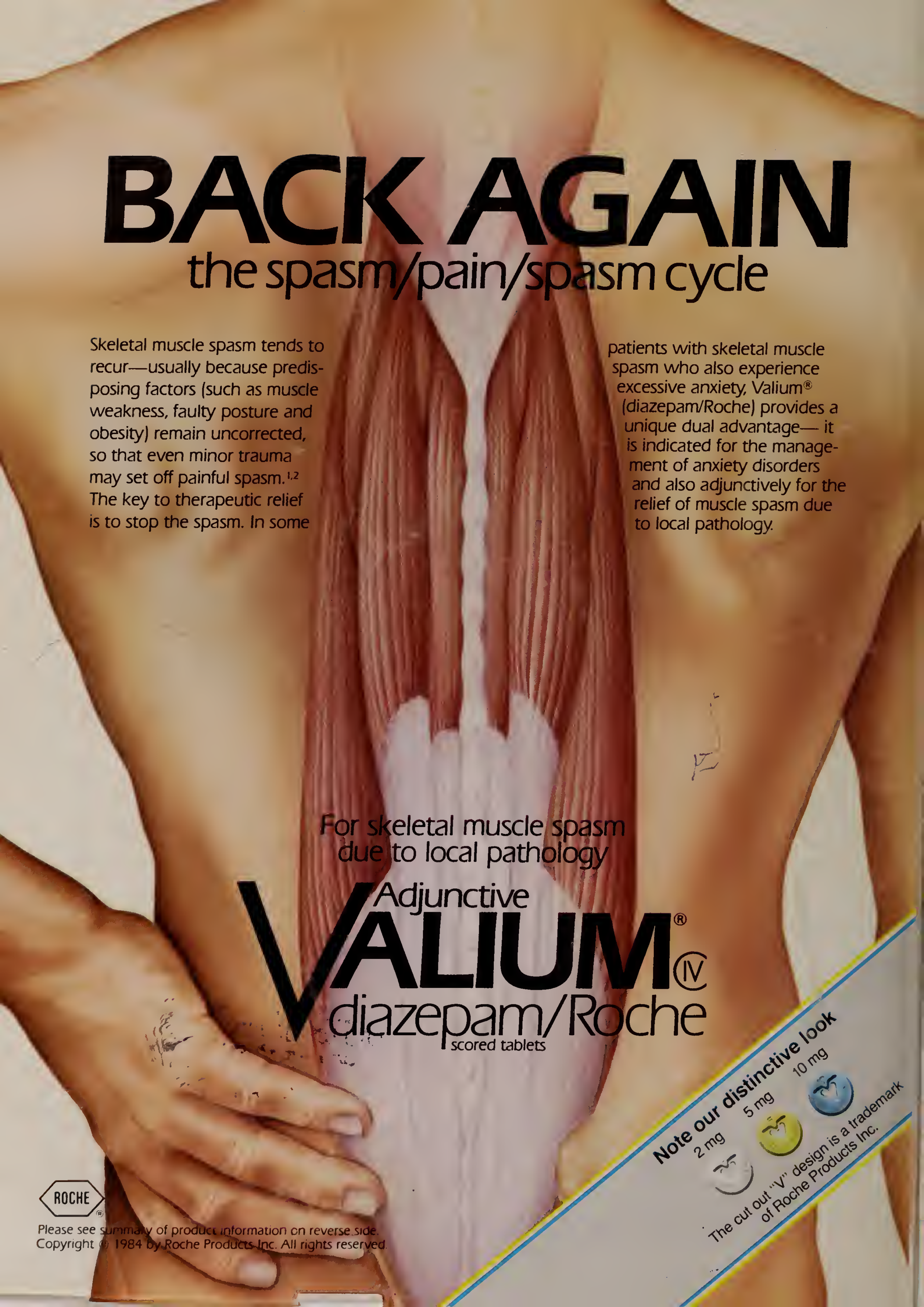


5 mg—5 VALIUM* (front)
ROCHE (scored side)

10 mg—10 VALIUM* (front)
ROCHE (scored side)



ROCHE®
ROCHE PRODUCTS INC
Manati, Puerto Rico 00701



BACK AGAIN

the spasm/pain/spasm cycle

Skeletal muscle spasm tends to recur—usually because predisposing factors (such as muscle weakness, faulty posture and obesity) remain uncorrected, so that even minor trauma may set off painful spasm.[1,2] The key to therapeutic relief is to stop the spasm. In some patients with skeletal muscle spasm who also experience excessive anxiety, Valium® (diazepam/Roche) provides a unique dual advantage— it is indicated for the management of anxiety disorders and also adjunctively for the relief of muscle spasm due to local pathology.

For skeletal muscle spasm due to local pathology

Adjunctive

VALIUM® ⓘⓥ

diazepam/Roche

scored tablets

The cut out "V" design is a trademark of Roche Products Inc.

ROCHE®

Please see summary of product information on reverse side.

Copyright © 1984 by Roche Products Inc. All rights reserved.

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

FEBRUARY 1985 • Volume 85, Number 2

Contents

COMMENTARIES

Searching for alternatives to war 55
BERY ENGEBRETSEN, MD

Preventing alcohol-related violence: The physician's role 56
SHEILA B. BLUME, MD

Psychiatry and the prevention of violence 57
THOMAS J. CRAIG, MD

SYMPOSIUM ON VIOLENCE

The wounds of war 61
WILLIAM D. SHARPE, MD

Violence in psychiatric institutions 64
STEVEN E. KATZ, MD, ROBERT COHEN, PhD, CORNELIUS L. J. STOKMAN, PhD

The obligation to assess dangerousness 67
DANIEL W. SCHWARTZ, MD

Violence on the highway 69
JOHN D. STATES, MD

How the police view violence 72
PATRICK V. MURPHY

Violence in the Third World 74
SISTER HELENE O'SULLIVAN

HISTORY

The physician and war 77
ABRAHAM BLINDERMAN, PhD

LETTERS TO THE EDITOR

Nd:YAG laser photocoagulation 59
RICHARD S. McCRAY, MD, WILLIAM P. WINKLER, MD

A program for rubella surveillance and control 59
ERNEST B. VISCONTI, MD, SUZANNE HOWE, MD, KATHRYN I. MURRAY

Extra-adrenal myelolipoma 60
V. JAGADHA, MD, G. RAMASWAMY, MD

BOOK REVIEWS 80

BOOKS RECEIVED 83
OBITUARIES 84
MEDICAL SOCIETY AFFAIRS 88
GUIDELINES FOR AUTHORS
MEDICAL MEETINGS AND LECTURES

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
FEB 21 1985

It may be that the advocates of "nuclear preparedness" are among the most dangerous romantics ever to achieve political power in human history. Be they Soviets or Americans or French, they put to risk all the primary human values—life, society, health, family, friendship, work, freedom, and justice—in the alleged service of all sorts of secondary values such as wealth, prestige, power, precedent, law, nationality. Having seen so much suffering during your professional life, would you not prefer to be healthy rather than wealthy? Having known the fickleness of prestige, would you not prefer to undergird your life with a few solid friendships? So goes the process of ranking personal values. Such rankings are needed for collective values as well. The connection between one's personal and collective lives is now intimate. It is a contradiction to prefer the life of one's nation at the expense of the lives of most of its citizens. National self-interest is the life of citizens, apart from whom the nation has no existence. People willing to destroy the nation in order to preserve it are confused about means and ends.

M.D.S.

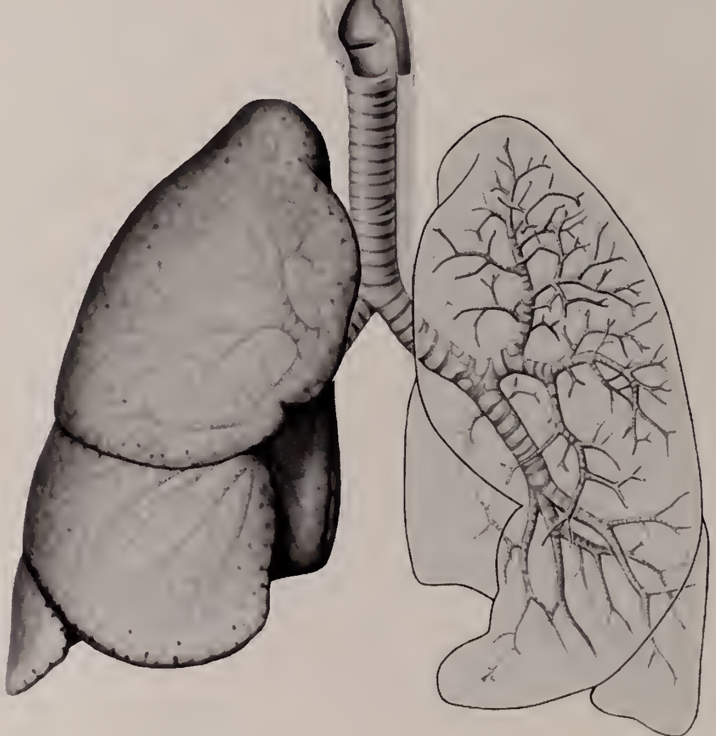
DONALD W. SHRIVER, JR.
President, Union Theological Seminary
"The realism of peace"

Address at the Symposium on Violence, Annual CME Assembly, Medical Society of the State of New York, New York City, June 30, 1984.

Six papers based on presentations at the Symposium, which was coordinated by Stephen Nordlicht, MD, appears in this issue of the Journal.

Library of the College Physician
of Philadelphia Dec. 85
19 S. 22nd St.
Phila, PA 1910

Consider the causative organisms...



Ceclor®
cefaclor
250-mg Pulvules® t.i.d.

offers effectiveness against the major causes of bacterial bronchitis

H. influenzae, *H. influenzae*, *S. pneumoniae*, *S. pyogenes*
(ampicillin-susceptible) (ampicillin-resistant)

Brief Summary Consult the package literature for prescribing information.

Indications and Usage Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae)*, *Haemophilus influenzae*, and *S pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor

Contraindication Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

Warnings IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES

Antibiotics including Ceclor should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins), therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics Such colitis may range in severity from mild to life-threatening

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C difficile* Other causes of colitis should be ruled out

Precautions: *General Precautions* – If an allergic reaction to Ceclor® (cefaclor, Lilly) occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e g , pressor amines, antihistamines, or corticosteroids

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms Careful observation of the patient is essential If superinfection occurs during therapy, appropriate measures should be taken

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

Ceclor should be administered with caution in the presence of markedly impaired renal function Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly)

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis

Usage in Pregnancy – Pregnancy Category B – Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor® (cefaclor, Lilly). There are, however, no adequate and well-controlled studies in pregnant women Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers – Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses Average levels were 0.18, 0 20, 0.21, and 0 16 mcg/ml at two, three, four, and five hours respectively Trace amounts were detected at one hour The effect on nursing infants is not known Caution should be exercised when Ceclor is administered to a nursing woman

* *Usage in Children* – Safety and effectiveness of this product for use in infants less than one month of age have not been established

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70)

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment Nausea and vomiting have been reported rarely

Hypersensitivity reactions have been reported in about 1 5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy No serious sequelae have been reported Antihistamines and corticosteroids appear to enhance resolution of the syndrome

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain – Transitory abnormalities in clinical laboratory test results have been reported Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician

Hepatic – Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40)

Hematopoietic – Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40)

Renal – Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200)

[061782R]

Note Ceclor® (cefaclor, Lilly) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

© 1984, ELI LILLY AND COMPANY

Lilly

400441

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285

Eli Lilly Industries, Inc
Carolina, Puerto Rico 00630

Kodak presents...

New KODAK EKTACHEM DT60 Analyzer

A timely invest

The KODAK EKTACHEM DT60 Analyzer creates an extra service for your patients without extra investment in labor. And because it can pay for itself in three months, it's a timely investment in your future.

The chemistry tests you need

With the DT60 Analyzer you perform key chemistry tests in your own office instead of using an outside laboratory. Available tests include glucose, cholesterol, triglycerides, BUN, uric acid, sodium, and potassium, with total hemoglobin and bilirubin coming soon.

The time you need

Get test results in five minutes or less; perform up to 75 tests an hour. Save time waiting for results to assist in your diagnosis, and on follow-up phone calls.

The accuracy you need

The DT60 Analyzer uses proven technology and methodology from the KODAK EKTACHEM 400 and 700 Analyzers, which

© Eastman Kodak Company, 1984

ment for your office.

provide millions of accurate, precise results to clinical laboratories nationwide.

The simplicity you need
The DT60 Analyzer, compact as a personal computer, features dry slide technology to eliminate wet reagents. It is automated to free up your staff, and training takes only minutes. From the finger-stick sample to results printout, the DT60 Analyzer is simplicity itself.

To see what the DT60 Analyzer can do for you, write Eastman Kodak Company, Dept. 740-B, 343 State Street, Rochester, NY 14650, or call **1 800 44KODAK,** Ext 423 (1 800 445-6325, Ext 423) today.

Leading the way in healthcare technology for over 100 years.

KODAK EKTACHEM Clinical Chemistry Products

MAY NOT BE AVAILABLE IN ALL AREAS

NEW YORK STATE
JOURNAL OF MEDICINE

LIBRARY, ARCHIVES, AND PUBLICATIONS COMMITTEE

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD	GERALD P. MURPHY, MD
ELIZA H. CALDWELL, MD	RICHARD B. NOLAN, MD
URSULA MATULONIS (Medical Student)	CARL E. POCHEDLY, MD

JOHN T. PRIOR, MD

Editor	ALAN BLUM, MD
Deputy Editor and Book Review Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Managing Editor	VICKI GLASER
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Administrative Assistant	IMELDA MORRISSEY
Editorial Assistant	JEANNE CONNERY
Librarian	ELLA ABNEY
Library Assistant	ELEANOR BURNS
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1985

RANDALL BLOOMFIELD, MD	ROBERT LANDESMAN, MD
ROBERT D. BRANDSTETTER, MD	STEPHEN NORDLICHT, MD
JOHN S. DAVIS, MD	IRA A. POLISAR, MD
MARK C. HOMONOFF, MD	JOSEPH SCHLUGER, MD
ALFRED P. INGEGNO, MD	BJORN THORBJARNARSON, MD
FLORENCE KAVALER, MD	RODRIGO E. URIZAR, MD

NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. Synergistic Media Sales, 50 East 42nd St., Suite 504, New York, N.Y. 10017. Telephone: (212) 682-2200.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

ALLISON B. LANDOLT, MD, Westchester	*President*
BERNARD J. PISANI, MD, New York	*Past-President*
DANIEL F. O'KEEFFE, MD, Warren	*President-Elect*
JOHN A. FINKBEINER, MD, New York	*Vice-President*
JOHN H. CARTER, MD, Albany	*Secretary*
GEORGE LIM, MD, Oneida	*Assistant Secretary*
CHARLES D. SHERMAN, JR, MD, Monroe	*Treasurer*
MORTON KURTZ, MD, Queens	*Assistant Treasurer*
JOHN L. CLOWE, MD, Schenectady	*Speaker*
CHARLES N. ASWAD, MD, Broome	*Vice-Speaker*

Councilors

Term Expires 1985	JAMES H. COSGRIFF, JR, MD, Erie SAMUEL M. GELFAND, MD, Nassau RICHARD A. HUGHES, MD, Warren ANTONIO F. LASORTE, MD, Broome
Term Expires 1986	ROBERT A. MAYERS, MD, Westchester THOMAS D. PEMRICK, MD, Rensselaer RALPH E. SCHLOSSMAN, MD, Queens STANLEY A. STECKLER, MD, Suffolk
Term Expires 1987	DAVID M. BENFORD, MD, New York DUANE M. CADY, MD, Onondaga JAMES M. FLANAGAN, MD, Wayne M. THEODORE TANENHAUS, MD, Brooklyn

Trustees

CARL GOLDMARK, JR, MD, New York, *Chairman*
G. REHMI DENTON, MD, Albany
RALPH M. SCHWARTZ, MD, New York
EDGAR P. BERRY, MD, New York
RICHARD D. EBERLE, MD, Onondaga
JOSEPH F. SHANAPHY, MD, Richmond
VICTOR J. TOFANY, MD, Monroe

Executive Vice-President	MILTON ROSENBERG, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Associate	IRMA A. ERICKSON
Director, Division of Scientific Publications	ALAN BLUM, MD
Director, Division of Management Services	GEORGE FORREST, JR
Assistant Director, Division of Governmental Affairs	JERRY S. HOFFMAN, ESQ
Director, Division of Communications	EDWARD HYNES
Director, Division of Insurance and Membership Benefits	BERNARD M. JACKSON
Director, Division of Membership	EUNICE SKELLY
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Scientific Activities	GEORGE J. LAWRENCE, JR, MD
Director, Division of Advertising-Sales and Meeting Services	ROBERT MILLER
General Counsel	DONALD R. MOY, JD
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Medical Services	ROBERT J. O'CONNOR, MD
Director, Division of Long-Range Planning	DEBORAH R. SIEGEL
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. The proceedings of the Society's House of Delegates meeting are published in March as a separate issue of the *Journal* with limited circulation. Editorial material published in the *Journal* is covered by copyright. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

Classified Advertising

PHYSICIANS WANTED

INTERNIST WANTED, NASSAU COUNTY, NY Board certified/eligible, with/without subspecialty to join growing private practice. Reply Dept. 358, c/o NYSJM.

FAMILY PHYSICIAN—Northeastern New York Board Family Physician wanted to join active, private, group practice of family medicine, all board certified family physicians, including obstetrics and hospital care in an excellent local community hospital. Suburban Albany area. Contact: Business Manager 1-(518) 785-4471.

PHYSICIAN FOR CHILDREN'S SUMMER CAMP. Located in lower NYS Catskills, CAMP ECHO seeks Physicians for July and August 1985. Physicains for two or four week periods, with or without camp age children. Excellent medical facility, 2 R.N.'s + aide, private rooms and facilities for M.D. Camp accommodates 350 boys and girls from 6-16 yrs. of age. Write CAMP ECHO, P.O. Box 255, Roslyn, NY 11576 or call (516) 742-2679.

GENERAL/FAMILY PRACTITIONERS, if you are looking for an opportunity to be in the forefront of medical care, practice preventive medicine, work with other innovative professionals, and earn a comfortable living in pleasant surroundings, send your curriculum vitae to Physician Placement Dept.—38. An equal opportunity employer. CIGNA Healthplans of California, 700 N. Brand Blvd., Suite 500, Glendale, CA 91203.

OBSTETRICIAN/GYNECOLOGIST: Second OB/GYN needed to join this rural group practice of 10 physicians. Primary care is emphasized. Formal tertiary linkages to Rochester General Hospital for high risk. Base salary, then income tied to productivity. Contact: Richard F. Endres, M.D., Sodus Health Center, P.O. Box A, Sodus, NY 14551. EOE.

FAMILY PRACTICE GROUP seeks BC/BE Family Practitioner. Early Partnership, five weeks vacation. Excellent hospitals. Beautiful college town in the Hudson Valley vacation area 90 miles north of N.Y.C. Address CV to Administrator, Medical Associates of New Paltz, PC, 40 Sunset Ridge, New Paltz, NY 12561.

PHYSICIAN DURING JULY AND AUGUST, for children's camp located at Beach Lake, PA, accommodates 350 campers, age 6–16; complete modern Health Center; 2 R.N.'s in attendance; will accept one M.D. for each month; no children accepted who are of camp age. Camp opens June 30 and closes August 24. Private room and facilities. Write to: Trail's End Camp, c/o Beach Lake, Inc., 215 Adams Street, Brooklyn, NY 11201 and include your phone number.

PHYSICIANS WANTED—CONT'D

PHYSICIANS needed for 75 residential children's camps located in Northeast. July and/or August. Contact: Association of Independent Camps (M), 60 Madison Ave., New York, NY 10010. (212) 679-3230.

FAMILY PRACTICE PHYSICIAN: Excellent full-time opportunity for a Board Certified Family Practitioner for a single specialty group. Offering guaranteed income plus partnership opportunity. Facilities include attractive expandable office, growing practice, admitting privileges at large teaching Hospital. Located in scenic Northwestern Pennsylvania. Call Tina Carrigan (215) 896-5080.

TWO POSITIONS ARE AVAILABLE for a Board Certified/Eligible radiologist in a Central New York Community Hospital. One year locum tenens and permanent position with attractive salary leading to partnership. General diagnostic and interventional radiology in three man group. Write Gerald Segal, M.D., 2206 Genesse Street, Utica, New York 13501.

MEDICAL DIRECTOR—Attractive opportunity for Board Certified Physician. 250 bed church affiliated Hospital located in Eastern Pennsylvania serving population of 500,000. Income negotiable; excellent benefits. Requires proven leadership skills and administrative experience. Call Tina Carrigan (215) 896-5080.

STAFF POSITION EMERGENCY DEPARTMENT: 400 bed university affiliated teaching Hospital located in a major metropolitan area in New York State. Must be Board Eligible or Board Certified through Emergency Medicine Residency. Guaranteed salary, excellent relocation and benefits package. Call Carol Burke 1-800-441-0996 or in PA (215) 896-5080.

BOARD CERTIFIED INTERNIST: Preventive & Occupational Health—a leader in field offers varied opportunities (full-time, half-time or per diem) at our clinics and corporate medical units. Send C.V. to Executive Health Examiner, 777 Third Ave., N.Y., N.Y. 10017, Attn: Personnel Director N. [EOE M/F].

POSITIONS WANTED

AP/CP CALIFORNIA PATHOLOGIST, 36, seeks to relocate near NYC, small or medium-sized hospital. Strong community-type background, eight years experience. Well grounded in medical staff and clinical laboratory functions. C.V. on request. J. Vasser, M.D., 7816 Summerplace Drive, Citrus Heights, Calif. 95621.

PEDIATRICIAN: University trained, Board eligible, 3 years private practice experience (NJ). Wishes to relocate for better opportunities. Hospital, Health Organization or partnership potential with private practitioner. Available immediately. Reply Dept. 377 c/o NYSJM.

New variable rate
U.S. Savings Bonds
guarantee
investment growth...

The employees at BURLINGTON INDUSTRIES keep their future plans on target when they rely on the Payroll Savings Plan.

Nancy B. Turner — "Currently, these bonds are accumulating for a college fund for our son. And too, we feel it's necessary for each of us to re-invest in America."

John Maier — "I would tell other employees that it is an easy way to use small amounts of money through payroll deduction to build toward a lump sum gift for a child or loved one where that person benefits from the earnings."

Lois Bailiff — "In a small way we are helping the future of our country as well as building up our savings for retirement. Bonds have given our family extra money to buy items for our home and for graduation and wedding gifts."

Take stock in America.

Director of Sales
U.S. Savings Bonds Division
Department of the Treasury
Washington, D.C. 20226

Yes, please send me *Free* information about the Payroll Savings Plan.

Name ____________________

Position ____________________

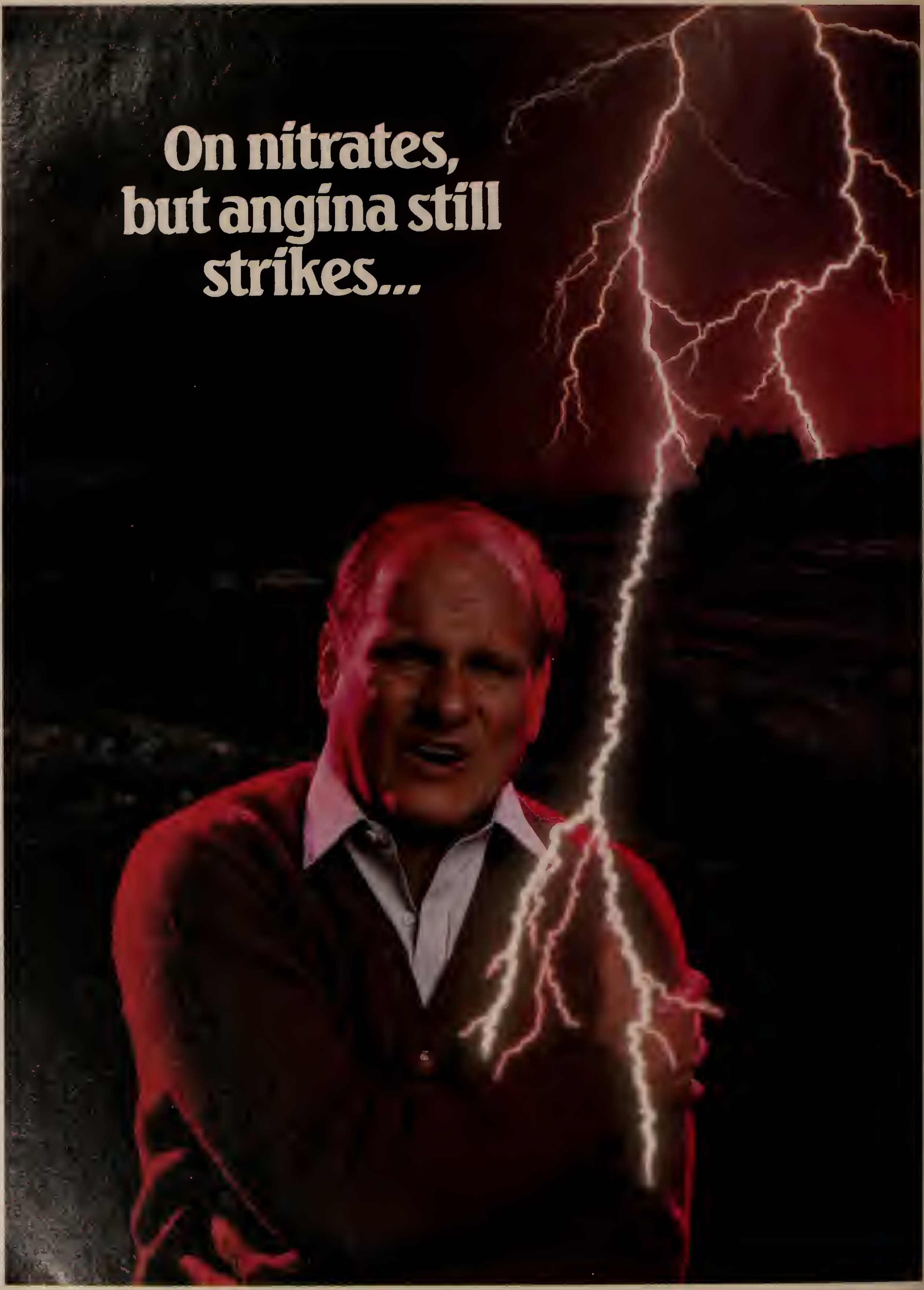
Company ____________________

Address ____________________

City ____________ State ______ Zip ______

Ad Council

A public service of this publication and the Advertising Council


On nitrates, but angina still strikes...

After a nitrate, add ISOPTIN®

(verapamil HCl/Knoll)

To protect your patients, as well as their quality of life, add Isoptin instead of a beta blocker.

First, Isoptin not only reduces myocardial oxygen demand by reducing peripheral resistance, but also increases coronary perfusion by preventing coronary vasospasm and dilating coronary arteries — both normal and stenotic. These are antianginal actions that no beta blocker can provide.

Second, Isoptin spares patients the beta-blocker side effects that may compromise the quality of life.

With Isoptin, fatigue, bradycardia and mental depression are rare. Unlike beta blockers, Isoptin can safely be given to patients with asthma, COPD, diabetes or peripheral vascular disease. Serious adverse reactions with Isoptin are rare at recommended doses; the single most common side effect is constipation (6.3%).

Cardiovascular contraindications to the use of Isoptin are similar to those of beta blockers: severe left ventricular dysfunction, hypotension (systolic pressure <90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no artificial pacemaker is present) and second- or third-degree AV block.

So, the next time a nitrate is not enough, add Isoptin... for more comprehensive antianginal protection without side effects which may cramp an active life style.

ISOPTIN. Added antianginal protection without beta-blocker side effects.

Please see brief summary on following page.

ISOPTIN® **TABLETS**
(verapamil HCl/Knoll)
80 mg and 120 mg

Contraindications: Severe left ventricular dysfunction (see *Warnings*), hypotension (systolic pressure <90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block. **Warnings:** ISOPTIN should be avoided in patients with severe left ventricular dysfunction (e.g., ejection fraction <30%) or moderate to severe symptoms of cardiac failure. Control milder heart failure with optimum digitalization and/or diuretics before ISOPTIN is used. ISOPTIN may occasionally produce hypotension (usually asymptomatic, orthostatic, mild, and controlled by decrease in ISOPTIN dose). Occasional elevations of liver enzymes have been reported; patients receiving ISOPTIN should have liver enzymes monitored periodically. Patients with atrial flutter/fibrillation and an accessory AV pathway (e.g., W-P-W or L-G-L syndromes) may develop a very rapid ventricular response after receiving ISOPTIN (or digitalis). Treatment is usually D.C.-cardioversion. AV block may occur (3rd degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema, and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with ISOPTIN. **Precautions:** ISOPTIN should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Studies in a small number of patients suggest that concomitant use of ISOPTIN and beta blockers may be beneficial in patients with chronic stable angina. Combined therapy can also have adverse effects on cardiac function. Therefore, until further studies are completed, ISOPTIN should be used alone, if possible. If combined therapy is used, patients should be monitored closely. Combined therapy with ISOPTIN and propranolol should usually be avoided in patients with AV conduction abnormalities and/or depressed left ventricular function or in patients who have also recently received methyldopa. Chronic ISOPTIN treatment increases serum digoxin levels by 50% to 70% during the first week of therapy, which can result in digitalis toxicity. The digoxin dose should be reduced when ISOPTIN is given, and the patient carefully monitored. ISOPTIN may have an additive hypotensive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after ISOPTIN administration. Until further data are obtained, combined ISOPTIN and quinidine therapy in patients with hypertrophic cardiomyopathy should probably be avoided, since significant hypotension may result. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. *Pregnancy Category C:* There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. It is not known whether verapamil is excreted in breast milk; therefore, nursing should be discontinued during ISOPTIN use. **Adverse Reactions:** Hypotension (2.9%), peripheral edema (1.7%), AV block: 3rd degree (0.8%), bradycardia: HR<50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%). The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain: confusion, paresthesia, insomnia, somnolence, equilibrium disorders, blurred vision, syncope, muscle cramps, shakiness, claudication, hair loss, maculae, and spotty menstruation. Overall continuation rate of 94.5% in 1,166 patients. **How Supplied:** ISOPTIN (verapamil HCl) is supplied in 80 mg and 120 mg sugar-coated tablets. July 1982 2068

KNOLL PHARMACEUTICAL COMPANY
knoll® 30 NORTH JEFFERSON ROAD, WHIPPANY, NEW JERSEY 07981

2195

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meetings and lectures unless the information is submitted at least two months prior to the event.*

MARCH 1985

AROUND THE STATE

MANHATTAN

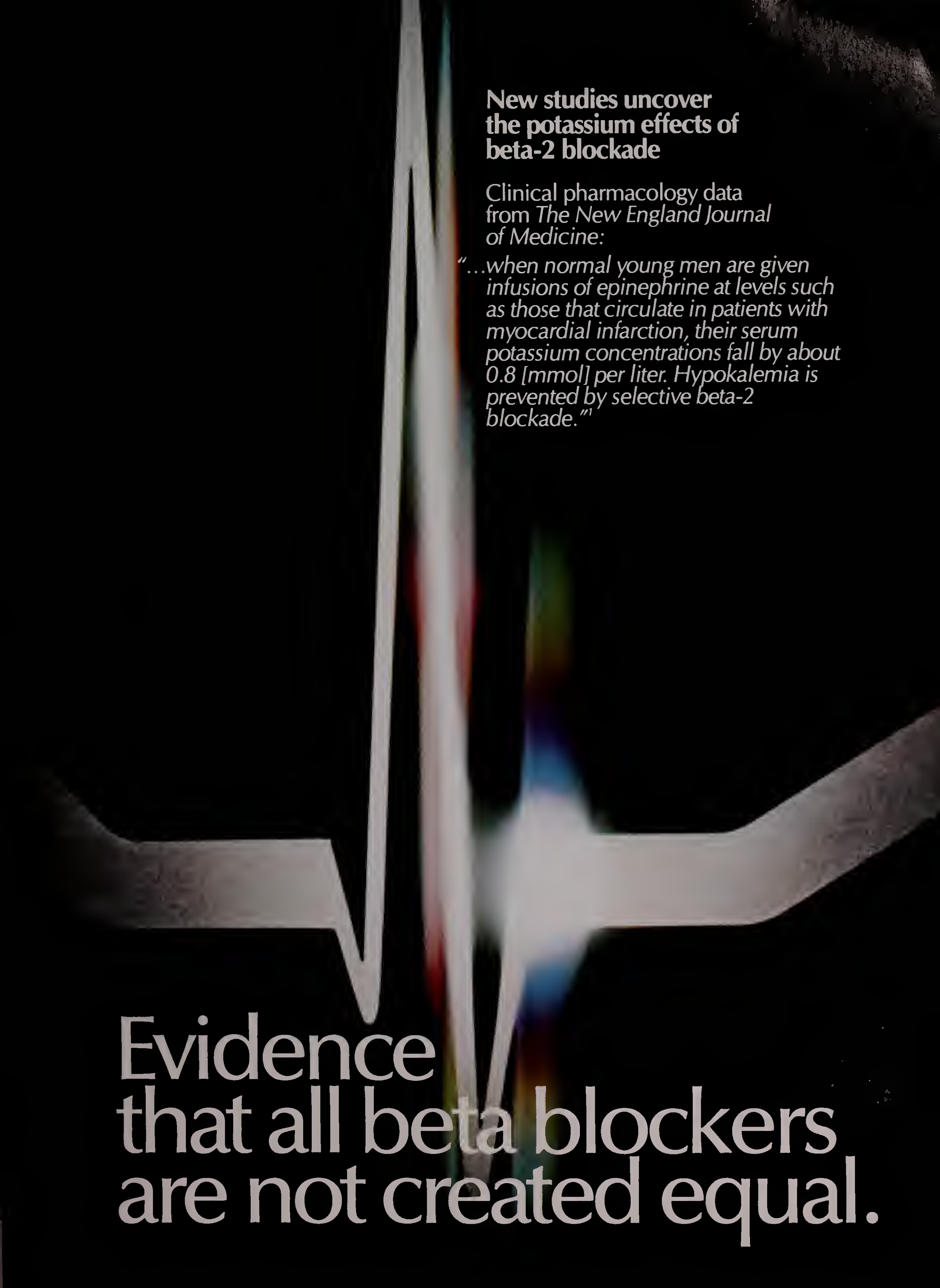
Mar 5, 12, 19, 26. **Hypnosis for Physicians and Dentists.** *Mar 8–9.* **An Update in Internal Medicine for Practicing Psychiatrists and Neurologists.** *Mar 25, 26, 27.* **American College of Cardiology Course: Recent Advances in Interventional Cardiology.** *Mar 29.* **Communication Disorders.** Mount Sinai Medical Center. Info: Page & William Black Post-Graduate School of Medicine, One Gustave L. Levy Place, New York, NY 10029. Tel: (212) 650-6737.

Mar 7–9. **The Second World Congress on Cancers of the Skin.** Grand Hyatt Hotel. Info: The Skin Cancer Foundation, 475 Park Ave South, New York, NY 10016. Tel: (212) 725-5176.

Mar 11–15. **Electromyography and Electrodiagnosis.** Hotel Mayflower. Info: Daoud B. Karam, MD, Rehabilitation Medicine, New York Medical College at Coler Memorial Hospital, Roosevelt Island, NY 10044. Tel: (212) 688-9400, ext 385.

Mar 13–14. **Medical and Surgical Problems in Workers' Compensation.** Roosevelt Hotel. 14 Cat 1 credits. Info: American Academy of Compensation Medicine, PO Box 10, Roosevelt Island, NY 10044. Tel: (212) 750-6777.

Mar 18–22. **Seminar in Advanced Rheumatology.** 29 Cat 1 credits. *Mar 25–29.* **Anesthesiology Comprehensive Review IV.** 40 Cat 1 credits. *Mar 30–31.* **Lasers and Cavitrons in Urology.** 15 Cat 1 credits. Info: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.



New studies uncover the potassium effects of beta-2 blockade

Clinical pharmacology data from *The New England Journal of Medicine:*

"...when normal young men are given infusions of epinephrine at levels such as those that circulate in patients with myocardial infarction, their serum potassium concentrations fall by about 0.8 [mmol] per liter. Hypokalemia is prevented by selective beta-2 blockade."[1]

Evidence that all beta blockers are not created equal.

Right from the start in hypertension...

Once-daily INDERAL LA (propranolol HCl) for smooth blood pressure control without the potassium problems of diuretics

Once-daily INDERAL LA (propranolol HCl) avoids the risk of diuretic-induced ECG abnormalities due to hypokalemia.[2,3] In addition, INDERAL LA preserves potassium balance without additive agents or supplements while providing simple, well-tolerated therapy with broad cardiovascular benefits.

Once-daily INDERAL LA for the cardiovascular benefits of the world's leading beta blocker

Simply start with 80 mg once daily. Dosage may be increased to 120 mg to 160 mg once daily as needed to achieve additional control.

Like conventional INDERAL tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.

For beta-1/beta-2 blockade

Once-daily

INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES

80 mg 120 mg 160 mg

The appearance of these capsules is a registered trademark of Ayerst Laboratories.

Please see brief summary of prescribing information on the next page for further details.

Once-daily
For beta-1/beta-2 blockade INDERAL® LA
(PROPRANOLOL HCl) LONG ACTING CAPSULES

The appearance of these capsules
is a registered trademark
of Ayerst Laboratories

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION SEE PACKAGE CIRCULAR.)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use Effects on plasma volume appear to be minor and somewhat variable INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential The significance of the membrane action in the treatment of arrhythmias is uncertain

The mechanism of the antimigraine effect of propranolol has not been established Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

Propranolol is not significantly dialyzable

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely or INDERAL should be discontinued (gradually, if possible)

IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)— PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g, dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA. Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General* Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure

Clinical Laboratory Tests Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy Pregnancy Category C INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman

Pediatric Use Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular bradycardia, congestive heart failure, intensification of AV block, hypotension, paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type

Central Nervous System lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances, hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics

Gastrointestinal nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory: bronchospasm

Hematologic agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION—*Dosage must be individualized* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic The dosage may be increased to 120 mg once daily or higher until adequate blood pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

REFERENCES

1. Epstein FH, Rosa RM Adrenergic control of serum potassium. *N Engl J Med* 1983. 309 1450-1451 **2.** Holland OB, Nixon JV, Kuhnert L Diuretic-induced ventricular ectopic activity *Am J Med* 1981,70 762-768. **3.** Holme I, Helgeland A, Hjermann I, et al Treatment of mild hypertension with diuretics The importance of ECG abnormalities in the Oslo study and in MRFIT. *JAMA* 1984,251 1298-1299

9411/1184

Ayerst
AYERST LABORATORIES
New York, N Y 10017

Copyright © 1984 AYERST LABORATORIES
Division of AMERICAN HOME PRODUCTS CORPORATION

Medical Society of the State of New York

Annual Meeting of the HOUSE OF DELEGATES

June 6-9, 1985

ANNUAL CME ASSEMBLY

HEALTH CARE TODAY

June 7-9, 1985

The New York Hilton
New York City

HIGHLIGHTS. . . . .
30 Sessions • Panel Discussions
- Meet the Professors
- Technical Exhibits
- President's Dinner Dance

CLIP & MAIL

Director of Front Office Operations
The New York Hilton
1335 Avenue of the Americas
New York, N.Y. 10019

Phone: 212-586-7000

for hotel use only

NAME ____________________ TITLE ____________
FIRM ____________________ PHONE ____________
STREET ______________________________
CITY/STATE/ZIP ______________________________
REMARKS ______________________________
(please print)

NOTE: Reservations must be received no later than May 16, 1985 and will be held only until 6 p.m. on day of arrival unless later hour is specified.

please print

ARRIVAL DATE ____________________
☐ 2-4 p.m. ☐ 4-6 p.m. ☐ 6-8 p.m. ☐ after 8 p.m.
DEPARTURE DATE ____________________
(checkout time is 1 p.m.)

HAVE A CREDIT CARD? You can register at our QUICK CHECK desk. If you wish to take advantage of this credit card PAYMENT GUARANTEED reservation, please complete the information below.

☐ AMERICAN EXPRESS
☐ DINNERS CLUB
☐ CARTE BLANCHE

Expiration Date __________
____
Credit Card No.

Signature ____________________

MEDICAL SOCIETY OF THE STATE OF NEW YORK
Annual Meeting of the House of Delegates
June 6-9, 1985
Annual CME Assembly
June 7-9, 1985

Circle preferred rate:

Singles:	$120	$135	$145
Double/Twin:	$145	$160	$170
Executive Tower:	$190, Single/Double		
Suites: (P&1):	$315	$350	
Suites: (P&2):	$425	$460	

Rates subject to 8¼% N.Y. State sales tax and $2.00 per room per night N.Y.C. occupancy tax. (If room at the rate requested is unavailable, one at the nearest available rate will be reserved.)

General Information

The Medical Society of the State of New York's 1985 Annual CME Assembly sessions and exhibits are open to all physicians, their guests, and the allied professions. The two and one-half days of meetings will provide the individual physician with a possible 18 credits out of a total of 93 Category 1, CME credit hours being offered. Over 135 outstanding speakers, including a number from out-of-state, will present papers at 30 sessions.

On Friday, June 7th and Sunday, June 9th, five sessions of Meet the Professor will be held simultaneously. These two-hour sessions will include a continental breakfast.

The theme of the CME sessions will be *"Health Care Today"*. Some highlights of this year's sessions are, the Developments in the Relationship between Diet and Cancer; Update on Pacemakers; Computer Applications in Clinical Medicine; the Physician as a Witness; and today's concepts of Chest Diseases diagnosis and management. Our program will offer the clinician new approaches in diagnosis and treatment of diseases in a wide range of medical specialties. Don't miss this opportunity to gain Category 1 credits over a weekend when you are free from office hours. This year there is no registration fee for member physicians which makes it an economical way to brush up on your skills. Professional representatives from more than 70 companies will be in the Rhinelander Gallery Exhibit Hall to discuss their products and services. Technical Exhibits will include drug products, clinical laboratories, office equipment, computer services, educational and financial services.

COURTESY N.Y CONVENTION & VISITORS BUREAU

PULITZER FOUNTAIN AT GRAND ARMY PLAZA

For the first time since 1979, the CME Assembly will be combined with the Annual Meeting of the House of Delegates, and the Auxiliary to the Medical Society.

SPECIAL EVENTS

Thursday, June 6: The Annual Dinner Dance will be held in the Trianon Ballroom of the New York Hilton, in honor of Allison B. Landolt, M.D., President, preceded by a reception in the Mercury Ballroom at 7:00 p.m. All attendees are welcome. Tickets should be ordered in advance from the Medical Society.

Friday, June 7 and Saturday June 8: A reception for physicians, the allied professions, and exhibitors will be held on both days in the Exhibit Hall, between 5:00 p.m. and 6:00 p.m., compliments of the Medical Society of the State of New York.

HOTEL RESERVATIONS

Physicians must make their own reservations with the New York Hilton no later than May 16, 1985. If making reservations by telephone, please mention that you are attending the Medical Society Convention for billing at the special rate.

REGISTRATION

CME Registration will take place in the Rhinelander Gallery Exhibit Hall, located on the second floor of the hotel and accessible by escalators from the lobby. Registration will take place at the following hours:

Friday, June 7th–
7:30 a.m.-5:00 p.m.
Saturday, June 8th–
8:00 a.m.-5:00 p.m.
Sunday, June 9th–
7:30 a.m.-12:30 p.m.

House of Delegates Registration will take place on the third floor, West Promenade, at the following hours: Wednesday, June 5 from 12 noon-7:30 p.m.; Thursday, June 6 from 8:30 a.m.-5:30 p.m.; Friday, June 7 from 8:30 a.m.-5:30 p.m.; Saturday, June 8 from 8:30 a.m.-5:30 p.m.; and Sunday, June 9 from 8:30 a.m.-12 noon.

CME MEETING HOURS

Friday, June 7th–
Meet the Professor–
8:00 a.m.-10:00 a.m.
All other sessions–
10:00 a.m.-1:00 p.m.
2:00 p.m.-5:00 p.m.

Saturday, June 8th–
All sessions–
10:00 a.m.-1:00 p.m.
2:00 p.m.-5:00 p.m.

Sunday, June 9th–
Meet the Professor–
8:00 a.m.-10:00 a.m.
All other sessions–
10:00 a.m.-1:00 p.m.

1985 ANNUAL CME ASSEMBLY

HEALTH CARE TODAY

Medical Society of the State of New York
New York Hilton, New York City

FRIDAY, JUNE 7

8:00 a.m. - 10:00 a.m.	Meet the Professor Breakfast Meetings (5)
10:00 a.m. - 1:00 p.m.	Cardiovascular Diseases, *Beekman Parlor* Pediatrics/Dermatology, *Sutton Parlor North* Obstetrics/Gynecology, *Sutton Parlor South* Physical Medicine & Rehabilitation, *Sutton Parlor Center* Neurosurgery, *Regent Parlor*
2:00 p.m. - 5:00 p.m.	Neurology, *Sutton Parlor Center* Medical Legal & Workers' Compensation/Occupational Medicine, *Beekman Parlor* Cancer Update, *Sutton Parlor North* Chest Diseases, *Sutton Parlor South* Computers in Medicine, *Regent Parlor*

SATURDAY, JUNE 8

10:00 a.m. - 1:00 p.m.	Ophthalmology (All day), *Sutton Parlor North* Urology/Pathology, *Sutton Parlor Center* Internal Medicine (All day), *Beekman Parlor* General Surgery/Orthopedic Surgery/Hand Surgery/Gastroenterology Anesthesiology/Radiology, *Sutton Parlor South* Psychiatry/Alcoholism (All day), *Regent Parlor*
2:00 p.m. - 5:00 p.m.	Ophthalmology, *Sutton Parlor North* Otolaryngology—Head and Neck Surgery, *Sutton Parlor Center* Emergency Medicine, *Sutton Parlor South* Internal Medicine, *Beekman Parlor* Psychiatry/Alcoholism, *Regent Parlor*

SUNDAY, JUNE 9

8:00 a.m. - 10:00 a.m.	Meet the Professor Breakfast Meetings (5)
10:00 a.m. - 1:00 p.m.	Allergy & Immunology, *Beekman Parlor* Women's Medical Society of New York State/Family Practice, *Sutton Parlor Center* The Physician as a Witness, *Sutton Parlor North*

◆

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis.

◆

Technical Exhibits

Advance Registration Form

Complete and return this form to the Medical Society address as indicated below.

1985 Annual CME Assembly, June 7–9

The New York Hilton, New York City

PLEASE PRINT

NAME ______________________________
Last *First*

Address ______________________________

City/State/Zip ______________________________

Medical Specialty ______________________________

REGISTRATION FEE

Please check category

☐ Non-Member Physician	$50.00
☐ Allied Profession	$10.00

Please check category

- ☐ Nurse
- ☐ Physician Assistant
- ☐ Dentist
- ☐ Lab Technician
- ☐ PT/OT
- ☐ Other (specify) ______________

NO FEE

- ☐ MSSNY Active Member
- ☐ MSSNY Life Member
- ☐ Resident/Intern/Fellow
- ☐ Medical Student
- ☐ Medical Assistant
- ☐ Speaker
- ☐ Spouse/Family
- ☐ News Media/Medical Writer

MEET THE PROFESSOR—Continental Breakfast Meetings.

Fee: $35.00 per session. Time: 8:00 a.m.-10:00 a.m. Please indicate session(s) attending. Each session is limited to 30 persons.

Friday, June 7

- ☐ Charles Hollander, M.D.
- ☐ Harold Itskovitz, M.D.
- ☐ Harold Lebovitz, M.D.
- ☐ Donald Louria, M.D.
- ☐ Carroll Leevy, M.D.

Sunday, June 9

- ☐ Arnold Aberman, M.D.
- ☐ Michael Grieco, M.D.
- ☐ Thomas Kantor, M.D.
- ☐ Simon Pollack, M.D.
- ☐ Anthony Rebuck, M.D.

(see this issue for subjects to be presented by each Professor)

REMITTANCE

Registration Fee $ ____________
Meet the Professor session(s) $ ____________
TOTAL $ ____________

Please make check payable to:
Medical Society of the State of New York, and return this form with remittance to: Division of Meeting Services, MSSNY, 420 Lakeville RD., P.O. Box 5404, Lake Success, N.Y. 11042

Important: Your ID Badge will be held at the MSSNY Registration Desk located in the Exhibit Hall, on the second floor of the New York Hilton.

Meet the Professor - June 7th and 9th

at the Medical Society of the State of New York's Annual CME Assembly, June 7-9, 1985, the New York Hilton, New York City. Five continental breakfast meetings, 2 hours each, will be offered on Friday and Sunday mornings from 8:00 a.m.-10:00 a.m. Sessions are limited to 30 attendees each, and there is a $35.00 fee. Please see the advance registration form in this section.

Hollander

Itskovitz

Lebovitz

Louria

Leevy

Aberman

FRIDAY, JUNE 7

Charles Hollander, M.D.
Professor of Medicine & Chief of Endocrinology, New York University School of Medicine, New York City. To provide a very brief review of our understanding of the physiology and pathophysiology of thyroid, pituitary and hypothalamic disorders.

Harold D. Itskovitz, M.D.
Professor of Medicine & Pharmacology, New York Medical College, Valhalla, New York. To familiarize the physician with new drugs used to treat hypertension; to stress changing concepts concerned with etiology, work-up and management of hypertension.

Harold E. Lebovitz, M.D.
Chief, Endrocrinology & Metabolism, SUNY, Downstate Medical Center, Brooklyn, New York. To provide an understanding of the pathophysiology of the two major types of diabetes, and to develop a rational set of goals for the treatment of patients with diabetes.

Donald Louria, M.D.
Professor & Chairman, Department of Preventive Medicine & Community Health, New Jersey Medical School, College of Medicine & Dentistry of New Jersey, Newark, New Jersey. To develop a programatic approach to the seriously ill patient with a putative infection.

Carroll Leevy, M.D.
Professor & Chairman, Department of Medicine, New Jersey Medical School, College of Medicine & Dentistry of New Jersey, Newark, New Jersey. Immunologic abnormalities are key factors in pathogenesis of hepatitus due to infections, alcoholism, and drug induced toxicity. Prevention, diagnosis and treatment have been greatly facilitated by recent advances in genetics, availabilty of monoclonal antibodies and the use of recombinant D.N.D. (Deoxyribonucleic Acid) technology.

SUNDAY, JUNE 9

Arnold Aberman, M.D.
Chief of Medicine, Mt. Sinai Hospital, University of Toronto Faculty of Medicine, Toronto, Canada. To approach the fluid and electrolyte management in the hospitalized patient with particular emphasis on hypo and hypernatremia.

Continued on next page

Meet the Professor - continued

Michael H. Grieco, M.D.

Director, R.A. Cooke Institute of Allergy, St. Luke's-Roosevelt Hospital Center, New York City. A review of current concepts concerning the immunologic and nonimmunologic mechanisms involved on pathogenesis of bronchial asthma, and review the management of ambulatory asthmatics.

Grieco

Kantor

Thomas Kantor, M.D.

Professor of Medicine; Chief, Division of Rheumatology, New York University School of Medicine, New York City. This session will demonstrate the all-purpose analgetic activity of the nonsteroidal, anti-inflammatory drugs in addition to their antiarthritic activity.

Simon Pollack, M.D.

Professor of Medicine, Albert Einstein College of Medicine, Bronx, New York. The objective of this presentation will be to explain current concepts of iron absorption and utilization, and the diagnosis and treatment of iron deficiency and iron overload.

Pollack

Rebuck

Anthony Rebuck, M.D.

Professor of Medicine, University of Toronto Faculty of Medicine, Toronto, Canada. Physicians attending this session will be made familiar with the proper use of oxygen in the every day care of caridac and pulmonary patients.

NEW YORK STATE
JOURNAL OF MEDICINE

February 1985 • Volume 85, Number 2

COMMENTARIES

Searching for alternatives to war

It is heartening to see physicians, scholars, and groups such as Physicians for Social Responsibility in the forefront of the worldwide debate on nuclear weapons. There are at least two issues central to this debate that deserve further scholarly study.

The first of these issues involves the way in which the basic problem is perceived by the medical profession. An increasing number of persons throughout the world feels that the central concern is not the use of nuclear weapons, but the broader issue of the use of organized violence for political purposes. The argument for this position can be illustrated by the current research, development, and stockpiling of chemical and biological weapons. The new technology of genetic engineering can be used to develop chemical and biological weapons as unpredictable and frightening as nuclear energy was just a few decades ago. While the streets of the cities around the world fill with demonstrators against nuclear weapons, the world powers are quietly amassing arsenals of new weapons.[1–3] The current debate on nuclear weapons concentrates on a symptom (nuclear weapons), while the disease (violence for political purposes) goes unaddressed, or while a new symptom (chemical and biological weapons) emerges.

The second critical and related issue is addressed in the eloquent article by Blinderman[4] in this issue of the *Journal.* This essay recounts the troubled history of the struggle that physicians and other scholars have had through the centuries with their conscience and the issue of war. How do we reconcile an ethical commitment to the health of humanity with the personal desire to defend ourselves from the aggression of others who want to take from us those things we hold venerable? In theological circles this debate takes form in what is called the theory of "just war"—that war is at times justifiable in defense of the things we hold sacred. Blinderman chronicles those who protested the making of war only to ultimately support their own country's "just war." He also recalls those few brave souls who held out against all war. Referring to nuclear war in a sobering conclusion, Blinderman issues the following challenge: "Where statesmen, clerics, teachers, philosophers, and lawyers have failed in the past, physicians have an almost sacred responsibility to succeed."

If the medical profession is to be equal to this challenge, there is at least one proposal that should be read and discussed by all physicians and medical societies. It has been termed non-violent civilian-based defense; and the process of converting from the violent defense systems now common to civilian-based defense has been termed "transarmament," to distinguish it from "disarmament."[5]

The concept of using non-violence to accomplish political or social goals is not new. An increasing number of researchers feels that the American colonists were in the process of accomplishing most of their goals by non-violence prior to the Revolution.[6] Europeans caused serious disruptions of German objectives during World War II using non-violence—the Dutch being the most notable example. Ghandi's work in India is the best known instance of non-violent political action, but Solidarity in Poland may soon lay claim to that title. Solidarity has officially adopted non-violent tactics as its means of resistance to outside control of its political system.[7] There have been casualties in Poland, but far fewer than would occur in warfare, and death in the struggle there means an almost instant place in Polish history.

The proposal to substitute non-violent civilian-based defense for violent defense systems is more recent in origin. In Europe, both Sweden and, more recently, France, have formally addressed the possibilities of "transarming."

Neither country has put forth any specific policy proposals, but the study of these possibilities at a national level is encouraging. In the United States, Gene Sharp, PhD (Director, Program on Non-violent Sanctions, Center for International Affairs, Harvard University) is the leading scholar in the field of non-violent alternatives.[5,8,9] He notes that past attempts at non-violence have been relatively primitive. His point is that no nation in history has ever undertaken the systematic study, training, and preparation necessary for the use of non-violence in advance of the need for such a system. What would a national "Manhattan Project" of non-violence produce? That is, what could a nation such as the United States accomplish if it dedicated its resources to the development of a non-violent defense system with the same intensity that it used to develop the first nuclear weapon?

Transarmament does not rest on morality, but on hard political realities and pragmatic approaches. Inherent in all current defense debates is the concept of deterrence. It is at the heart of the United States government's strategy of a strong nuclear force to deter either a nuclear attack or an attack by conventional forces in Europe. Transarming to a non-violent civilian-based system would approach deterrence by making a society virtually unconquerable. Advance preparation would ready every segment of a society including the print and broadcast media, the educational and scientific sectors, the economic system, the clergy, labor, government, courts, and the general population. Sharp has catalogued 198 methods that can be used in non-violent resistance.[9] Some examples include work slowdowns, strikes and pickets, nonobedience in the absence of direct supervision, sit-ins, media publicity and protest, selective boycotts, and protest prayer and worship.

Transarmament clearly separates a nation's defense posture from aggression, since it cannot be used for aggression. It resolves the ancient ethical controversy over a "just war." Since participation by citizens is voluntary, it is inherently democratic. It goes beyond conflict resolution because in the end, when a society feels it cannot compromise on certain principles, it allows for a method of applying sanctions. It can be used with equal effectiveness as a deterrent against the four grave political problems of the modern world: dictatorship, war, genocide, and systems of social oppression. Its disadvantages include the likelihood of casualties. A major difficulty would be overcoming the current large economic investment in the military. It is not clear how much of a factor transarmament might be in deterring nuclear war, but it certainly would make a nation appear less hostile to an existing nuclear power and could enhance a country's bargaining position. This issue needs intensive study.

Non-violence follows in the healing profession's finest traditions of preventing ill health. We fear the abandonment of our vast, violent arsenals of defense, but we also feel the destabilizing influence of these systems. At the very least, the reasoned study and criticism of a rational transition from violent to non-violent means of defense deserves our attention as both citizens and professionals who are involved in the often emotional debate on war and the future of the world.

BERY ENGEBRETSEN, MD
Director
Primary Care Center
Broadlawns Medical Center
18th and Hickman Road
Des Moines, IA 50314

1. Editorial: Chemical & Bacteriological Weapons. *Lancet* 1984; 11:141-143.
2. Blum A: Chemical weapons: a question of morality. *NY State J Med* 1984; 84:104-105.
3. Schechter DC: Unorthodox warfare. *NY State J Med* 1971; 71:2560-2568.
4. Blinderman, A: The physician and nuclear war. A layman's view. *NY State J Med* 1985; 85:77-79.
5. Sharp, G: *Social Power & Political Freedom.* Boston, Porter Sargeant, 1980.
6. McCarthy RM: Nonviolent action in defense of social and political institutions: Massachusetts in 1774. *Civilian Based Defense* 1984; 2:1-5.
7. Zielonka J: Lessons from Poland. *Civilian Based Defense* 1983; 1:1-3.
8. Sharp G: *Making the Abolition of War a Realistic Goal.* New York, World Policy Institute, 1980.
9. Sharp G: *The Politics of Nonviolent Action. II. Methods of Nonviolent Action.* Boston, Porter Sargent, 1974.

Preventing alcohol-related violence: the physician's role

The associations between alcohol abuse and a range of violent behaviors and traumatic events are well known.[1] Both the physiologic effects of alcohol as a drug and the psychosocial consequences of an ongoing pattern of alcohol abuse interact with other factors to cause needless tragic deaths and injuries. The Department of Health and Human Services' *Fourth Special Report to the United States Congress on Alcohol and Health* cites reports of alcohol involvement in half of fatal automobile crashes nationwide. A study by Boston University School of Law showed that 48% of drivers responsible for their own fatal injury were problem drinkers, as were 41% of those who survived but killed others, and 31% who fatally injured a pedestrian.[2] Falls, industrial accidents, drownings, suicides, and criminal acts such as child abuse have been linked to the use and abuse of alcohol.

Because fatal outcomes are so frequent among abusers of alcohol, data from medical examiners help to document this association. In a recent study, data from 3,293 cases investigated by the Erie County (NY) Medical Examiner were analyzed.[3] This represented all persons 15 years of age or older who had died within eight hours of injury during a 10-year period. Using a standard blood alcohol level of 0.1 grams % as a measure of intoxication, the investigators found that 35% of the men and 22% of the women were intoxicated at the time of death. This included 44% of drivers, 33% of passengers, 30% of pedestrians, and 23% of bicyclists. Twenty-one percent of those who died in falls, 25% of those who drowned, and 32% of homicide victims were also legally intoxicated at the time of death. Since the ability of alcohol at such levels to produce impaired coordination, reaction time, and judgment is well known, it would be absurd to suppose that intoxication did not play a significant causative role in many of these violent deaths.

What role can the practicing physician play in preventing alcohol-related violence? To be a part of effective prevention, the physician must become sensitive to problem drinkers and their families in his or her practice; develop a systematic method for diagnosis, intervention, and treatment or referral; follow these families over time to support the lengthy and difficult recovery process; and speak out in support of enlightened public policies relating to alcohol problems.

Nationwide surveys have indicated that approximately 10% of adults who drink alcoholic beverages report significant alcohol-related problems.[4] Because problem drinkers tend to develop a variety of both physical and emotional symptoms, they are likely to visit health care facilities of all kinds more frequently than other patients. Thus, medical practices are ideal settings for screening and early intervention for alcohol problems. Research on the effects of treatment has generally demonstrated that the patient who has not lost social stability—that is, remains employed and married or in a stable family relationship—has the best prognosis, regardless of the treatment setting or program type. Early and successful treatment of alcoholic patients and their families will prevent future traumatic death and injury. Even in patients who relapse or are unwilling to cooperate in therapy, treatment of family members can prepare them to minimize the threat of injury to themselves and others by understanding the illness and by taking protective steps. Such steps might include not letting the alcoholic member have access to the family car, not engaging in arguments when he or she has been drinking, installing fire safety devices, and similar measures. The changes in attitude and behavior that occur in family members who participate in self-help groups or therapy programs have also been found to help the alcoholic patient to develop motivation for treatment.

Considerable evidence has accumulated in recent years that policies that influence the availability of alcoholic beverages, such as increasing taxes and raising the minimum purchase age, are associated with significant decreases in alcohol-related mortality.[5] One example is the relationship between the real price of rum (price divided by average disposable income) and the number of road traffic accidents in Trinidad and Tobago between 1966 and 1975.[6] As the relative price of rum fell, accidents increased. Raising taxes increased the beverage price and was associated with a fall in the number of accidents. Similar data have been gathered from the United States, Canada, and Europe.[5] Policies relating to law enforcement and criminal penalties, and those that promote rehabilitation for alcohol-impaired drivers and other offenders have also received increased attention in recent years as prevention measures.

A model public policy statement issued in 1984 by the American Assembly of Columbia University makes recommendations for sweeping changes in alcohol policy based on current knowledge.[7] Individual physicians and organized medicine in general can play pivotal roles in promoting such policies at all levels of government.

SHEILA B. BLUME, MD
Medical Director
Alcoholism & Compulsive Gambling Programs
South Oaks Hospital
Amityville, NY 11701

1. DeLuca J, Wallace J (eds): *Fourth Special Report to the US Congress on Alcohol and Health.* Washington, DC, Dept of Health and Human Services, 1981.
2. Boston University School of Law: *Analysis of Drivers Most Responsible for Fatal Accidents Versus a Control Sample.* Final report to US Dept of Transportation No. DOT HS-801-916, Washington, DC, Dept of Transportation, 1976.
3. Abel E, Zeidenberg P, Regan S: Alcohol and violent death—Erie County, NY, 1973-1983. *Morbid Mortal Wkly Report*, 1984: 33:226-227.
4. Blume SB: National patterns of alcohol use and abuse. *Ann NY Acad Sci* 1981; 362:4-15.
5. Moore MH, Gerstein DR (eds): *Alcohol and Public Policy: Beyond the Shadow of Prohibition.* Washington, DC, National Academy Press, 1981.
6. Beaubrun MH: *Epidemiological Research in the Caribbean Context: Road Accidents and the Price of Rum in Trinidad and Tobago 1966-1975.* Toronto, Addiction Research Foundation Seminar on International Collaboration, 1976.
7. American Assembly: *Public Policy on Alcohol Problems, 66th American Assembly*, New York, Columbia University, 1984.

Psychiatry and the prevention of violence

The debate over the role of physicians, specifically those in the field of psychiatry, in the prevention of violent behavior has escalated in the wake of publicity surrounding events such as the attempted assassination of the President. Most of the attention has focused on the issue of retribution: What is to be done with the mentally ill person who has committed a violent act? A less well addressed but more crucial issue for the practitioner relates to the responsibilities and authority of the clinician faced with a patient who is suspected of being dangerous to others.

It has been asserted that psychiatrists and other mental health professionals are notoriously incapable of predicting violent behavior in patients.[1] Specifically, whereas mental health professionals may be quite accurate in predicting the absence of violence, they tend to produce an inordinate proportion of false positives (that is, those persons who were predicted to become violent but who did not commit an act of violence). Thus, the argument goes, mental health professionals should have no authority to commit a patient involuntarily based on an assessment of potential violent behavior. In recent years, laws concerning commitment have been passed in many states; the laws have progressively curtailed the clinician's authority to commit a patient because he or she presents a "clear and immediate danger"—usually no more clearly defined. Unfortunately, the studies on which these assumptions are based have often failed to take into account the effect of treatment on the clinician's predictive accuracy. If it is reasonable to assume that competent psychiatrists who consider a patient to be potentially violent will initiate treatment to alleviate the mental illness that underlies the violent behavior, then a negative correlation might be expected between prediction of violence and subsequent violent behavior. That is, those considered to be most violent might be treated most intensively and, therefore, would be least likely to commit a violent act.

It has been reported that for certain diagnostic categories (acute psychoses, for example), violent behavior is almost exclusively confined to the psychotic episode.[2] For these individuals, commitment to a mental hospital for treatment of the acute episode can be considered preventive of such behavior. However, individuals with other conditions such as personality disorders may engage in violent behavior without concomitant psychosis. For such individuals, violent behavior may represent a deliberate, criminal act, and not the consequence of a treatable illness. A psychiatrist examining such a person before the commission of a violent act may not find evidence sufficient to institute involuntary commitment, even though there may be a clear danger of future violent behavior.

In the latter case, clinicians are often placed in the dilemma of having recognized a potential future danger but being legally incapable of acting to prevent it. In this situation, the role of a law enforcement agency may be crucial. Such a dilemma is highlighted by the Tarasoff case as reviewed by Schwartz in this issue of the *Journal.*[3] Although the patient had clearly threatened the life of the victim, there were not sufficient grounds for commitment and treatment. The clinician was found culpable for not notifying the victim, but Schwartz rightly raises the question of what legal steps could have been taken to prevent the murder even if the victim had been warned. In the absence of premonitory criminal behavior, such as harassment, law enforcement agencies are legally powerless until a crime is committed. Thus, the clinician may be forced to choose between therapeutic preventive detention—involuntary commitment in the absence of sufficient evidence of a treatable mental illness—which may incur a risk of legal jeopardy for unlawful imprisonment, or a notification of law enforcement agencies and, if possible, victims, which is a legal course of action. The latter may result in risk for both the victim and the clinician if there is insufficient evidence to permit criminal detention. Even in the presence of criminal behavior, the criminal is often arrested only to be released on bail, thus permitting the risk to continue.

Finally, what is the role of clinical judgment in the decision of whether to inform potential victims? It is not uncommon for distressed patients to make statements in therapy that could be construed as threatening to others. The clinician has traditionally used his clinical knowledge of the patient to assess the seriousness of the threat and to decide whether notification of victims is indicated. The recent trend of legal decisions could be seen as an intrusion on this clinical prerogative by making the clinician liable should the decision prove to be in error. An unstated issue in the Tarasoff case is what would have happened had the therapist warned no one and not documented this threat, since, presumably, Poddar did not reveal his threats to others? Would any liability have been incurred? Unfortunately, for some clinicians whose primary objective is to avoid legal jeopardy, this may appear to be the most prudent course of action unless both clinicians and legal authorities can agree on a mutually satisfactory set of guidelines regarding clinician authority and responsibility that does not, as is the current situation, force the clinician to choose between legally untenable paths.

THOMAS J. CRAIG, MD
Department of Psychiatry
and Behavioral Science
Health Sciences Center
SUNY-Stony Brook
Stony Brook, NY 11794-8101

1. Kroll J, Mackenzie TB: When psychiatrists are liable: risk management and violent patients. *Hospital and Community Psychiatry* 1983; 34:29-37.
2. Craig TJ: An epidemiological study of problems associated with violence among psychiatric inpatients. *Am J Psychiatry* 1982; 139:1262-1266.
3. Schwartz DW: Adam Berwid and the obligation to assess dangerousness in New York State. *NY State J Med* 1985; 85:67-68.

This section is designed to offer rapid publication (usually within 1-3 months) of new clinical, epidemiologic, and laboratory observations, as well as of comments on articles published in the Journal *or on any topic related to the role of the physician. Letters should be typed double-spaced and should include the professional affiliation, highest academic degree, address, and signature of each author. Preference is given to letters not exceeding 450 words. All letters are personally reviewed and acknowledged by the Editor. Address correspondence to Editor,* New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042.*

Nd:YAG laser photocoagulation

TO THE EDITOR: In his discussion of the options available for the palliation of obstructing carcinomas of the esophagus, Dr Santos[1] omits a promising new therapy: endoscopic neodymium:ytrium, aluminum, garnet (Nd:YAG) laser photocoagulation. To date at St. Luke's Hospital, 14 patients with unresectable esophageal carcinoma have been treated with the laser for symptomatic esophageal obstruction. Thirteen of the 14 patients experienced relief of their obstruction and were able to tolerate at least a soft diet. The one patient who failed to tolerate this diet after laser therapy had involvement of the upper esophageal sphincter. This patient was, however, able to clear oral secretions after laser therapy and did not suffer significant pulmonary aspirations. We have not treated patients with tracheoesophageal fistulas with the laser, and we agree with Santos that intraluminal intubation is the appropriate form of therapy in such patients.

The patients have been followed for two to 11 months after initial treatment. During this period they have received follow-up therapy every three to four weeks, usually as outpatients. Five patients have died, but none died primarily as a result of esophageal obstruction. Among all the patients, the mean survival has been 19 weeks. The patient who has survived the longest had complete esophageal obstruction after having received the maximum acceptable therapeutic radiation dose. The obstruction was relieved after two laser treatment sessions, and he is alive and well, eating a normal diet, and gaining weight 44 weeks after he first sought treatment.

Other than two cases of "benign pneumoperitoneum" associated with the high-flow carbon dioxide gas used for cooling the light guide, there have been no complications. The pneumoperitoneum resolved spontaneously in both patients.

In our experience, Nd:YAG laser photocoagulation has proven to be an effective treatment of malignant esophageal obstruction. The esophageal lumen is rapidly reestablished, and the morbidity and mortality associated with surgery is avoided. We have had similar success using the laser in the palliative treatment of bleeding and obstruction due to tumors of the stomach, duodenum, and recto-sigmoid.

RICHARD S. Mc CRAY, MD
WILLIAM P. WINKLER, MD
St. Luke's Hospital Center
114th Street and Amsterdam Avenue
New York, NY 10025

---

1. Santos GH: Palliation of unresectable esophageal carcinoma by intraluminal intubation. *NY State J Med* 1984; 84:449-451.

A program for rubella surveillance and control

TO THE EDITOR: Rubella surveillance and control are important issues not only for the prevention of congenital rubella syndrome, but also for the management of sporadic outbreaks of the disease that may affect pregnant women.[1,2] A man who lacks immunity to rubella also serves as a potential reservoir of infection.

A program of rubella surveillance and control is in progress at Lutheran Medical Center both in the outpatient facility (Sunset Park Family Health Center) and in the inpatient obstetric service. Since June 9, 1970 we have attempted to screen all patients who are more than 12 years of age for rubella susceptibility. Immunization has been offered to those who have no evidence of immunity to the disease.

We have recently examined the rubella susceptibility rates among women of varying socioeconomic status as part of a Quality Assurance Program aimed at vaccinating all patients who are seronegative for rubella. As suggested by the Committee on Infectious Diseases of the American Academy of Pediatrics,[3] seronegative women were vaccinated after giving birth or upon termination of the pregnancy. A difference in the rubella susceptibility rate was anticipated because of a dissimilarity in living conditions among the women and data suggesting different patterns of medical use in this population.[4]

A review of 5,534 sera samples tested with the hemagglutination inhibition method from 1978 to 1982 gave the following rubella susceptibility rates for women: patients on Medicaid, 17.6% (314/1785); working, low income, 17.4% (324/1858); middle income 17.1% (324/1891). These values are not statistically different and are compatible with previously reported data.[5,6] It is likely that the hemagglutination inhibition test artificially inflated the pool of susceptible subjects[7]; the ELISA method is currently used.

No susceptible women were inadvertently vaccinated while pregnant. No cases of congenital rubella syndrome were noted in the study group. A preliminary review of seronegative patients disclosed a lack of follow-up rubella vaccinations in the majority of patients. This was most alarming among the women who delivered. In addition, lack of serologic testing for men was noted. The following Quality Assurance Programs were instituted and are in progress: (1) All women who give birth or have their pregnancies terminated are vaccinated immediately before being discharged from the hospital. Since this program began, four women have been vaccinated each month. (2) Emphasis has been placed on vaccinating all other seronegative patients, including men, with follow-up audits on a regular basis. Finally, in reviewing the rubella surveillance program, it was apparent that susceptible men were not being identified. We have since attempted to vaccinate this group, as they pose a threat when in contact with pregnant women.

ERNEST B. VISCONTI, MD
SUZANNE HOWE, MD
KATHYRN I. MURRAY
Lutheran Medical Center
150 55th St
Brooklyn, NY 11220

---

1. Rubella outbreak among office workers. *City Health Information* 1983; 2:25.
2. Horstmann DM: Rubella: the challenge of its control. *J Infect Dis* 1971; 123:640-654.

3. American Academy of Pediatrics: *Report of the Committee on Infectious Diseases*. 1982; (19 ed): 235.

4. US Dept of Health and Human Services: *Health of the Disadvantaged Chart Book-II*. 1980; 633:11-5a.

5. Blouse LE, Lathrop GD, Dupuy HJ, Ball RJ: Rubella screening and vaccination program for US air force trainees: an analysis of findings. *Am J Public Health* 1982; 72:280-283.

6. Robinson RG, Dudenhoeffer FE, Holroyd HJ, Baker LR, Bernstein DI, Cheery JD: Rubella immunity in older children, teenagers, and young adults: a comparison of immunity in those previously immunized with those unimmunized. *J Pediatr* 1982; 101:188-191.

7. Harnett GB, Palmer CA, Mackay-Scollay EM: Single-radial-hemolysis test for the assay of rubella antibody in antenatal, vaccinated, and rubella virus-infected patients. *J Infect Dis* 1979; 140:937-944.

Extra-adrenal myelolipoma

TO THE EDITOR: Myelolipoma is a benign tumor containing hematopoietic and fatty elements, most often encountered in the adrenal glands. Extra-adrenal myelolipomas are extremely rare tumors composed of fat and hematopoietic tissue without adrenal elements, occurring in patients without marked hematologic disturbances.[1,2] We describe the incidental finding of a presacral myelolipoma in a woman with macrocytic anemia and breast carcinoma for which she had undergone surgery.

Case Report. An 84-year-old woman was admitted to the hospital for sudden loss of consciousness. No further information was available as to her present medical condition; she had undergone a right radical mastectomy for breast carcinoma several years previously.

She was a cachectic woman, not oriented in time, place, or person. A neurologic examination was unremarkable. Other significant findings included marked pitting edema of the extremities, bilateral rales in the chest, and a grade II/VI systolic murmur in the precordium. Laboratory investigations revealed the presence of megaloblastic anemia: hemoglobin, 7.2 g/dL; hematocrit, 22.4%; mean corpuscular volume, 117 μ^3; mean corpuscular hemoglobin, 39 $\mu\mu$g; mean corpuscular hemoglobin concentration, 32.3%; white blood cell count, $8.5 \times 10^3/\text{mm}^3$; differential, 96% polymorphonuclear leukocytes and 4% lymphocytes; peripheral smear, macrocytic hypochromic anemia with anisopoikilocytosis and hypersegmentation of neutrophils; platelets, adequate; serum B_{12}, 159 pg/mL (normal range, 200-900 pg/mL); serum folate, 5 mg/mL (normal range, 4-20 mg/mL); bone marrow, megaloblastic changes. The blood chemistry test results were within normal limits except for a mild elevation of serum bilirubin (0.4 g/dL). Sputum cultures grew Gram positive diplococci and *Staphylococcus aureus*.

The patient was treated with antibiotics, respiratory support, vitamin B_{12}, and hematinics, but she expired shortly after admission.

At autopsy a large reddish blue, well encapsulated cystic mass was found lying over the anterior aspect of the sacrum between the two common iliac arteries. The mass measured 13 × 10 × 10 cm and weighed 150 g. On sectioning, a variegated cut surface with yellowish fat and tan firm areas was seen. On microscopic evaluation mature lipocytes were seen to mingle with the hematopoietic elements, including mature erythroid, myeloid, and megakaryocytic precursors. Erythroid hyperplasia with moderate megaloblastic change and mild plasmacytosis was also evident. Bony spicules were absent (Fig 1). The other significant findings at autopsy included severe coronary and aortic atherosclerosis, left ventricular hypertrophy, bilateral emphysema, pulmonary embolism, bronchopneumonia of the upper lobe of the right lung, bilateral pleural effusion, congestion of the liver, congestive splenomegaly, atrophic gastritis, and marked megaloblastic changes in the bone marrow.

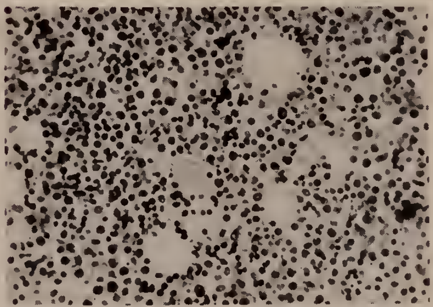


FIGURE 1. Myelolipoma with plasmacytosis and megaloblastic changes. (hematoxylin and eosin stain, original magnification × 125)

Discussion. Extra-adrenal myelolipomas have recently been reviewed.[1,2] The age at presentation ranges from 40 to 81 years, the tumor size varies from 2.5 cm to 16 cm, and the weight ranges from 25 g to 790 g.[1,2] The tumors are almost always encapsulated and well circumscribed.[1] The most frequent locations include the presacral region, followed by the intrathoracic, paravertebral, retroperitoneal, peri-renal, and gastric regions, and the right iliac fossa.[1,2] Associations with other conditions in previously reported cases include anemia (five cases, with two cases of megaloblastic anemia), malignancies (two cases), Paget's disease, and patients on steroid therapy (one case).[1,2]

The most important condition frequently confused with extraadrenal myelolipoma is mass-forming extramedullary hematopoiesis. Tumor-forming extramedullary hematopoiesis is rare in adults and is most often associated with myeloproliferative disorders; its symptoms include myelofibrosis, splenomegaly, hepatomegaly, anemia with anisopoikilocytosis and tear drop forms, presence in the peripheral blood of immature precursors, and an elevated leukocytic alkaline phosphatase.[3,4] These tumor masses may also be encountered in carcinomatosis with marrow replacement, hemolytic anemias, and anemias due to red cell enzyme defects.[1,2] The masses may be symptomatic and may produce compression of the spinal cord, proliferative obliteration of the vessels, ureter, or bowel, and thromboembolic disease of the lungs.[4] Occasionally, heterotopias of the bone marrow may occur in the absence of blood dyscrasias.[5] On pathologic examination, in contrast to extra-adrenal myelolipoma, mass-forming extramedullary hematopoiesis is poorly circumscribed, non-encapsulated, and does not contain fat as an integral part of the lesion.

The etiology of myelolipoma is a matter of controversy. Adrenal myelolipomas have occurred in patients with burns or cancer and have been associated with endocrinopathies such as Cushing's disease, suggesting that prolonged adrenocorticotropic hormone stimulation may contribute to the pathogenesis. Supraphysiologic levels of adrenocorticotropic hormone are known to be associated with fatty tissue increases in other body sites.[6] Associations with malignancies and steroid therapy have been observed in extra-adrenal myelolipomas[1,2]; their association with anemias suggests that prolonged erythropoietin stimulation may also play a role in the development of extra-adrenal myelolipomas.

V. JAGADHA, MD
G. RAMASWAMY, MD
Department of Pathology
New York Medical College
Metropolitan Hospital Center
1901 First Ave.
New York, NY 10029

Acknowledgment. Mrs Hazel Douglas provided secretarial assistance.

1. Chen KTK, Felix EL, Flam MS: Extraadrenal myelolipoma. *Am J Clin Pathol* 1982; 78:386-389.

2. Fowler MR, Williams RB, Alba JM, Byrd CR: Extra-adrenal myelolipomas compared with extramedullary hematopoietic tumors: a case of presacral myelolipoma. *Am J Surg Pathol* 1982; 6:363-374.

3. Dodge OG, Evans DMD: Haemopoiesis in a presacral fatty tumor (myelolipoma). *J Pathol Bacteriol* 1956; 72:313-317.

4. Glew RH, Haese WH, McIntyre PA: Myeloid metaplasia with myelofibrosis. The clinical spectrum of extramedullary hematopoiesis and tumor formation. *Johns Hopkins Med J* 1973; 132:253-270.

5. Saleeby ER: Heterotopia of bone marrow without apparent cause. *Am J Pathol* 1925; 1:69-78.

6. Selye H, Stone H: Hormonally induced transformation of adrenal into myeloid tissue. *Am J Pathol* 1950; 26:211-233.

The wounds of war

With particular reference to 1861–1865

William D. Sharpe, MD

And there are some who have no memorial,
who have perished as though they had not lived;
They have become as though they had not been born,
and so have their children after them.
—*Ecclesiasticus 44.9*

War is probably the oldest organized human activity involving groups larger than an extended family. Rivalry over territory may be one explanation for war, but it may be that men like to fight and obscurely appreciate that captives (when neither killed nor eaten) strengthen the gene pool. Ancient wars were brief and evidence for them is sparse. A few depressed skull fractures and an occasional skeleton with an embedded arrowhead cannot define where random homicides or hunting accidents ended and wars began. Classical Greek wars were modest affairs, usually occurring in the fall during hunting season after the crops were in and lasting a few weeks. The Roman army, organized for conquest and occupation, not only decimated cowardly regiments but provided steady employment and upward social mobility for alien tribes. Not until real national and religious animosities developed did war become all-out slaughter.

SOMA

Condemned by Pope Innocent II in 1139 as "hateful to God and unfit for Christians," the crossbow was permitted to be used against Muslims, and Christians eventually used crossbows against one another. This hateful weapon fell into disfavor only after the military application of gunpowder. The earlier form of the longbow—the favorite English weapon—was clumsy, but boys began to practice with it as soon as their epiphyses closed. A reasonably competent longbowman could put an iron-tipped arrow through a man in armor at 300 ft. The crossbow was a major improvement. An ordinary crossbow had a pull of about 350 lb, and crossbowmen wore broad leather or canvas belts to prevent dislocating their spines during the pull. By the end of the 14th century crossbows had spring steel bows and used gears or pulleys to attain a pull of between 1,000 and 1,200 lb.[1] Whether used with an arrow or metal bolt, neither was trivial, but could transfix a man, immobilizing his ribcage as he suffocated or bled to death.

Ambroise Paré, whose experience of war was considerable, was jolted at Turin in 1537—he was a young man then, only 27 years of age. His words are worth quoting:

> We entred the throng in the Citty, and passed over the dead bodyes, and some which were not yet dead, we heard them cry under our horses feete. . . . Being in the Citty, I entred into a stable thinking to lodge my owne, and my mans horse, where I found foure dead souldiers, and three which were leaning against the wall, their faces wholly disfigured, and neither saw nor heard, nor spoake; and their clothes did yet flame with the gunpowder which had burnt them. Beholding them with pitty, there happened to come an old souldier, who asked me if there were any possible meanes to cure them, I told him no: he presently approached to them, and gently cut their throates without choler. . .[2]

The old soldier seems to have shocked the young surgeon, but each knew what he was about. Gunpowder was then a new weapon.

Ingenuity had progressed enough to make the American Civil War the bloodiest in Western history: caused by a combination of modern weapons and medieval medicine. Smooth bored muskets that fired round shot with low muzzle velocity were inaccurate beyond a few hundred yards, but a rifled barrel imparted rotary spin to soft .58 calibre conical Minié balls to increase range, velocity, and accuracy.[3] Confederate surgeon J. Julian Chisolm discussed this in 1864:

> This conical ball seldom fails to take the shortest cut through a cavity or limb, and it has at times been seen to pass through the bodies of two men and lodge in that of a third. Rarely are they deflected from their course, as is the round ball, which is turned by every little obstacle, taking up a position at striking variance with any rule of propulsive forces.

In an experiment, most conical shot traversed five one-inch planks lined up 20 inches apart from a distance of 500 yards. Small wonder that "this projectile seldom impinges upon a bone without leaving frightful traces of devastation. Such a conical ball rarely remains embedded, but, acting on the principle of a wedge, splits and comminutes the bone, driving the loose spiculae in every direction before it."[4]

Manassas, a pleasant town in northern Virginia, is near a little creek called Bull Run. The First Battle of Manassas,

---

Based on a presentation at the Symposium on Violence, Annual CME Assembly, Medical Society of the State of New York, New York City, June 30, 1984.

Address correspondence to Dr Sharpe, Director of Laboratories, Cabrini Medical Center, 227 East 19th St, New York, NY 10003.

July 18, 1861, was a rout; Second Manassas, August 29, 1862, a slaughter. After First Manassas, one soldier with his arm amputated above the elbow, another "with a large hole through both thighs and the scrotum," and a third with "a large hole through both cheeks, a broken jaw and his tongue nearly off" walked the 25 miles back to Washington.[5] At Second Manassas, wounded and killed Confederate soldiers comprised 17% of the 55,000 rebels engaged. Seven regiments lost more than half of their troops, and the Twenty-first Georgia had 76% of its men killed or wounded. Second Manassas was not a weekend outing:

> "Brains, fractured skulls, broken arms and legs, and the human form mangled in every conceivable and inconceivable manner," exclaimed a Virginian as he rendered his account of an enemy charge. "At every step they take they see the piles of wounded and slain and their feet are slipping in the blood and brains of their comrades."[6]

Mere numbers of deaths and wounds are surreal. Chisolm provides corroborative detail:

> Should a bayonet or sabre wound transfix one of the natural cavities, the internal injury may be rapidly fatal from hemorrhage, or the injury inflicted upon the contained organs may, sooner or later, lead to the destruction of the patient by visceral inflammation. . . . The sabre-bayonet, when plunged into the body among the viscera, leaves but little work for the surgeon. Such cases seldom leave the battlefield alive.[7]

Conventional, non-nuclear war is quite lethal enough, as Chisolm noted:

> One of the most fearful cases on record . . . is one in which the entire face was carried away by a cannon-ball, leaving nothing but the skull proper appended to the vertebral column. The opened gullet marked the former site of the features. The patient lived ten hours, and from the frequent changes of position, and the squeezing of the hand when his pulse was taken, it was thought that consciousness remained up to the time of death.[8]

There were other ways to die. Cannons blew up and muskets backfired. Frequent fires burned many to death, especially the wounded who were unable to move. Horses rolled on cavalrymen, carts ran over people, locomotives cut them in two, and steam boiler and magazine explosions were both frequent and lethal. Camp diseases killed many times those lost in combat, especially dysentery, typhoid, small pox, epidemic measles, malaria, and yellow fever. Some soldiers went mad, some committed suicide or drank themselves to death, and an astonishing number were executed as a result of military court martials. The treatment of prisoners of war was, if anything, worse at Elmira than at Andersonville. Much of the random, and less random, violence in the American West after the war was due to psychopaths who first tasted blood during the war—one of the origins of America's abiding but bloody love affair with handguns.

Before Listerian surgery, what happened if a wounded man lived? Chisolm describes the protracted course of a gunshot wound of a limb:

> In twelve days the affected limb is swollen to sometimes half the size of the body; a violent phlegmonous inflammation may pervade the whole; serous effusion has taken place in the limb, and abscesses are forming in the great beds of cellular texture through which the ball has passed; from the wound pus is escaping in large quantities, impoverishing the blood and rendering the system irritable. In three months, if the patient has labored through this lengthened agony, the bones are carious; the abscesses are interminable sinuses, from which are kept up a continued discharge; the patient is pale and emaciated, with hectic flushes and diarrhea, and the constitutional strength ebbs to the lowest degree. All these conditions must be rapidly considered, and with them the more immediate dangers of mortification, and the remote dangers of erysipelas, pyaemia, and hectic [fevers], and the questionable utility of the limb, when, after several months of continued trials, the wound has been healed, but the limb remains weak, shrunken, stiff, painful, and nearly useless.[9]

Amputation made sense, but in an economy that depended on physical labor—on a farm or in a factory—most men who had lost an arm or leg were economic burdens, and they knew it.

PSYCHE

Deaths and wounds are easily counted, but war also wounds the human spirit and human society—deeply and, perhaps, permanently. Enormous fortunes can be made during wars by those who do not fight them, but poverty soon besets the dependents of those killed or maimed in the conflict. Destitution may destroy decency in the struggle to stay alive just one more day. Manhattan's diarist George Templeton Strong recorded the following on November 26, 1864:

> . . . the harlotry of the city is largely reinforced by Southern refugee women who were of good social standing at home, but find themselves here without means of support and forced to choose between starving and whoring. . . . It seems a just retribution on the Southern slaveholding chivalry.[10]

It was bad enough that a respectable woman, in the wake of war, should sell herself to be able to feed herself, but it was worse that so principled and conscientious a man as Strong, Trustee of Columbia University and Vestryman of Trinity Parish, should consider this just. Soldiers of occupying armies often comment on the cheapness of the local women—a pack of cigarettes, a chocolate bar, or a can of food were payment enough. Are these women cheap whores or wounded?

From Appomattox until the 1870s, few white Southerners were permitted to vote, to hold office, or to practice law. Newly enfranchised slaves and carpetbaggers, whatever their merits, rendered less than impartial justice. The police and courts were a shambles, and using this disorder as an excuse, the Ku Klux Klan took matters into its own hooded and masked control. The lynchings and floggings that began during the 1860s and 1870s did not finally end until the 1940s. That a proud people, heir to six centuries of the common law, should embrace lynch law was an unanticipated wound, but one that still bleeds.

On Memorial Day 1984, an unknown "serviceperson" of the Vietnam War was buried at Arlington National Cemetery amid simple republican pomp. It was hoped that this symbol would heal a wound caused when the United States sent an army of poor boys—Hispanics, blacks, Appalachians—who were jobless and could not afford college to the other side of the world to fight a war that some university professors thought could be "limited." When it appeared that the draft might reach into college dormitories, the United States abandoned them in the most anti-proletarian act in American history. The then protestors and temporary Canadian residents flourish, smug in their superior virtue: Congress granted them amnesty. It is not clear that the American people have, or that this wound will heal in our lifetimes.

Shelby Foote wrote of the men of military age, roughly 15 to 50 years old, during the Civil War:

> Approximately one out of ten able-bodied Northerners was dead or incapacitated, while for the South it was one out of four, including her noncombatant Negroes. Some notion of the drain this represented, as well as of the poverty the surrendered men came home to, was shown by the fact that during the first year of peace the state of Mississippi allotted a solid fifth of its revenues for the purchase of artificial arms and legs for its returning veterans.[11]

Some of these lives were destroyed or blighted by permanent incapacity. But dead men grow nothing, beget nothing, plant nothing, compose nothing, build nothing, write nothing, invent nothing, heal nothing, and discover nothing. Not until after World War II did the South recover from the psychological and economic wound of having lost a quarter of its able-bodied men. It took Germany a century to recover its population after the slaughter of the Thirty Years' War. Losses of this magnitude create shortages of people, because so many boys die before they can sire sons and daughters. What about the women who found no husbands because there were no men?

CODA

If one departs Manhattan and visits other regions of the United States, if one goes, so to speak, to where people drink beer rather than Perrier water, one finds almost universal male satisfaction at having served in the armed forces or been in a war. In a sense, military service is a masculine rite of passage. A man who has missed the armed forces is thought, somehow, to be an incomplete man. Little boys still play soldier, and, if "Star Wars" rather than Bull Run, it is still war. Many of these ordinary men look back on their soldiering with pride—never were they more alive, more themselves, more fully involved, even if they suppress some of their memories.

This is probably what William James had in mind when he deplored our lack of a moral equivalent of war. We cannot be sure that our persisting inability to find this moral equivalent of war is not the gravest wound of all.

REFERENCES

1. Harmuth E: Belt spanners for crossbows, in Held R (ed), *Art, Arms and Armour: An International Anthology* Vol I, 1979–1980. Chiasso, Switzerland, Acquafresca Editrice, 1979, pp 100–107.
2. Paré A: *The Apologie and Treatise.* London, Falcon Educational Books, 1951, pp 21–22.
3. Sharpe WD: The Confederate states medical and surgical journal, 1864–1865. *Bull NY Acad Med* 1976; 52:373–418.
4. Chisolm JJ: *A Manual of Military Surgery*, 3rd ed. Columbia, Evans and Cogswell, 1864, pp 119, 120, 358.
5. Cunningham HH: *Field Medical Services at the Battles of Manassas.* Athens, University of Georgia Press, 1968, p 18.
6. Ibid., pp 87, 90.
7. Chisolm, op. cit., pp 213–214.
8. Ibid., p 305.
9. Ibid., pp 359–360.
10. Nevins A, Thomas MH (eds): *The Diary of George Templeton Strong.* New York, Octagon Books, 1974, vol 3, p 521.
11. Foote S: *The Civil War: A Narrative. Vol 3: Red River to Appomattox.* New York, Random House, 1974, pp 1040–1041.

FROM THE LIBRARY

ATOM-BOMB-PROOF HOSPITAL

The first underground atomic-bomb-proof hospital soon will be in operation in West Germany. The hospital has been constructed in Neuhaus am Schliersee in the district of Oberbayern. The building was erected nine feet underground and the walls of the building serve as a foundation for a convalescent home built on top of the hospital structure. The underground compound has two operating rooms and a number of rooms for patients. Fresh air is supplied through a combination of filtered ducts and an artesian well serves as a source for fresh water. This hospital is supposed to be the prototype of approximately 120 others which are planned for the future. (*Du und Deine Gesundheit*, No. 10, 1967).

(*From the International Comments section of* JAMA *1968; 203:245*)

Violence in psychiatric institutions

STEVEN E. KATZ, MD, ROBERT COHEN, PHD, CORNELIUS L. J. STOKMAN, PHD

In the field of mental health, attention has been given to determining the extent of assaultive or violent behavior in institutions, understanding the environmental and patient characteristics that lead to and precipitate violence, and investigating effective approaches for preventing, reducing, and managing violent behavior. While this paper focuses on an understanding of the nature and dynamics of violent behavior in institutions, it also recommends programs that might be useful for physicians working in institutional settings. Although the data used relate primarily to psychiatric hospitals, it is reasonable to assume that some of the information and findings may also be applicable to other institutional settings.

VARIABILITY IN REPORTING

Violence in institutions can be defined as physically aggressive behavior directed toward patients or staff. The most frequent assaultive incidents are hitting of other individuals and throwing objects; however, self-destructive behaviors occur and include head banging, swallowing objects, and self-inflicted cuts.

What do we know about the extent of violence in institutions? At least one study indicates that assaultive behavior occurs far more frequently than is reported. An examination of daily ward records at a state hospital in Maryland revealed that only 203 assaults were formally reported, compared to an estimated 1,100 actual assaults during the year.[1] Although most assaults involve fighting between patients,[2] several studies found that at least 40% of psychiatrists and psychiatric residents have been assaulted at least once.[3-5]

In a study in New York State, institutional staff reported that approximately 2,000 patients, or roughly 9% of the total patient population, displayed assaultive behavior during a one-month period.[6] These data are subject to various interpretations. One might conclude that nine out of every 10 patients with psychiatric problems had not displayed assaultive behavior during this period. On the other hand, there is a significant amount of assaultive behavior in the state system.

Conclusions about the rate of violent behavior in institutions should be tempered by two factors. First, incident reporting—the major source of information on assaultiveness—is a subjective measure, since it is dependent on staff assessment of behavior. Differing perceptions of assaultiveness lead to variability in reporting, and, hence, diminished reliability of reported data. The second factor involves the role of culture. In several countries, including Sweden and Great Britain, there is considerably less assaultive behavior in psychiatric facilities than there is in the United States. In a retrospective survey of mental hospitals in Sweden, only 34 inpatients were reported to have participated in assaultive incidents over a 10-year period.[7] In Britain, during a three and a half year period, 311 violent assaults on staff were reported throughout the entire psychiatric system.[8] These results, even taking into account differences in perception and reporting of assaultive behavior, demonstrate that the propensity for violent behavior in institutions is influenced by culture. Assaultive behavior is, at least in part, a learned response, sanctioned by cultural and social values. Schizophrenia, for example, is a disease that has behavioral and cognitive cultural manifestations. It is not surprising that in the United States, where violence is so prevalent, assaultive behavior is an expression of serious mental illness.

MYTHS OF DANGEROUSNESS

Although the public perceives that mental illness, dangerousness, and violence are related, research evidence does not bear out this perception. Recent studies have shown higher arrest rates for patients with psychiatric illness, particularly for violent crimes.[9-11] However, the inflated rates of arrest among this population are attributable to a subgroup of individuals with psychiatric illness who have prior arrest and conviction records. Patients without criminal records actually have equal or lower arrest rates after being released from an institution.[10,12] In addition, for similar criminal offenses, patients with a history of psychiatric problems are more likely to be arrested than other offenders.[13]

In recent years, the arrest rates of patients discharged from psychiatric hospitals have increased. This rise may be attributable to two factors: an increase in the number of patients with psychiatric illness who have arrest records preceding their admission to psychiatric hospitals; and increased numbers of younger persons being admitted and discharged from psychiatric hospitals (criminal behavior and arrest rates are generally higher among younger persons). Young adult chronic psychiatric patients pose one of the greatest problems for psychiatry today. They avoid treatment and institutions; are prone to violence, alcohol, and drug abuse; and manifest repeated episodes of antisocial behavior. Mentally retarded patients who are placed in psychiatric inpatient facilities also pose problems regarding assaultive behavior. This group is not necessarily more violent; however, they become victims of provocative behavior by other patients, and staff may not be sufficiently trained to deal with this population.

A criminal record is not the only factor associated with violent behavior among the mentally ill. In spite of the

---

Based on a presentation at the Symposium on Violence, Annual CME Assembly, Medical Society of the State of New York, New York City, June 30, 1984.

From the New York State Office of Mental Health, Albany. Dr Stokman is Director of Research, Bureau of Forensic Services. Dr Cohen, formerly Director of the Bureau of Forensic Services, is Director, Virginia Treatment Center for Children, Richmond.

Address correspondence to Dr Katz, Commissioner, New York State Office of Mental Health, 44 Holland Ave, Albany, NY 12229.

public's perception of mentally ill offenders as extremely dangerous, clinical assessment—based in part on the incidence of assaultive behavior in hospitals—indicates that persons judged not guilty by reason of insanity are generally less dangerous in the hospital, present few management problems, and have low rates of violent behavior.[14] The importance of environment as a determinant of violent behavior is demonstrated by this population, which is clearly perceived as dangerous in the community, but has manifested relatively little violent behavior in the institutional setting. These studies, along with the cross-cultural comparisons cited above, indicate that environment clearly influences the expression of violent behavior.

PREDICTORS OF VIOLENCE

The literature on predicting dangerousness and accompanying manifestations of violent behavior is complex and sometimes confusing. One study reported that manic patients, especially in the early stages of their illness, are more likely to be assaultive.[15] A one-year study of psychiatric inpatients found that schizophrenic men were significantly more assaultive than patients in other diagnostic categories,[16] whereas another research project reported that schizophrenic patients had a lower than expected rate of violent behavior.[17] Some studies have concluded that abusers of alcohol tend to be assaultive, whereas other researchers have found little tendency for this subpopulation to become assaultive.[16]

Most researchers conclude that any assessment or prediction of violence must be linked to specific diagnostic subgroups. However, there is no consensus on which diagnostic groups are most likely to be associated with violent behavior in the institution. Furthermore, a distinction should be drawn between short-term and long-term predictions. Attempts at predicting violent behavior beyond the immediate future have generally not been successful. However, short-term predictions, during the course of several hours or days, have fared better. Using diagnostic, clinical, and behavioral indicators, it is often possible to anticipate assaultive episodes. For example, patients who are manic, withdrawn, or delusional are at high risk. Likewise, increased motor activity, raised voice, and significant postural shifts may indicate that a person is likely to become assaultive.[18]

It should be noted that violence in institutions is not limited to patients. While the majority of violent incidents are initiated by patients, there is evidence that a small number of staff members engages in abusive behavior.

CONTEXTUAL VARIABLES

A review of the growing body of literature on violence leads one to conclude that violent behavior in institutions can only be understood through a framework that takes into account the interactions of organismal and environmental variables. Adapting Zubin's vulnerability model for schizophrenia,[19] Stokman has developed a conceptual framework with the following elements:

1. Any person has the potential for violent behavior, and immediate environmental conditions can be sufficiently provoking to elicit violence in any person.
2. The degree of vulnerability to internal and external stimuli determines a person's threshold for violence. When the threshold is reached, a person will react in a violent manner.
3. Vulnerability to violent behavior—dangerousness—consists of two distinct but interdependent phases: a relatively permanent state of vulnerability, reflecting the long-term effects of biological and psychosocial stressors; and periodic episodes of increased vulnerability, reflecting the relatively short-term effects of contemporaneous stressors.
4. While both organismal and environmental variables are necessary, they are not sufficient by themselves to elicit violent behavior. The contextual contingencies trigger violence in a dangerous person and these contingencies determine the likelihood, type, and severity of a violent act in a particular situation.
5. The periodic episodes and contextual triggers are more directly related to the occurrence of violence than long-term vulnerabilities.[20]

To understand violence in institutional settings, therefore, it is important to have adequate knowledge of patient characteristics as well as contextual variables related to the environment. In terms of patient characteristics, several factors are being explored in regard to diagnosis, perceived assessment of dangerousness, and legal status. However, it appears that the best predictor for violent behavior in the long term is a history of past repetitive violent or dangerous behavior.

There are several contextual variables that may contribute to increased incidence of violence in institutional settings. Stokman reported that patients appear to be most vulnerable to violent incidents immediately after admission and when important clinical decisions are being made regarding transfer, discharge, or return to court.[17] Incidents occur most frequently during mealtimes and are more likely to occur in the dining room. Other researchers have suggested that negative staff expectations for patient change, staffing levels, the degree of activity on the ward, and submission-dominance relationships between patients are also possible contributing factors to increased rates of violent behavior.[21] Finally, crowding and increased density have been used as explanations for high incident rates, but evidence in this area is not conclusive.[22]

PREVENTION STRATEGIES

Given the complex array of factors apparently involved in producing assaultive behavior and the absence of definitive conclusions in research studies on this subject, caution must be used in moving from conceptual understanding to clinical applications designed to control violent behavior. However, the magnitude of the problem, as well as the growing body of knowledge emerging from empirical studies, dictate that serious attention be given to developing practical means of intervention to prevent, reduce, and manage institutional violence. Approaches to dealing with assaultive behavior may be divided into two categories: system approaches and individual interventions.

The significant role that contextual variables play in triggering violent behavior suggests the need to examine the environmental demands of the institutional setting in order to determine the major sources of stress. Once these stressors are identified, efforts should be made to modify the environment to reduce the level of stress or to assist patients in coping with particularly difficult situations. For example, knowing that the time immediately following admission increases vulnerability to violent behavior should be a guide in tailoring environmental conditions to minimize unnecessary sources of stress. Likewise, information about increased rates of incidents during mealtimes should guide decisions about staffing patterns during these periods.

Patient grouping is also an important consideration. The concept of a secure ward has merit. In practice, however, the lack of adequate physical space, programming, and staff training often mitigates against effective treatment. Furthermore, staff "burn-out" is a significant problem on secure wards.

At another level, greater attention should be given to staff training, designed specifically to make staff members more aware of precipitating factors, early warning signs, and therapeutic approaches to managing assaultive behavior. The attitudes—both negative and positive—and behavior of staff contribute significantly to the incidence of assaultive behavior.[23] Violent behavior is most likely to occur when patients sense that staff members are frightened or not in control. Staff members tend to be more cautious and tentative when they do not know the patient well (for example, following admission) and may overreact to minor outbursts, thereby creating more serious incidents, which can lead to assaults and subsequent restraint or seclusion. In addition, provocative and authoritarian behavior, indifferent and superior attitudes, and feelings of inadequacy on the part of the staff in the clinical setting all increase the likelihood of violent outbursts.

One of the most difficult policy questions is whether predictive indicators of dangerousness should be used to commit individuals on an emergency basis. The conflict between preserving individual liberties and protecting the community is particularly controversial and difficult when decisions are based on factors other than actual acts of violent behavior. Furthermore, it has been found that patients who are committed on an emergency basis pose a great risk to other staff and patients until their illnesses have been properly diagnosed, treated, and brought under control.[24]

INTERVENTION

Programs should be designed to prevent and reduce modifiable stress and to assist individual patients in managing anxiety and stress, particularly by helping them to develop alternative response patterns to stressful situations. Staff should be well trained to recognize and respond quickly and appropriately to the warning signs of impending violence. In-service training, whenever possible, should help the clinical team to coalesce into a cohesive unit which can act in a calm manner, is secure in its role, and is in control of any situation. What the patient fears most is his or her impending loss of control. All interventions should attempt to alleviate this fear. Violence can further be minimized by utilizing techniques such as desensitization of staff and patients, debriefing following incidents, and instruction in the appropriate use of isolation. Development of social skills and behavior modification efforts based on modeling appropriate behaviors and providing reinforcement have shown promise for reducing assaultive behavior.[25] Finally, every attempt should be made to ensure that the physical milieu of the ward discourages violent behavior.

Even with adequate clinical programming it is inevitable that patients will, at times, become agitated and assaultive. When psychologic and pharmacologic interventions have not been sufficient, appropriate use of seclusion and restraint may be required. In these instances, intensive staff training, non-abusive management techniques, and adequate professional supervision and monitoring are essential. Mechanical restraints should only be used in situations that demand intensive treatment, and not as a routine or "preventive" measure. As Soloff points out, "the use of mechanical restraints for less than intensive care is symptomatic of subjective distortions in patient-staff relationships."[26]

As the demand for protective services increases, particularly due to the growing population of young adults with serious mental illnesses, we can expect continued high levels of assaultive behavior in psychiatric institutions. By recognizing that the expression of violence is a behavioral and cultural manifestation of coping with vulnerability and stress, it is possible to design and implement constructive approaches to preventing, treating, and managing violent behavior in institutional settings.

REFERENCES

1. Lion JR, Snyder W, Merrill GL: Underreporting of assaults on staff in a state hospital. *Hosp Community Psychiatry* 1981; 32:497-498.
2. Incident Report Study, New York State Office of Mental Health, Bureau of Evaluation and Research, June 12, 1984.
3. Madden DJ, Lion JR, Penna MW: Assault on psychiatrists by patients. *Am J Psychiatry* 1976; 133:422-425.
4. Tardiff K, Maurice WL: The care of violent patients by psychiatrists: A tale of two cities. *Can Psychiatr Assoc J* 1977; 22:83-86.
5. Ruben I, Wolkon G, Yamamoto J: Physician attacks on psychiatric residents by patients. *J Nerv Ment Dis* 1980; 168:243-245.
6. Level of Care Study: 1982. New York State Office of Mental Health, Bureau of Evaluation and Research, June 12, 1984.
7. Ekblom B: *Acts of Violence by Patients in Mental Hospitals.* Stockholm, Scandanavian University Press, 1970.
8. COHSE. *The Management of Violent or Potentially Violent Patients.* Banstead, Surrey, England. Confederation of Health Service Employees, 1977.
9. Rappaport JR, Lassen G: Dangerousness: arrest rate comparison of discharged patients and the general population. *Am J Psychiatry* 1965; 121:776-783.
10. Steadman HJ, Cocozza JJ, Melick ME: Explaining the increased arrest rate among mental patients: the changing clientele of state hospitals. *Am J Psychiatry* 1978; 135:816-820.
11. Zitrin A, Hardesty AS, Burdock EI, Drossman AK: Crime and violence among mental patients. *Am J Psychiatry* 1976; 133:142-149.
12. Rabkin JG: Criminal behavior of discharged mental patients: a critical appraisal of the research. *Psychol Bull* 1979; 86:1-27.
13. Teplin LA: Criminalizing mental disorder: the comparative arrest rate of the mentally ill. *American Psychologist* 1984; 39:794-804.
14. Stokman CLJ, Smith HE: Characteristics of criminal patients. Paper presented at the Twentieth French Congress of Criminology, Lille, France, October 17, 1981.
15. Ionno JA: Prospective study of assaultive behavior in female psychiatric inpatients, in Lion JR, Reid WH (eds): *Assaults within Psychiatric Facilities.* New York, Grune and Stratton, 1983, pp 71-80.
16. Craig TJ: An epidemiological study of problems associated with violence among psychiatric inpatients. *Am J Psychiatry* 1982; 139:10:1262-1266.
17. Stokman CLJ, Heiber P: Incidents in hospitalized forensic patients. *Victimology: An International Journal* 1980; 5:175-192.
18. Dubin WR, Stolberg R: *Emergency Psychiatry for the House Officer.* New York, SP Medical and Scientific Books, 1981.
19. Zubin J, Spring B: Vulnerability: a new view of schizophrenia, *J Abnorm Psychol* 1977; 86:103-126.
20. Stokman CLJ: Dangerousness and violence in hospitalized mentally ill offenders. *Psychiatr Q* 1984; 56: 138-143.
21. Depp FC: Assaults in a public mental hospital, in Lion JR, Reid WH (eds): *Assaults within Psychiatric Facilities.* New York, Grune and Stratton, 1983, pp 21-45.
22. Megargee EI: The association of population density, reduced space and uncomfortable temperatures with misconduct in a prison community. *Am J Community Psychol* 1977; 5:289-298.
23. Lion JR, Reid WH (eds): *Assaults within Psychiatric Facilities.* New York, Grune and Stratton, 1983.
24. Rofman ES, Askinazi C, Fant E: The prediction of dangerous behavior in emergency civil commitment. *Am J Psychiatry* 1980; 137:1061-1064.

The obligation to assess dangerousness

DANIEL W. SCHWARTZ, MD

On December 6, 1979, in Mineola, New York, 42-year-old Adam Berwid murdered his former wife Ewa Berwid, while he was on a pass from a New York State psychiatric hospital. The District Attorney attempted to bring criminal charges of manslaughter in the second degree (based on the legal concept of recklessness) or the lesser charge of criminally negligent homicide (negligence being a lesser degree of culpability than recklessness) against the two psychiatrists who had authorized Berwid's pass that day.* Although the grand jury voted not to indict the psychiatrists who were treating the patient, it did make recommendations for changes in procedure before the release of "potentially dangerous individuals." As a result, various agencies, including the District Attorney's office, now must be given written notice in advance of the proposed release of an incapacitated person.

The Berwid case, which raised the question of the failure of a psychiatrist in a custodial setting to take proper steps in assessing the dangerousness of a patient, and in undertaking to warn the potential victim, forces consideration of the so-called "Tarasoff rule":

> When a therapist determines, or pursuant to the standards of his profession should determine, that his patient presents a serious danger of violence to another, he incurs an obligation to use reasonable care to protect the intended victim against such danger.[1]

This concept originated in 1974 with a decision by California's highest court known as "Tarasoff I."[2] Two years later that same court issued an amended decision, known as "Tarasoff II,"[3] which absolved the police from any such responsibility. As far as psychotherapists were concerned, however, the triable issue remained the same.

THE TARASOFF CASE

On October 27, 1969, Prosenjit Poddar, an Indian graduate student at the University of California killed Tatiana Tarasoff, an undergraduate student. Poddar and Miss Tarasoff met in the fall of 1968 while both were attending weekly folk dance classes at the University of California at Berkeley. He fell in love with her and misinterpreted her New Year's Eve kiss as the recognition of a serious relationship. When Miss Tarasoff learned of his erroneous belief and informed him that the feelings were not mutual, Poddar became severely depressed. He remained by himself, wept often, and neglected his appearance, his studies, and his health. When he and Miss Tarasoff met occasionally during this period, Poddar tape-recorded various of their conversations in an attempt to ascertain why she did not love him.

In the spring of 1969 Miss Tarasoff left for South America. Following her departure Poddar began to improve, and in early June 1969 he began outpatient psychotherapy at the university's mental health clinic. On August 18 his therapist, a psychologist, interpreted from Poddar's remarks that he would kill Miss Tarasoff when she returned from Brazil. Two days later, after consulting with two psychiatrists at the university, his therapist attempted to have Poddar confined to a hospital by notifying the campus police that the patient was suffering from paranoid schizophrenia and represented a danger.

The campus police detained Poddar. When he appeared rational to them, they themselves released him on his promise to stay away from Miss Tarasoff. The director of the Department of Psychiatry, upon learning of what had occurred, allegedly ordered all records on the case destroyed and "ordered that no actions leading to Poddar's detention be taken."[4] No one warned Miss Tarasoff or her parents, and soon after her return from South America she was killed.

At his trial Poddar attempted to have the charge of murder reduced to manslaughter, on the grounds that (because of his paranoid schizophrenia) he was not capable of mature and meaningful reflection on the nature of his act before the killing. Despite the testimony of three psychiatrists, the treating psychologist, and a neurologist (who found evidence of organic brain abnormalities), the jury agreed with the court-appointed psychiatrist who testified that Poddar was "merely schizoid and could harbor the requisite mental states of first or second degree murder." The Court of Appeal reduced the verdict to manslaughter,[5] but in February 1974 the Supreme Court of California reversed the conviction.[6] Upon return to trial court, Poddar pleaded guilty to reduced charges and was sentenced to probation, with the condition that he leave the country. He thereupon returned to India, where he is reportedly healthy, married, and employed.[7]

THE QUESTION OF NEGLIGENCE

Miss Tarasoff's parents brought a civil suit against the police and the Regents of the University of California. On July 1, 1976, in a pre-trial decision on what issues were triable, the Supreme Court of California clearly set forth the responsibility of the therapist:

> to use reasonable care to protect the intended victim . . . The discharge of this duty . . . may call for him to warn the intended victim or others likely to apprise the victim of the danger, to notify the police, or to take whatever other steps are reasonably necessary under the circumstances.[8]

The court reasoned that, although as a general rule one person had no duty to control the conduct of another, there were exceptions, including "the special relationship that arises between a patient and his doctor or psychotherapist."[9] The court cited other situations in which such a relationship has supported affirmative duties, even for the benefit of third persons. For instance, a doctor must warn a patient if, as a result of medication, it will be dangerous to others for him to drive a car. As another example, a doctor is liable to others infected by his patient if he negligently fails to diagnose a contagious disease or, having done so, fails to warn members of the patient's family.

In regard to the need for privileged communication between therapist and patient, the court pointed out that the need to prevent threatened danger to the patient himself or to the person or property of others was by law "a specific and limited exception to the psychotherapist-patient privilege . . . . The protective privilege ends where the public peril begins."[10]

It was this duty that a therapist "pursuant to the standards of his profession should determine, that his patient presents a serious danger of violence to another"[11] (and then warn the intended victim or others) that so alarmed and incensed the psychiatrists. The American Psychiatric Association has submitted an *amicus curiae* brief that therapists cannot accurately predict whether or not a patient will resort to violence. The court acknowledged that diffi-

---

Address correspondence to Dr Schwartz, Associate Professor, Department of Psychiatry, Downstate Medical Center, 450 Clarkson Ave, Brooklyn, NY 11203.

* Dr. Schwartz was one of two outside psychiatrists who were asked to provide an expert opinion on the Berwid case to the grand jury.

culty but pointed out that in this case the therapists had correctly predicted the killing and the only issue was whether they were "negligent in failing to warn."[12]

For physicians, one of the more disturbing aspects of the court's decision is that it dwelt heavily on the duty to warn, without explaining how such a warning would help. Had Miss Tarasoff's parents been notified of the danger, it is not clear what they could have legally done to save their daughter's life.

The court also stated that one of the ways in which the therapist may discharge his duty to "use reasonable care to protect"[13] that person is by notifying the police. In this case, while the therapist did just that, he unfortunately notified the wrong police. There were only two hospitals in Berkeley that were legally authorized to hold psychiatric patients against their will, and the University hospital was not one of them. On the other hand, the campus police were not permitted to take someone to a hospital off campus. Whether there was negligence on the part of the therapist for calling the wrong police or on the part of the campus police for not turning the matter over to the Berkeley police is an issue of fact that could have been argued in court.

The case never came to trial. Both sides agreed to an out-of-court settlement, with the amount sealed by court order. The decision established certain grounds upon which the family of the deceased could bring suit, including the allegation that "Tatiana's death proximately resulted from defendant's negligent failure to warn Tatiana or others likely to apprise her of her danger."[14] It would still remain for the plaintiffs to prove that such negligence had occurred.

Subsequent court decisions, in California and elsewhere, have not been as devastating to the psychiatric profession. In 1978, for instance, in *Bellah v Greenson*,[15] the California Court of Appeals refused to extend the Tarasoff decision to a case of suicide. A New Jersey Superior Court, in 1979, was the first court outside of California to adopt the Tarasoff rationale for holding a trial (*McIntosh v Milano*),[16] but in 1981 the psychiatrist was acquitted. In other jurisdictions, even where courts have held that a therapist had a duty to warn an intended victim, the therapist would not be negligent if he did not know, or have good reason to know, the identity of the intended victim.

CONCLUSION

The Tarasoff rule has not yet been applied in New York State. In the civil action of Ewa Berwid's estate against the State of New York, the plaintiff put this forth as one of the grounds for judgment, namely, that Berwid's therapists, pursuant to the standards of their profession, should have determined that Berwid presented a serious danger of violence to his former wife, and, therefore, had incurred an obligation to use reasonable care to protect her against such danger by not issuing a day pass. However, the court, in finding for the plaintiff, felt it unnecessary to apply the Tarasoff rule, because by the time the killing occurred, Berwid was considered to be an escapee, and his therapists were violating their duty to warn certain people if he escaped. (The court also found evidence of liability for the issuance of the day pass.)

The issue of the obligations of a therapist to determine whether his patient represented a serious danger of violence to another and then to use reasonable care to protect the intended victim against such danger remains a difficult and complex one. It could be argued that there has always existed a moral or ethical obligation to use such reasonable care. In any event, it is to be hoped that the final legislative or judicial decision will not result from an innocent person having been killed or assaulted, but rather from a psychiatrist having helped to prevent such a tragedy.

REFERENCES

1. *Tarasoff v Regents of University of California. Pacific Reporter, 2nd series* 1976; 551:340.
2. *Tarasoff v Regents of University of California. Pacific Reporter, 2nd series* 1974; 529:553.
3. 551 P.2d 334.
4. 551 P.2d 334, 352.
5. *People v Poddar. California Reporter* 1972; 103:84.
6. *People v Poddar. Pacific Reporter, 2nd series* 1974; 518:342.
7. Stone A: "The *Tarasoff* decisions: suing psychotherapists to safeguard society." *Harvard Law Review* 1976; 90:358.
8. 551 P.2d 334, 340.
9. 551 P.2d 334, 343.
10. 551 P.2d 334, 347.
11. 551 P.2d 334, 340.
12. 551 P.2d 334, 345.
13. 551 P.2d 334, 340.
14. 551 P.2d 334, 342.
15. *Bellah v Greenson. California Reporter* 1978; 146:535.
16. 168 N.J. Super. 466, 403 A.2d 500 (Law Div. 1979).

Violence on the highway

A national public health problem

JOHN D. STATES, MD

By 1965 more than 50,000 lives were lost each year on highways in the United States. These losses began to decrease in the early 1970s as federal motor vehicle safety standards went into effect. The oil shortage of October 1973, followed by an economic recession, brought about a precipitous drop in fatalities of nearly 20%. The effect lasted only three years, and fatalities increased again until 1979. A second oil shortage and another economic recession initiated a decrease that continues despite plentiful oil supplies and an economic recovery. The number of fatalities in 1983 was 42,500 in spite of a record number of miles driven by American motorists.[1] Notwithstanding these apparent successes, motor vehicle accidents remain the greatest single cause of death between ages one and 34.[2]*

REDUCING HIGHWAY FATALITIES

How can this violence on our highways be controlled and reduced? The major approaches include driver education, traffic control and enforcement, highway design, and vehicle design. Since 1965 vehicle design for improved occupant protection has been effective in reducing accidents. Driver education is a well-intentioned but ineffective means of controlling injuries resulting from traffic accidents. McGuire and Kersh[3] reported in 1967 that driver education courses were correlated only with high academic performance and good citizenship. When data samples were normalized for these factors, the effect of driver education on the students' subsequent accident rate disappeared. There is even evidence that driver education increases the exposure of teenagers to traffic accidents.[4] Overall, driver education does not reduce the accident rates of beginning drivers, but puts additional drivers on the highway at a younger age than would otherwise occur. Insurance companies have persisted in granting premium reductions to graduates of driver education courses on the assumption that the graduates are better than average drivers. Support for driver education through tax supported school systems has diminished in the past decade principally because of fiscal constraints.

Traffic control laws require enforcement to be effective. Speed limits are essential to maintain reasonable uniformity of traffic speeds and to prevent excessive speeding. Highway engineers used the 85th percentile rule for two decades prior to the oil shortage of 1973:[5] the rule recommended that speed limits be set at the 85th percentile speed, the speed under which 85% of motorists drove without other constraints. Speed limits determined by this rule have resulted in the lowest accident rates for the section of highways used in the determinations.[6] Traffic should move at close to the same speed for maximum safety. By 1973, statutory speed limits on Western interstate highways had risen to 70 mph, with enforcement limits at 80 mph. The 1973 oil shortage led to a national 55 mph speed limit for fuel conservation. Statistical analysis by the National Highway Traffic Safety Administration revealed that one-third of the fatality reduction in 1974, when the 55 mph limit was imposed, was brought about by the decreased speeds. The economic recession and improved vehicle occupant protection designs were responsible for the remaining two-thirds reduction in fatalities.[7]

The intensity of traffic law enforcement remains an ongoing controversy.[8] New York State has one of the lowest average speeds in the nation as revealed by federal surveys.[9] Experiments with more intensive enforcement have not decreased accident rates. It may be reasonable to conclude that enforcement levels in New York State are optimal, and that more intensive enforcement, at least of speed limits and traffic control laws, would not improve traffic safety. Connecticut's experience with increased enforcement in 1955–1956 revealed that accidents increased.[10] The fatality rates decreased in proportion to similar decreases in other states that had not modified their enforcement efforts. The one exception appears to be control of the intoxicated driver. Public outcry during the past five years has resulted in a significant increase in conviction rates for drivers who had been drinking and may be one of the major explanations for a continued decrease in highway fatalities in the 1980s.

Highway design for accident and injury control is highly effective but costly, and it can be detrimental to the environment. The death rate on limited access highways is half that of ordinary highways. The interstate highway system is partly responsible for the relative safety of the United States highways: death rates on a mileage basis are lower in the United States than in any other nation in the world.[11] The interstate system is now 92% complete. In 1962, six years after construction of the interstate system was begun, the public first rebelled against further sacrifices of land and living quarters, particularly in large metropolitan areas. Politically, expansion of the current highway system will not be acceptable to another generation.

VEHICLE DESIGN IS BEST APPROACH

Improved vehicle design for occupant protection remains the most hopeful avenue for reducing highway accidents.

Dr States has served as a consultant to General Motor's Biomedical Science Laboratory, Warren, MI since 1976.

Based on a presentation at the Symposium on Violence, Annual CME Assembly, Medical Society of the State of New York, New York City, June 30, 1984.

Address correspondence to Dr States, Chairman and Professor, Department of Orthopaedics, Rochester General Hospital, Doctor's Office Building, 1445 Portland Ave, Rochester, NY 14621.

* Motorcycles and trucks, which account for 10% of fatalities, are excluded from the scope of this discussion.

The scientific discoveries that are the basis for current occupant protection systems were begun by Hugh DeHaven,[12] an engineer and a fighter pilot in World War I who was the first to prove that safety belts were a highly effective means of occupant protection. His research was initially rejected by the United States government but then was reconsidered and resumed in 1936. This resulted in the installation of safety belts in all United States military planes in World War II and subsequently in automobiles. John P. Stapp, MD, initiated a large-scale research program in the United States Air Force following World War II. His studies established the basic principles for human occupant protection and became the basis for the restraint programs since 1961.

Preston Wade, MD, a trauma surgeon, and John Moore, a young engineer, established the Accident Crash Injury Research Program at Cornell Medical School in 1942. This accident investigation research study was moved to Buffalo, NY and expanded nationwide. The field accident studies provided the first evidence pursuasive enough for Ford Motor Company to introduce a safety equipment package that included safety belts on its 1956 models. This safety effort failed commercially but provided direction in safety research for the entire industry. Chrysler and Ford agreed to voluntary compliance in 1961, but General Motors refused to join these efforts stating that safety could not be sold and would be rejected by the public and stockholders.

The American Association for Automotive Medicine (Arlington Heights, IL), established in 1959 by a group of physicians, engineers, and other professionals interested in all aspects of traffic safety, brought about the preliminary agreements. In 1964 the association joined with Ralph Nader and supported the federal legislation that brought about the enactment of the National Traffic and Highway Safety Acts of 1966.[13] These acts established the National Highway and Traffic Safety Administration and the Federal Motor Vehicle Safety Standards. A physician, William Haddon, MD, was appointed the first administrator of the administration, then called the National Highway Safety Bureau, and promulgated the first standards.

These standards have become the most powerful means of improving vehicle safety design for occupant protection and injury control. Lap safety belts were required in all cars sold in the United States beginning in 1967. In 1968, shoulder harnesses were added for both outboard front seat occupants. Other standards cover windshields, dashboards, interior padding, steering wheel and column energy absorbing capability, head restraints, seat design, and side impact and roof protection. Safety belts, recognized as the single most effective safety device for the motorist, are now in virtually every vehicle in the United States; yet, usage rates are below 15% throughout the country (16% for 1983 in New York State).

Thirty-three other developed nations have achieved usage rates between 50% and 95% through educational campaigns and safety belt usage laws.[14,15] Educational campaigns alone have uniformly failed to raise safety belt usage rates above the 20% level. In 1984 New York State enacted the first safety belt usage law in the United States. It is expected that a usage rate of 50% to 55% will be achieved, similar to that of Ontario, which has succeeded in reducing road fatalities by 40% and serious injuries by 60% to 70%.[15]

PASSIVE RESTRAINTS

Passive restraints remain a major need in spite of safety belt use laws. The experience of similar population groups indicates that New York State can expect 45% of its motorists to go unrestrained. It is these motorists who will benefit from passive restraint systems.

The most well known passive restraint is the air bag. In 1969 Secretary of Transportation John Volpe recommended that they be installed in all cars sold in the United States. Development proceeded more slowly than anticipated and, currently air bags can be installed only in intermediate and large sized cars. The cost has escalated beyond expectation. Although the government contends that the cost will not exceed $200 to $300 per vehicle, industry sources estimate the cost at between $800 and $1800. The only available system is a steering wheel-air bag in the Mercedes Benz, and it costs $880. Cars equipped with air bags are more difficult to service, because an unsuspecting mechanic may short circuit the air bag and deploy it. Replacement costs are expected to exceed the initial cost. Operating a car with a deployed bag is far more dangerous than a standard car with the current padded dashboards.

Airbags and other passive restraint systems will be gradually introduced by 1989 as required by the new passive restraint standard (no. 208) issued in July 1984. If two-thirds of the population of the United States is covered by enforced safety belt use laws by September 1989, the standard will be rescinded.

In 1975, Volkswagen introduced an automatic safety belt system that consisted of a diagonal shoulder belt attached to the door and to an inertia reel on the seat. The car was not equipped with a lap belt but instead had a kneebar to catch the knees in head-on impact accidents, and a special seat designed to utilize the buttocks as energy input targets. The kneebar was constructed of a deformable metal core and staff plastic foam. This permitted knee impact without significant injury to the lower extremities. The lower seat cushion was a metal plastic pan filled with plastic foam. The forward edge of the pan was sloped upwards at a 45 degree angle, so that the buttocks went downward and forward and were restrained by the metal pan. The front end of the car was designed so that it did not pitch in head-on impacts. This allowed the occupant to jack-knife into the knee bar and seat cushion and not be pitched upwards into the windshield. An estimated two-thirds of the body weight, that weight below the diaphragm, was restrained by the knee bar and cushion alone. The shoulder belt would require only the restraining of the upper torso, neck, and head.

The initial system cost $30. Since 1977 it has been a part of an optional trim package at a cost of several hundred dollars. The system has proved to be highly effective. The Fatal Accident Reporting System of the United States Department of Transportation found that the death rate for Volkswagen Rabbits with the automatic belt system was half that of other 2,000-lb cars. One study revealed that the automatic system was as effective as the 3-point standard restraint system when used.[16] A study of nearly 300 accidents by Volkswagen of America revealed that the system performed well without the shoulder belt.[17,18]

Other manufacturers have developed similar systems.

General Motors manufactured 20,000 cars with passive restraints: 10,000 had a system similar to the Volkswagen method and 10,000 had a 3-point automatic belt system. Neither was a commercial success. The industry has indicated that approximately 80% of its production would be automatic belt systems if passive restraints were mandated by the federal government.

The system developed by Volkswagen revealed the possibilities of restraint without safety belts and without air bags. In 1978 I reported that passive restraints with static systems were possible, economically feasible, and likely to be acceptable to the public.[19] Only General Motors has developed such systems. The Vehicle Safety Improvement Program of General Motors was initiated in 1978, and the safety devices and design features are now found in current model Corvettes, Fieros, and C-body cars (Oldsmobile 98, Buick Park Avenue and Electra, Cadillac Seville). The Fieros meet the injury criteria (Head Injury Criteria of 1,000, maximum chest acceleration of 60 g, and maximum femoral knee load of 2,250 lb) of federal motor vehicle safety standard 208 in 30 mph 90 barrier impact without restraint systems in use.

School buses present a unique issue concerning safety belts. For the past decade an average of fewer than 25 fatalities have occurred annually within school buses in the United States. School buses are one of the safest modes of transportation for children. From an educational viewpoint, however, there is a need for safety belts in school buses. All states now require that preschool children be restrained while riding in passenger cars. These children should be able to continue this practice when they enter school and use school buses. Safety belt use is a vital part of a child's education and should become a life long habit.

Vehicle design to better protect the occupant promises significant reductions in traffic accident injuries and fatalities. Safety belts are the single most effective safety device available to the motorist. Safety belt usage laws are essential to achieve effective usage rates of 50% or more. Passive protection for the unrestrained motorist is essential for those who refuse to wear safety belts. Passive protection is provided by air bags and improved dashboards, windshields, seats, and front-end structure. Some of these designs are available in current model cars.

REFERENCES

1. Cerreli EZ: Early assessment of 1983 traffic fatalities. *Research Notes April 1984*. US Department of Transportation, National Highway and Traffic Safety Administration, 1984.
2. Baker SP, O'Neil B, Karpf RS: *The Injury Fact Book*. Lexington, MA, Lexington Books, DC Heath and Co, 1981.
3. McGuire FL, Kersh RC: *Study of History, Philosophy, Research, Methodology, and Effectiveness in the Field of Driver Education*. University of California Press, Publication in Education, Vol 19, 1969.
4. Robertson LS: Crash involvement in teenage drivers when driver education is eliminated from high school. *Am J Public Health* 1980; 70:599–604.
5. Elmberg CM, Michael HL: Effective speed limit signs on speed on suburban arterial streets. *Highway Research Board Bulletin* 1961; 303:1–9.
6. *Accidents on Main Rural Highways Related to Speed, Driver and Vehicle*. US Department of Commerce, Bureau of Public Roads, US Government Printing Office, 1964.
7. Mela DF: *Review of Information on the Safety Effects of the 55 mph Speed Limit in the US*. US Department of Transportation HS802-383, May, 1977.
8. Joscelyn JB: "Role of our legal system in influencing driver behavior." University of Michigan Highway Safety Research Institute 1976; 6:15.
9. *Quarterly Speed Summary*, Federal Highway Administration, October 81–March 82.
10. Campbell DT, Ross HL: The Connecticut crackdown on speeding. *Laws and Society Review* 1968; 3:33–53.
11. Trinca G: *Road Trauma—The National Epidemic*. Royal Australian College of Surgeons, 1983.
12. DeHaven H: *Beginnings of Crash Injury Research*. 13th STAPP Car Crash Conference, 1969, pp 422–428.
13. United States Public Law 89-563 and 89-564, 1966.
14. McKay GM: "Seat belts in Europe—their use and performance in collisions." International Symposium on Occupant Restraint. June 1–3, 1981, IL, American Association of Automotive Medicine, 1981, pp 39-54.
15. Lonero LP, Pierce J: "History and evaluation of seat belt legislation in Ontario." International Symposum on Occupant Restraint, June 1–3, IL, American Association of Automotive Medicine, 1981, pp 119–132.
16. Reinfurt DW, Chi GYH: "Automatic versus manual safety belt systems: a comparison using state accident data involving 1975–1979 model VW Rabbits, International Symposium on Occupant Restraint, June 1–3, 1981, IL, American Association of Automotive Medicine, 1981.
17. States JD, Rosenau W, Welkey GM: "Field performance of Volkswagen automatic restraint system." Proceedings of Experimental Research Safety Vehicle Meeting, Kyoto, Japan, November 1982.
18. States JD, Miller SR, Seiffert UW: "Volkswagen's passive seat belts/knee bolster restraint, VW RA: a preliminary field performance evaluation." Society of Automotive Engineers 770935.
19. States JD: Static passive occupant restraint systems without air bags and without belts/is it possible? *J Safety Res* 1978; 1010:108–114.

TRAGIC, COSTLY NECESSITY

Designing better cars to solve problems that the automobile has brought us is exactly like building more freeways to reduce traffic congestion. Thirty years of experience has shown us that *that* "solution" only intensifies the problem it is designed to solve.

Technology isn't evil, but the uses of technology often are. The car is a bad machine—and the solution is not to build the better bad machine, but rather not to build bad machines. Yet this huge, wealthy nation is trapped with what is virtually a single transportation system, and to suggest simply abandoning that system is to suggest paralyzing the nation. We have become addicted to automobiles; they have become literally a necessity for sustaining life.

JOHN JEROME
The Death of the Automobile
New York, Norton, 1972, pp 20–21

How the police view violence

PATRICK V. MURPHY

No subject is more central to the mission of the police than the control of violence. To a significant degree, controlling crime and maintaining order means containing and defusing violence, thus preventing injury and death. Many professional groups deal with violence: lawyers, physicians, psychologists, among others, know the toll and tragedy of violence and seek to deter it. But police usually are the first ones on the scene. They come to see themselves as the curbstone practitioners society pays to quell violence, because no group deals with more violent incidents.

The toll can be heavy. The most dispiriting part of police work is dealing with violence: the endless round of family fights, parents abusing children, grown children abusing elderly parents, neighbors battling one another, muggers bloodying the defenseless, predators committing sexual assault. In short, the daily repetition of persons injuring, maiming, violating, and killing each other can be psychologically numbing to the police officer.

If police officers seem hardened, cynical, or emotionally disengaged, it is important to remember that they see citizens both rich and poor at their worst—fighting, clawing, and injuring one another. Some police officers react by assuming a thinly concealed contempt for "civilians." But this trait is no more true for police than it is for physicians.

MYTHS ABOUT CRIME

Who are, disproportionately, the violent members of society with whom the police must deal? They have always been the poor. For much of the 19th century in New York and other cities, police traveled to the family fights, gang warfare, armed robberies, and similar violent events involving the Irish, when they dared to enter Irish neighborhoods. Somewhat later, the poorer Jews, Italians, and other immigrant groups were involved in violence of one sort or another, and the police were called. Now, many descendants of the 19th and early 20th century immigrant groups have prospered. The police seldom go to their middle class neighborhoods on calls involving violence.

But the cities still have many poor people, now mostly black and Hispanic. There should be compassion and understanding for the most frequent victims of violence in society—blacks and Hispanics today but, not too many years ago, the forebears of many who are now well-established in society. This goes against the views of many public figures who, directly or through code words or other forms of winking and nodding, suggest that the police should meet violence with bravado crackdowns and tough-guy tactics—the popular gut reactions to violent crime.

There seems to be a growing popular sentiment, voiced often by people who should know better, that the police have been handcuffed in dealing with violent crime; that if society removes those figurative handcuffs and looks the other way, the police could deter and markedly reduce violent crime by and among the poor. That has never been true, desite all the blather about the good old days when the police supposedly kept the peace by meting out nightstick justice. Nightstick justice did not curb violence among the Irish, the Italians, or the other poor immigrants to these shores. Jobs, education, the chance for advancement, and the other familiar components of American social progress lifted these groups out of poverty and, for each group, violence became less of a problem. Violence begets violence. If the police are encouraged to be violent in attempting to contain it, they will be met with more violence. This lesson was learned in the 1960s. An arbitrary, unrestrained policy of control did not lessen violence, as was learned the hard way amid that decade's frequent inner-city riots and disorders.

In contrast, the restrained use of force does not encourage crime. Studies on the use of deadly force suggest that, although police tend to use their weapons more in neighborhoods where the incidence of violent crime is high, limitations imposed on the use of deadly force do not lead to an increase in the rate of violent crime. On the contrary, a study conducted by Dr James Fyfe of the Police Foundation demonstrates that the number of crimes did not increase after the New York City Police Department introduced strict guidelines and monitoring procedures to reduce the number of police shootings in 1972.[1]

As a first step toward helping police deal with violence, society should put violent crime in perspective. Despite what the tabloids and television news shows seem to suggest, violent crime amounts to a relatively small portion of the national crime rate. Each year nearly 10 times more nonviolent property crimes than violent crimes are committed.[2] The rate of violent crime is about 500 incidents for each 100,000 persons.[2] For property crimes, the rate is 5,000 incidents for each 100,000 persons.[2] This comparison provides no consolation for victims of violent crimes, but it is important to understand who most of these victims are.

Many violent crimes involve people who know each other under circumstances which, although tragic, seem ripe for trouble. Drugs, alcohol, guns, and a history of child abuse are common factors in the profiles of violent crimes and those who commit them with frequency and predictability. In more than half of homicides, the offender, the victim, or both were drinking alcohol at the time of the crime.[3]

The national love affair with handguns contributes markedly to the fatal nature of our national violence rates. In the past stabbings were the common expression of rage. But at least with stabbings, fewer people died. There were not as many handguns to grab in the heat of violent attack.

More than half of the homicides committed in this

Mr Murphy served as Commissioner, New York City Police Department, from 1970 to 1973.

Based on a presentation at the Symposium on Violence, Annual CME Assembly, Medical Society of the State of New York, New York City, June 30, 1984.

Address correspondence to Mr Murphy, President, Police Foundation, 1001 22nd St NW, Suite 200, Washington, DC 20037.

country involve people who know each other—spouses, siblings, relatives, friends, neighbors.[4] The same is true for assaults, in fact even more so if one counts domestic assaults and incidents of child abuse. Of violent crimes among strangers, drugs, alcohol, and delinquency figure prominently. Those who frequently commit violent crimes—labeled violent predators—often commit assault, robbery, and drug dealing in tandem. Violent predators make up 10% of the criminal population, but commit more than twice the number of robberies and five times more assaults than the other 90%.[5] And, as noted, whether committed by acquaintances or strangers, violent crime is disproportionately prevalent in those urban communities where poverty already makes life difficult and uncertain.

COMMUNITY INVOLVEMENT

Given this profile of violent crime, what can be done to contain and control it? First, the police must be closely involved with the communities they serve, especially the poor inner-city communities with heavy concentrations of violent crime. The police cannot hope to control crime, violent or otherwise, if they do not know the people they are supposed to protect. Information from citizens is the lifeblood of policing. If the police do not know the clergy, the merchants, the teachers, the leaders, and other residents of a neighborhood, they will have little success in identifying the local situations and bad guys that are the sources of violence. The police must know and have the confidence of neighborhood informants; the term informant does not necessarily mean shadowy figures living on the edge of the law who trade information for a few dollars. Successful day-to-day, street-level police work is almost always done through the subtle use of neighborhood informants.

Fifteen or 20 years ago, in the big cities, mostly white police departments, operating with a heavy hand, were estranged from poor black and Hispanic neighborhoods. The police have made great strides in reducing that estrangement. It is significant that blacks are now the top police executives in New York City, Chicago, Houston, Detroit, Baltimore, Atlanta, Washington, DC, Newark, and several other large cities. Many police departments are far better integrated than before, and no longer are only white police officers patrolling minority neighborhoods. It is troubling and discouraging to observe the doubters of affirmative action who encourage rear-guard attempts to reverse the integration of police departments.

In addition, some big city police departments have made determined efforts to be less remote from citizens. The police today are far less likely to be encased in their patrol cars when not answering calls for service than they once were. It is worth noting that a Police Foundation experiment in Kansas City 10 years ago showed that routine police preventive patrols had no significant effect on crime rates or citizen fear of crime.[6] Today, police officers are working with citizens on such community crime prevention efforts as Neighborhood Watch. In heavily concentrated areas of some cities, police are returning to foot patrols as a way of becoming closer to citizens and helping to allay citizens' fear of crime. A Police Foundation experiment in Newark showed that although foot patrols did not reduce crime rates, they did reduce fear of crime which so debilitates neighborhoods and erodes the vitality of commercial areas.[7]

But, for all the progress in the big cities, much more needs to be done to weave police officers into the social fabric of communities, so that they can control crime more effectively. Although conventional wisdom has it that the courts are the weakest link in the criminal justice system, the truth is that the police link is the major problem. It is also the most important component of the system. The key to crime control is prevention, which is the primary function of the police. Prevention keeps more than 90% of the population out of the criminal justice system. It is essential that the police see every crime or delinquent act as a failure on their part to prevent it. Whenever prevention is superceded by enforcement, the "cart is before the horse."

The police prevent crime by obtaining and analyzing large quantities of information about crime, delinquency, ex-offenders, parolees, probationers, suspects, families, schools, particular locations, illegal drugs, criminal networks and confederates, and community resources. Patrol officers should use this information to prevent criminals—whether minor deliquents or repeat violent offenders—from advancing to more serious crimes. Their methods include both helping the transgressor to avoid future temptations and monitoring his behavior to increase the likelihood of detecting future misconduct and vigorously prosecuting it. Without the additional eyes and ears as well as the participation of citizens, the police would be impotent.

CONCLUSION

Smaller police departments are generally effective because officers are more a part of the community and receive better citizen cooperation. It is in the large cities that cooperation breaks down. The anonymity of urban life weakens neighborhood networks because so many of the police, the criminals, the victims, and the citizen participants in the crime control system do not live where they work.

The reduction of violence will come much more from improving the police force than improving the prosecution system, courts, or prisons.

The American police force is undergoing a dramatic change due to the shift from racial segregation to an integrated society. It is weathering the storm much better than the community has any right to expect. Citizens do not understand or appreciate the complexity of the task during such a critical transition. The continuing improvement of the police force will mean a decline in violence in the future.

REFERENCES

1. Fyfe JJ: Observations on police deadly force, in Fyfe JJ (ed): *Readings on Police Use of Deadly Force*, Washington, DC, Police Foundation, 1982, pp 299–300.

2. *Crime in the U.S.; Uniform Crime Reports, 1982.* Washington, DC, Department of Justice, Federal Bureau of Investigation.

3. Moore MH: Controlling crimogenic commodities: drugs, guns, and alcohol, in Wilson JQ (ed): *Crime and Public Policy.* San Francisco, ICS Press, 1984, p 127.

4. Rand MR, Klans PA, Taylor BM: The criminal event, in *Report to the Nation on Crime and Justice.* Washington, DC, Bureau of Justice Statistics, 1983, p 15.

5. Chaiken JM, Chaiken MR: Crime rates and the active criminal, in Wilson JQ (ed): *Crime and Public Policy*, San Francisco, ICS Press, 1984, p 25.

6. Kelling GL, Pate T, Dieckman D, Brown CE: *The Kansas City Preventive Patrol Experiment.* Washington, DC, Police Foundation, 1974.

7. *The Newark Foot Patrol Experiment.* Washington, DC, Police Foundation, 1981.

Violence in the Third World

Sister Helene O'Sullivan

In his book *Spiral of Violence* (Denville, NJ, Dimension Books, Inc, 1971), Archbishop Dom Helder Câmara, of Recife, Brazil, discusses injustices in Third World Countries that reduce millions of people to "sub-human" conditions." He refers to "the heritage of poverty" that kills and renders powerless as surely as war.

In comparison to western countries, the proportions of poverty and power in underdeveloped nations are reversed. For instance, whereas approximately 80% of the American population lives above the poverty level, in Latin America only 20% receive goods and services sufficient for a life of human dignity, and half of the rest of the population lives in misery. Catholic theologian Joseph Gemillion (*Puebla and Beyond*, Maryknoll, NY, Orbis Books, 1979) has estimated that only 10% of the people in Latin America has access to political participation and civil rights comparable to those in the United States.

In South Africa, the Philippines, and many countries in Latin America, to cite a few examples, the enormous disparity between rich and poor can be most strikingly seen as the result of interconnected economic, political, and social structures that benefit the few at the expense of the many.

Apartheid is a clear example of such a system. Although white citizens constitute only 16% of the South African population of 4.5 million, they own almost 90% of the country's land. With the recent establishment of black reservations, known as homelands, millions of black inhabitants are being deprived of South African citizenship. The black population has no political rights, and employment and educational opportunities are severely limited.

In the Philippines, 80% of the population lives in poverty. Although 55% of the agricultural land is used for growing cash crops for export—such as sugar, tobacco, bananas, and pineapples—70% of the population suffers from malnutrition.

In Guatemala, 2% of the population owns 64% of the land, in the form of large plantations and grazing land. Only 30% of the rural population is literate. In Bolivia, the health of the population is directly affected by their poverty. The infant mortality rate is the highest in Latin America with 166/1,000 children dying before the age of one year; 50% of the children die before the age of 10 years, most often from respiratory and gastrointestinal infections and measles. At 46 years, the life expectancy is the lowest in Latin America. Only 34% of the population has access to clean water, and 66% of the population has deficient caloric and protein intake. Thirty percent of children do not attend school.

Structural Violence

Archbishop Câmara believes that such disparities between rich and poor constitute a kind of "structural violence" which is the wellspring of subsequent overt violence. An escalation of violence occurs when oppressed groups, such as squatters, workers, peasants, and racial or ethnic minorities, organize to protest and change their situation. Protest could take the form of starting unions, demonstrating for clean water, or demanding the replacement of a corrupt official. If demands for change are repeatedly denied, ignored, or met with punishment, the demonstrations can turn into riots, and the spiral of violence can escalate into repression by the government.

When people take to the streets to protest the forms of structural violence that affect them the most, the authorities may consider themselves obliged to preserve or reestablish public order, even if this means using considerable force. If the public or opposition groups are thought to be organized, then the police or military intelligence agencies are ordered to quash "agitators" or "subversives." There may be kidnappings, beatings, murder, torture, and disappearances. In Argentina, before the recent change of government, repression led to protest which led to further repression. The violence spirals and in a few cases escalates into revolutionary violence or terrorism.

The Role of the Church

In 1968, three years after the closing of the Second Vatican Council, representatives of the 700 Latin American bishops met in Colombia to examine the role of the Church in the transformation of Latin America. Before this conference, the bishops had examined the reality of their individual countries. When they came together, they were dismayed by the enormity and similarity of the economic, political, and social problems that faced the whole of Latin America. In the conference's final documents, the bishops explored the various tensions in Latin America that threatened national and international peace, including the inequalities among social classes, the exclusion of the poor in urban and rural areas from participation in the civic and economic life of the society, the forms of oppression used by dominant groups, and the growing awareness of oppressed people.

In 1983 in their pastoral letter, the bishops of the Sudan addressed the question of the growing restiveness among the poor:

> [many people are] ill-fed, inhumanly housed, illiterate, destitute of essential services, unable to exercise any political power . . . . Since the underprivileged zones are fully aware of the well-being in other parts of the country, they begin to wonder why they are excluded from these benefits. Such inequalities become a threat to peace. One time these people or their children will ask why,

---

Based on a presentation at the Symposium on Violence, Annual CME Assembly, Medical Society of the State of New York, New York City, June 30, 1984. The socioeconomic data cited in this article were largely drawn from governmental and university reports from the countries themselves.

Sister O'Sullivan is a member of the Maryknoll Sisters, a Roman Catholic missionary congregation with 1,000 members in 25 countries in Africa, Asia, and Latin America. Two of the four churchwomen killed in El Salvador in 1980 were Maryknoll Sisters.

Address correspondence to Sister O'Sullivan, Maryknoll Sisters Center, Maryknoll, NY 10545.

> and there will be a disturbance of a peace—a peace that really did not exist in the first place.

Social justice is a prerequisite for true peace in any society. The Catholic Church has frequently called for an end to all forms of violence—structural, repressive, and terrorist—while working for a more just and humane society.

In 1983, the Guatemalan bishops bluntly pointed to the horror of the most blatant violence: torture. They wrote the following:

> The denouncements of the outrages and abuses against people are an integral part of the Church's mission. Whenever people are trampled upon, whenever their rights are violated, when flagrant injustices are committed against them, when they are subjected to torture, when they are constrained by abduction or when a person's right to live is violated, a crime and a grave offense against God is committed.

Almost all governments publicly condemn torture, but more than a third of the world's governments use or tolerate torture or ill-treatment of prisoners in the 1980s, according to Amnesty International, which cites allegations of torture in 98 countries.

ACTS OF TORTURE

The United Nations Declaration Against Torture, adopted in 1975, provides the following definition of torture:

> Any act by which severe pain or suffering, whether physical or mental, is intentionally inflicted by or at the instigation of a public official on a person for such purposes as obtaining from him or a third person information or confession, punishing him for an act he has committed or intimidating him or other persons.

Such a simple definition belies the sophistication and multiplicity of torture methods, the widespread use of which can be said to constitute a new form of international language. Acts of torture can be roughly grouped into physical and psychological methods, although there is considerable overlap between the two:

Physical

1. Trauma
   a. beatings, extraction of teeth, blows to the head
   b. "La Falanga": beating of the soles of the feet with a metal rod
   c. "El Telefono": blows to both ears with cupped hands
   d. extraction of fingernails; insertion of needles under nails
2. Electrical shocks
3. Suffocation
   a. "Wet submarine": repeated immersion of the head in water, to the point of near drowning
   b. "Dry submarine": covering of the head with a plastic bag to the point of near suffocation
   c. garrote: tightening a rope or wet towel slowly around the neck
4. Burning
   a. burns on sensitive parts with cigarettes
   b. burns with a hot iron
5. Suspension
   a. "El Planton": victim forced to stand with weighted arms outstretched for hours
   b. "The Hook": victim hoisted off the ground by the hands, which are tied behind the back, resulting in permanent nerve damage
6. Sexual abuse
7. Administration of drugs

Psychological

Night arrest; being kept blindfolded; prolonged solitary confinement in small, dark, filthy cells; overhearing other people being tortured; sleep deprivation; marathon interrogation; mock execution; the torture of spouses in front of each other; torture of children in front of parents and vice versa.

The overall result of episodes of torture is one of physical and psychological disintegration. Powerless to intervene in behalf of the victim, families suffer as well. As civil unrest increases around the world, the use of torture as a means of repression can be expected to increase. Emergency legislation that allows wide powers of arrest and detention may facilitate torture. Suspects can be held on vague suspicions; crimes against the state are given broad, elastic definitions.

THE MEDICAL PROFESSION'S RESPONSE

In view of the extent and methods of torture as a worldwide reality, there are several ways in which the medical profession in this country can take action against the torture of other human beings. In addition to moral, humanitarian, or religious motives for trying to put an end to torture, health care professionals have a powerful incentive to stop this suffering. A number of international statements provide precedent for taking such action:

1. The Universal Declaration of Human Rights, adopted by the United Nations in 1948 states that "no one shall be subjected to torture or to cruel, inhuman or degrading treatment or punishment."
2. The World Medical Association declared in Tokyo in 1975 that physicians must not take part in torture, and should be supported in this refusal by the international medical community.
3. In December 1982, the UN General Assembly adopted *Principles of Medical Ethics* relevant to the role of health care personnel, especially physicians, in actively protecting prisoners against torture, rather than merely refrainly from participation in torture. The principles state that physicians "charged with the medical care of prisoners and detainees have a duty to provide them with protection of their physical and mental health, and treatment of disease of the same quality and standard as is afforded to those who are not imprisoned or detained."

HOW CAN PHYSICIANS HELP?

First, physicians in the United States can speak out, via their professional societies (national, state, and speciality), in denunciation of the very existence of torture in the 20th century. This is being done in a number of countries and organizations around the world. Physicians in Syria, for example, have denounced the use of detainment and torture by the government—at great cost to themselves: doctors have been arrested, and some have disappeared. In 1983, as a result of the testimony of Dr Anatoly Koryagin and others, the Soviet Union was expelled from the World Psychiatric Association because of the continued use of psychiatric hospitals for torture. Similarly, a publication of the United Nations Centre Against Apartheid has called for the expulsion of South Africa from international psychiatry. Physicians in several countries have formed Amnesty International Medical groups, working within their own countries to publicize and take action against torture, and occasionally going to other countries as members of evaluation and fact-finding teams.

Second, a special obligation of the medical profession around the world is that of condemning the participation of physicians in acts of torture, a phenomenon that appears

PHYSICIANS DETAINED

On the morning of June 24, 1982, Dr Jose Hurtado, 56, a prominent pediatrician and medical educator in Guatemala, was seized by armed men wearing civilian clothes in front of his clinic in Guatemala City. One week after his arrest, Guatemalan President Rios Montt announced on national television that Dr Hurtado was a prisoner of the government and charged that "he is not merely a communist but a captain of many units, a member of the leadership. His son—all his family—have been killing people." Throughout his career, Dr Hurtado has shown interest in the health needs of the Guatemalan Indian population, and since 1976 he has been responsible for a rural medical clinic at which several hundred Indians receive care each month. American doctors who pursued his case in Guatemala with the government concluded that whereas no official raised this involvement as a reason for his detention, in the past there has been suspicion and even resentment among some in Guatemala of volunteer health programs for the Indian population. No evidence or suggestion of medical assistance to anti-government guerrilla elements was found in this clinic.

Because of Dr Hurtado's work with the American Friends Service Committee, his case became well known in the United States, and much pressure was brought for his release. He was freed after 40 days, during which time he had not been treated well, and he was allowed to leave the country.

The detention of Dr Neil Aggett did not end this way. Dr Aggett was a 28-year-old white physician, whose family had immigrated to South Africa from Kenya in the 1950s. At the time of his detainment by the South African government on November 27, 1981, he was staff physician at a hospital in Soweto, and was an active trade union official in a black union. He was arrested together with other black and white trade union officials, students, and political activists. He was imprisoned under Section 6 of the Terrorism Act, regularly used against political activists in South Africa. Dr Aggett was found hanged in his cell at Johannesburg Security Police headquarters on February 5, 1982, the first white dissident to die in custody in South Africa. An autopsy and inquest revealed evidence of physical and psychological torture, including beatings, genital abuse, electric shock, and severe psychological maltreatment. During the last week of his life he had spent 110 of 168 hours in the interrogation room and had been interrogated continuously for more than 60 hours. Witnesses who saw him during the last week described him as walking with great difficulty, tearful, and as one person testified, "he seemed to have disintegrated and he looked like a zombie." People who had known him prior to the detention, including two university psychology professors, described him as "highly motivated, well-integrated, relaxed, in control of himself," and said "he had showed no signs of suicidal tendencies in the past." Nonetheless, the coroner's verdict was suicide, and all allegations of torture were denied.

to be increasing. An important example of the refusal of physicians to cooperate in any way in such activity occurred in Northern Ireland in the 1970s, when police surgeons in Ulster were required to examine all prisoners at regular intervals. They began to notice a pattern of injuries among "political" detainees—members of both Catholic and Protestant extremist groups—and came to the conclusion that these injuries were a result of interrogation techniques. After a series of meetings with prison and police authorities, the police surgeons threatened, through their national medical organizations, to resign en masse and to publicize the existence of interrogation methods that fell within the definition of torture. A government commission substantiated the doctors' claims, and conditions for prisoners were markedly improved. In a recent publication, *Against Torture* (London, Amnesty International Publications, 1984), there is a 12-point program for the prevention of torture which includes a detailed comment on the role of medical officers.

A third avenue for action is that of exerting immediate international pressure when health care professionals themselves are victims of torture. For example, at least 100 health care professionals have been reported missing in El Salvador, and the situation in Guatemala may be more serious. Among the urgent action groups sponsored by Amnesty International, there is one specifically for health care personnel who are detained for political reasons, and who are victims or potential victims of torture. Telegrams, letters, telephone calls, and official statements of concern on the part of physicians' organizations and directed to governmental officials and members of Congress can be very effective in protecting imprisoned health care workers from physical or mental abuse (see accompanying story).

Fourth, American physicians can provide medical assistance to torture victims living in the United States. It must be recognized that many such persons are undocumented aliens and are not recognized as refugees from political repression; thus, their access to medical care is limited by language, poverty, and the pervasive fear of being deported back to their home country. The present administration's policy on political refugees is clear: those fleeing leftist governments (for example, Cuba) are offered refugee status; those fleeing rightist regimes (such as Haiti) are defined as economic refugees and are sent back. Thus, persons fleeing Poland, Cuba, or the Soviet Union are given asylum and assistance, while persons from Haiti, El Salvador, South Africa, or South Korea are illegal aliens. There are an estimated 125,000 refugees from El Salvador and Guatemala in New York State and, based on figures from other countries where such refugees are granted political asylum, perhaps half of these men, women, and children have been subjected to ill-treatment or torture. Many will also have been traumatized by seeing loved ones murdered, or will have found the mangled corpse of a friend or relative on the roadside or in the morgue.

An example of the health care profession's response to this need is the program begun in Denver in 1983, where a group of family practitioners at a teaching hospital enrolled more than 100 Salvadoran and Guatemalan refugees in the Family Practice Group Plan without regard to their ability to pay. There is an ecumenical organization (New York Circus, PO Box 37, Times Square Station, New York, NY 10108) in New York City that is attempting to set up a medical referral network for undocumented El Salvadorans and Guatemalans.

No single physician or medical society can be expected to undertake all of these actions against torture, but the important thing is for each person to do something.

HISTORY

The physician and war

A layman's view

ABRAHAM BLINDERMAN, PHD

> *I believe I must make a Phisician of you. There seems to be a Propriety in your studying Physick, because your Great Great Grandfather after whom you were named, was of that Profession. Would it not please you to study Nature, in all her wonderful Operations, and to relieve your Fellow Creatures under the severest Pains and Distresses to which human Nature is liable. Is not this better than to be destroying Mankind by Thousands? If you are of this Opinion, you will change your title from General to Doctor.—John Adams to Thomas B. Adams, March 16, 1777.**

Of the 156 physicians practicing in Hiroshima when the atomic bomb devastated that city, only 65 survived the blast. Of the 1,780 nurses assisting them, 1,654 either died or were too severely injured to continue ministering to the sick and wounded.[1] The democracy of contemporary technological warfare is startling; class, race, ideology, and geography offer no human being exemption from atomic incineration or maiming.

Warfare has obsessed mankind since the dawn of history. To rationalize wars, philosophers, historians, poets, composers, and artists have portrayed the battlefield romantically. So-called "just wars" have been given religious benediction; even Hitler ended all of his orations with an appeal to God to give Germany victory. Machiavelli cynically advised statesmen to make public speeches with assurances to the people that God was with them. Patriotism and religion have brought Plato, Aristotle, St. Thomas Aquinas, the famed educator Bruno D'Arresso, Martin Luther, Hegel, John Dewey, and even socialist Upton Sinclair to defend just wars. The problem is that both factions in a war protest that they are fighting a just war.

Educators, theologians, lawyers, and philosophers have failed to eliminate the sorry failings in men that ultimately lead them to the battlefield. Scientists and scholars, who insist on objectivity in their specific fields, often display puerile emotionalism in their political and social relationships. Learning has not endowed professionals with the will to direct science and technology into altruistic enterprises.

Those who swear by Hippocrates should be above the petty lip-service given to the betterment of humanity by sectarians who almost invariably discover rationalizations to support just wars. In his youth, John Adams gave up the impecunious and inglorious trade of teaching for the brighter prospects of law after considering "Divinity and Physick."[2] Medicine appealed to him, but after long meditation, he wrote to his friend Peter Chardon in January 1761: "Nothing less than the Preservation of Health and Properties of the Human Species, is the object and design of the Law."[3] A physician might save scores of lives in his lifetime, but a wise legislator might save tens of millions of people by instituting humane laws. In his later years Adams despaired of mankind's salvation through the offices of education, religion, and law:

> You may read the history of all the universities, academies, and monasteries of the world, and see whether education extinguishes passion or corrects human vice. You will find in them as many parties and factions, as much jealousy and envy, hatred, malice, revenge, and intrigue as you will find in any legislative assembly or executive council, the most ignorant city or village.[4]

Physicians are not exempt from the list of human vices mentioned by Adams, but of all the professionals serving mankind in this age of Damoclean terror, physicians, surgeons, and their associates should be foremost in the struggle to eliminate war in this dangerous age of nuclear technology. Those who dedicate themselves to the obliteration of disease have a special responsibility to preserve life. Although Desiderius of Rotterdam was not a physician, he held life to be sacred and bravely chastised those who advocated war as an arbiter of human conflict. In an age that punished heretics severely, Erasmus lashed out vehemently at clerics, professors, and generals who, in the name of nationalism, defended the wars of their respective nations. These words of Erasmus unfortunately are not anachronistic: "Every Angle hates the Gaul; and every Gaul, the Angle. The Irishman because he is an Irishman, hates the Briton; the Italian hates the German; the Swabian the Swiss; and so on through the list . . . Why do these stupid names do more to divide us than the common name of Christ to unite us?"[5]

Wars, which Socrates had denounced in Xenophon's *Memorabilia* as futile instruments of national policy, are

---

Address correspondence to Dr Blinderman, 47 Tanager Lane, Levittown, NY 11756.

* Butterfield LH, Garrett WD, Sprague M (eds): *Adams Family Correspondence*. Cambridge, MA, Belknap Press, 1963, vol 2, p 698.

fought by people "who neither caused them nor profited by them. Ordinary men—once converted into soldiers, become profligate wretches, cutthroats, and unprincipled villains."[6]

Scholars and teachers who subvert learning are abominations to Erasmus. Materialistic and servile, they advise kings to substitute warfare for arbitration. War is an evil "that pours upon Mankind the whole Collected Legion of all the miseries and misfortunes that can distress human nature."[7] Undistinguished in the cause of peace are university men: "Scholars wage war with scholars; . . . in the same university, the rhetorician is at odds with the logician, and the theologist with the lawyer. Alas, what good can a king derive from this scurvy lot of venomous, abusive, and libelous pedagogues?"[8] Erasmus, alone among his contemporary intellectuals, and perhaps among ours, refused to support even just wars, writing to Sigismund the First, King of Poland, that "they love war who never tried it . . . , but a man of penetration will prefer an unjust peace to a just war."[9] Two hundred years later, Benjamin Franklin paraphrased this unpopular shibboleth—"there never was a good war nor a bad peace," but Franklin repudiated his dictum when the colonies revolted against England. America's cause was just.

Statesmen find it difficult to square their idealism with the dictates of political necessity. As President of the United States, Woodrow Wilson was compelled by his analysis of historical imperative to declare war against the Central Powers. His grief was poignantly expressed to his friend Tumulty as they watched the hectic stampeding of Congressmen after his solemn reading of the war speech. "My message today was a message of death for our young men. How strange it seems to applaud that."[10] And John Dewey, the beloved philosopher of the liberals, so adamant in his stand against the impending war, changed his pacifistic tune to a more strident one once war was declared: "I have been a thorough and complete sympathizer with the part played by this country in this war, and I have wished to see the resources of this country used for its successful prosecution."[11]

It is distressing that great scientists frequently lack the social understanding of men like Socrates and Erasmus. Pierre Curie naively hypothesized on the altruism of those who exploit scientific discovery:

> One may . . . imagine that in criminal hands radium might become very dangerous, and here we may ask ourselves if humanity has anything to gain by learning the secrets of nature, if it is ripe enough to profit by them, or if this knowledge is not harmful. The example of Nobel's discovery is characteristic: powerful explosives have permitted men to perform admirable work. They are also a terrible means of destruction in the hands of great criminals who lead people to war. I am among those who think, with Nobel, that humanity will obtain more good than evil from new discoveries.[12]

Like Curie, Louis Pasteur had faith in man's ability to abstain from ruinous use of scientific knowledge. Today, many fear that Mary Shelley was right in *Frankenstein*—that men cannot restrain themselves from perverting science for their egotistic or material pleasure. Pasteur is more hopeful:

> Two opposing laws seem to me in contest. The one of blood and death, opening out each day new modes of destruction, forces nations to be always ready for battle. The other, a law of peace, work, and health, whose only aim is to deliver man from the calamities which beset him. The one seeks violent conquests, the other the relief of mankind. The one places a single life above victories, the other sacrifices thousands of lives. Which of the two laws will prevail, only God knows. But of this we may be sure, that science in obeying the laws of humanity will always labor to enlarge the frontier of life.[13]

Great men like Pasteur, Curie, Wilson, Dewey, and Plato are not unlike W. H. Auden's unknown citizen who "held proper opinions for the time of year." This faceless average man was robot-like in thinking: "When there was peace, he was for peace; when there was war, he went."[14] Emerson opposed such puerile, timid thinking. In his essay "The American Scholar," he called on the scholar to become a free man, assume political independence, and to participate in the conflicts that shook the nation:

> Free should the scholar be—free and brave . . . It is a shame to him if his tranquility, amid dangerous times, arises from the presumption that like children and women his is a protected class; or if he seeks a temporary peace by the diversion of his thoughts from politics or vexed questions, hiding his head like an ostrich in the flowering bushes, peering into microscopes, and turning rhymes, as a boy whistles to keep his courage up.[15]

Emerson's appeal to scholars to honor intellectual independence has for the most part been ignored by learned men who too often succumb to the seductions of patronage and politics. History does not record many instances of the courage of a Socrates or a Bruno. Fortunately, there are unique individuals who dare to attack cant and hypocrisy when other thinking men are silent. A 16th century physician, the earthy hyperbolist François Rabelais, lived in dangerous times. His friend, Etienne Dolet, a great humanist, was hanged and burned for publishing a dialogue attributed to Plato denying the immortality of the soul. And in the year of Rabelais' death, Calvin burned Servetus at the stake for heresy.[16] Rabelais incurred the hostility of clerics in his monumental fictional satire *Gargantua and Pantagruel*, but he managed to escape their wrath because of his stated belief in God and the existence of an afterworld. He depicted warfare as a cruel and needless way to settle international disputes. Reducing the causes of war between the kingdom of Lerne and King Grangousier's land to absurdity, the gracious Grangousier lectures the defeated hordes of Lerne:

> The day of conquering kingdoms to your Christian neighbor's loss is long since past. To imitate the ancient Herculeses, Alexanders, Hannibals, Scipios, Caesars, and that ilk is contrary to the profession of the Gospel. Does not Christ enjoin us each to preserve, rule, keep, and administer his own lands and possessions? Does he not forbid us to make hostile invasion upon others?[17]

Alas, Grangousier's idealism was still wanting in the 20th century when another physician, Sir William Osler, surveyed the madness of his age and denounced nationalism as a curse to mankind in forceful rhetoric:

> In no other shape has the Demon of Ignorance assumed more hideous proportions; to no other obsession do we yield ourselves more readily. For whom do the hosannas ring higher than for the successful butcher of ten thousand poor fellows who have been made to pass through the fire to this Moloch of nationalism? A vice of the blood, of the plasma rather, it runs riot in the race, and rages today as of yore in spite of the precepts of religion and the practice of democracy. Nor is there any hope of change; the pulpit is dumb, the press fans the flames, literature panders to it,

> and the people love to have it so.[18]

In a moving lecture to medical students of the University of London in 1877, Henry Maudsley, Professor of Medical Jurisprudence, spoke on the need for physicians to participate in the political affairs of the nations, emphasizing that preachers and moral philosophers had failed signally to better the lot of men.[19] Since statesmen had done nothing to stanch the deluge of blood on the soil of warring Eastern Europe, he proposed that physicians take a hand in shaping man's destiny, for what other professionals were better qualified to carry on the development of man's nature—moral, intellectual, and physical—to its highest reach?[20]

Appalled by what he had seen at Bikini, David Bradley, MD, recorded his pessimistic conclusions on atomic warfare in *No Place to Hide.* Bradley concludes that there is no real defense against atomic weapons, that "there are no real medical or sanitary safeguards for the people of atomized areas," and that the unknown devastating influence of the bomb may affect the land and its wealth for centuries through the persistence of radioactivity.[21] In *The Politics of Therapy*, Seymour L. Halleck, MD calls on physicians to make their lives meaningful by actively opposing atomic warfare.[22] Dr Robert Jay Layton, Professor of Psychiatry at Yale University and author of two books opposed to the use of nuclear weapons, lectures regularly at colleges on preventing nuclear disaster. Physicians for Social Responsibility, a Cambridge, MA based anti-nuclear weapons organization, includes many noted physicians on its National Board of Sponsors. Boasting a membership of more than 30,000, Physicians for Social Responsibility supports "a bilateral, verifiable, nuclear weapons freeze, and a Comprehensive Test Ban Treaty.[23]

Recognizing that a unilateral campaign to outlaw nuclear weapons would have little effect, 350 physicians representing 53 countries met early in June 1984, under the auspices of the International Physicians for the Prevention of Nuclear War. Physicians from the United States and the Soviet Union attended the meeting, which won the support of Pope Paul II, Indira Ghandi, Gary Hart, Walter Mondale, President Ronald Reagan, and representatives of many nations. The convention concluded, in agreement with David Bradley's observations in 1948, that it would be impossible for physicians to cope with the havoc created by even a limited nuclear war.[24]

Where statesmen, clerics, teachers, philosophers, and lawyers have failed in the past, physicians have an almost sacred responsibility to succeed. Since ancient times, people have respected physicians and, although critical at times because of the unethical conduct of a handful of practitioners, they have always associated physicians with their well-being.

The physician must turn to the greatest role the times have forced upon him. Of what avail are his or her amazing discoveries in the laboratory and clinic if unreason prevails and life on earth is obliterated in a half hour's duration? The scholar's mission has been succinctly stated by Erasmus: "But to become a philosopher, it is not enough to wear a gown and a great wig, or take degrees, and annex capitals to your name. It is to despise these things, which all the Vulgar, great and small, dote upon, and to view things in a light very different from the corrupt majority."[25]

In "The American Scholar," Emerson chastised the timid scholar memorably:

> Against the heroism of soldiers, I set the heroism of scholars. These are giddy times . . . Giddy times went before them and new times are times of arraignment, times of trial and times of judgment. Tis because the scholars did not learn to teach because they were traders and left their altars and libraries and worship of truth and played the sycophant to presidents, generals, and members of Congress, and gave degrees to those whom they ought to have rebuked, incurring the contempt of those whom they ought to have put in fear.[26]

It is surprising that more medical men have not joined Rabelais, Maudsley, and Osler in denouncing war. Although other professionals have deep commitments to serve mankind, physicians have a more compelling reson to cry out against war because of their intimate relationship with life and death. When Dr Andrew Manson, in Cronin's *The Citadel*, gives life to a stillborn, he cries out exultantly: I've done something, Oh God! I've done something real at last."[27] Each physician who ushers in new life, discovers causes and cures of disease, repairs damage to defective bodies, and helps the terminally ill to easier finales, should ask himself an urgent question: "What can I as a healer of bodies and minds contribute to the healing of an ailing world speeding chaotically to doomsday?"

REFERENCES

1. Hersey J: *Hiroshima.* New York, Viking Press, 1946, p. 33.
2. Butterfield LH, Garet W, Friedlaender M: *The Earliest Diary of John Adams.* Cambridge, MA, Belknap Press, 1966, p 37.
3. *Ibid*, p 40.
4. Koch A, Peden W: *The Selected Writings of John Quincey Adams.* New York Alfred A. Knopf, 1946, p 184.
5. Ulich R (ed): *Three Thousand Years of Educational Wisdom.* Cambridge, MA, Harvard University Press, 1973, vol 2, p 259.
6. Erasmus D: Letter to Anthony A. Berges, 1513, in Mayer P (ed): *The Pacific Conscience.* New York, Holt, Rinehart, and Winston, 1966, pp 55–56.
7. Erasmus D: A digression on the folly of kings, in *The Complaint of Peace.* London, Routledge, 1975, p 121.
8. *Ibid*, pp 46–47.
9. Erasmus D: *In Praise of Folly.* Hudson HH (transl). Princeton, Princeton University Press, 1951, p 133.
10. Tumulty JJ: *Woodrow Wilson As I Knew Him*, New York, Literary Digest, 1921, p 251.
11. Bourne RS: *War and the Intellectual.* Boston, Porter Sargent, 1943, p 194.
12. Curie E: *Madame Curie.* New York, Literary Guild, 1937, pp 227–228.
13. Levy H: The military medicinemen, in Ehrenreich J (ed): *The Cultural Crisis of Modern Medicine.* New York, Monthly Review Press, 1977, p 288.
14. Auden WH: The unknown citizen, in Perrine L (ed): *Sound and Sense.* New York, Harcourt, Brace, and Jovanovich, 1973, p 120.
15. Emerson RW: The American scholar, in MacDowell T (ed): *The Romantic Triumph.* New York, Macmillan and Company, 1940, p 158.
16. Rabelais F: *The Five Books of Gargantua and Pantagruel*, Le Clercq J (transl). New York, The Modern Library, 1944, p xxi.
17. *Ibid*, p 131.
18. Osler W: Chauvinism in medicine, in *Aequanimitas.* Philadelphia, P Blakiston, 1932, p 271.
19. Maudsley H: Lecture at University College, London. *The Popular Science Monthly*, January 1877; 57:341.
20. *Ibid*, p 345.
21. Bradley D: *No Place to Hide.* Boston, Little Brown, and Company, 1948, p 165.
22. Halleck SL: *The Politics of Therapy.* New York, Science House, 1971, p 165.
23. Don't let us die. *Physicians for Social Responsibility.* Cambridge, MA 1984.
24. Kerr P: "Physicians urge end to arms race." *New York Times*, June 10, 1984, L-17, column 1.
25. Erasmus D: A digression on the folly of kings, in *The Complaint of Peace.* London, Routledge, 1975, pp 121.
26. Quoted by Emerson E: Emerson and scholars. *Harvard Graduate Magazine*, March 14, 1906, p 389.
27. Cronin AJ: *The Citadel*, Boston, Little Brown and Company, 1938, p 76.

PROGRESS IN CARDIOLOGY

Edited by Paul N. Yu, MD and John F. Goodwin, MD. 290 pp. Philadelphia, Lea & Febiger, 1983. $45.00 (hardcover)

This hardy annual is informative and well written, with a good mixture of basic considerations and clinical applications of the subjects covered. This is a relatively small volume with a distinguished international editorship from the United States and Great Britain. The authors are internationally known and generally outstanding.

About two-thirds of the text is devoted to a symposium on nuclear cardiology, ably edited by George Beller. This section provides a timely and thorough review of established methods and newer imaging techniques. It includes fine chapters on radionuclide angiography in valvular heart disease (by Boucher and Okada), with a good discussion on the limitations of the techniques, but rather vague—permissably vague in view of the lack of definitive knowledge in this area—guidelines as to surgery in asymptomatic mitral and aortic regurgitation. The chapters also consider radionuclide studies of ventricular performance in coronary artery disease (by Zaret and Berger), and clinical applications of myocardial perfusion scintigraphy (by Gibson and Watson), including a long section on methodology which, like spinach, you may not like but is good for you. Newer techniques discussed include digital subtraction angiography (by Higgins et al), proton nuclear magnetic resonance imaging of the heart (by Ratner et al), myocardial imaging with positron emitters (by Goldstein et al)—a hefty and informative discourse of 43 pages, perhaps too hefty for a volume of this size.

A potpourri of topics follow, all of which are well written. These include an intelligent and interesting chapter from the Soviet Union by Chazov on the arterial wall in atherosclerosis. The genetics of cardiomyopathies is covered by Emanuel and Withers. A group of Japanese authors (Kawai et al) provides a chapter on recent advances in the study of hypertrophic and dilated cardiomyopathy, which is thorough, except for the lack of information on the utility of myocardial biopsies in formulating therapeutic programs. There is a chapter on clinical experience in over 2,000 endarterectomies from 1968 to 1982 by Gersbach and Hahn from Switzerland, which is impressive in its numbers, but, in my view, somewhat disappointing in its results—disappointing enough to question whether this is truly a fundamental advance. A chapter on new antihypertensive drugs by Frohlich presents a crisp review, but the topic is well worn and I think suffering from overexposure.

The final chapter, by Kennedy, discusses intracoronary streptokinase treatment of acute myocardial infarction. This is a balanced and timely discussion, but one would have liked to see more analysis of intravenous streptokinase and a discussion of other antithrombotic agents.

In general this is a fine volume. The editors and authors have done their job well. The publishers should also be commended for the clear type, good illustrations, and careful proofreading. Even with the 50% price increase over the last volume, the cost appears acceptable. The strongest section is on imaging techniques.

As they say on Wall Street, this is a definite "buy." If that is not possible, it is a definite "read" for all students and practitioners with more than casual contact with cardiologic diseases.

LESLIE A. KUHN, MD
The Mount Sinai Medical Center
New York City 10029

THE HORROR OF LIFE

By Roger L. Williams. 381 pp. Chicago, The University of Chicago Press, 1980. $22.50 (softcover)

Does disease influence cultural creativity? In this book, Professor Roger L. Williams—a historian whose area of specialization is 19th century France—sets out to trace the connections between the presumed tertiary syphilis of five major French writers of this period—Charles Baudelaire, Jules de Goncourt, Gustave Flaubert, Guy de Maupassant, and Alphonse Daudet—and the grimly pessimistic tone and substance of their literary output.

Well into his research he confesses that he discovered several facts that undermined his thesis. While each author suffered from a neurologic ailment that was ultimately fatal, a prior syphilitic infection was not proven as the precipitating factor in every case. Even for the authors in whom syphilis might have been a factor, most of their major writing preceded the debilitating phase of the illness. The relationship between an early syphilitic infection and later neurologic involvement leading to general paresis, insanity, and death (in 30% of those affected) had not been recognized. Thus, anticipation of such a dreaded outcome could not have troubled the minds of these men or colored their writing. Finally, in France, where by conservative estimate 15% of the population had syphilis, this ailment was not a source of monstrous shame and self-loathing, but almost a rite of passage in certain circles. It was inconvenient, unpleasant, and vulgar, but also a source of adolescent schoolboy boasts.

By the time of these revelations, Williams had learned enough of the early lives of these men to posit an alternative thesis. The "horror of life" expressed in the bleakness of their writings was in each case the literary projection of individual neuroses, shaped by early emotional attachments and disappointments. To make this case, he offers the five minibiographies—varying in length from 50 to 100 pages—that make up the body of this book.

There can be no denying the impact of such life experiences on the writers' world views. In fact, this biographical technique is all but a cliché of 20th century post-Freudian literary analysis. However, at least two considerations must give us pause in accepting this explanation as definitive. Not every writer who has grown up in an emotionally compromised setting has written in a similarly bitter and depressed manner. Other factors must also be at play here that limit the predictive authority of Williams' theory. Secondly, limited emphasis is given to the deterministic and pessimistic intellectual, social, cultural, and political climate in which these authors matured, thereby down-playing significant potential sources of influence.

One must assume that Williams, as a professional historian, is so familiar with these larger social currents that he takes it for granted that the audience for this book is equally aware of them. For a broader readership it is probably as unwise to omit elaboration of these influences upon the authors' outlook as it is for a physician to elicit the particulars of a patient's medical history while ignoring the environmental, social, and cultural contexts in which ailments originate and progress.

A basic paradox remains unresolved if we accept Williams' premise that these writers were truly despairing of the "horrors of life:" How, then, did they answer the question of what purpose was to be served, in an alien unresponsive universe, by their passionate persistence in writing to the very end of their lives?

What appeal can this book have for physicians who have not cultivated a specific interest in the 19th century French literary scene?

At least one-third to one-half of each mini-biography is devoted to a detailed description of the symptoms of emotional and physical illness suffered by the individual authors from childhood on, with the greatest emphasis given to the course of their terminal neurologic diseases. In each case Williams has done apparently exhaustive research to uncover the recorded opinions, arguments, diagnoses, and prescribed treatments of the various physicians called in for consultation.

Those interested in the history of science will find titillation in the medical dogmas of the period: the quaint taxonomies, the curious theories, the compelling fads, or the appalling treatments. They will also be heartened by the occasional rejection of conventional wisdom and received knowledge. A few prescient individuals formulated imaginative clinical hypotheses, which only the improved technology of a later era could prove accurate. For example, in 1894, after a decade of clinical observation, Dr Alfred Fournier submitted to the Académie de Médecine the supposition that syphilis was responsible for general paralysis. Not until 1913 were laboratory tests available to confirm this.

In sum, Williams' fall-back thesis, that personality shapes product, is both too narrow and too shopworn to excite. But the details of his study offer an elucidating glimpse into the beliefs and practices of 19th century French medicine. Analogizing from the rapid obsolescence of those once dearly-held convictions, we must caution restraint in the self-congratulatory tendencies of our own medical community.

TALAH BELLIN, MA
SUNY Downstate Medical Center
New York City 11203

THE HEALTH OF CHINA

By Victor W. Sidel, MD, and Ruth Sidel, PhD. 251 pp. Boston, Beacon Press, 1983. $13.50 (hardcover), $7.95 (softcover)

Ruth and Victor Sidel are the ranking American experts on medical affairs in China. They were members of the first American medical delegation to go to China in 1971, five months after the Chinese table tennis team visited the United States. Since that time they have made several return visits, and their son Mark Sidel, who has contributed a chapter on education, has resided in China for an extended period of time. The Sidels have brought to this volume not only a seasoned knowledge of China, but also a profound understanding of medical care systems around the world.

Much has happened in China since the Sidels' first visit. The Cultural Revolution has passed, but some of its effects linger; the "Gang of Four" (Maoist hard liners) was removed from power in 1976 and Deng Xiaoping, who emerged in 1977, has now become a major force in Chinese life and politics. Under Deng's policies, China has accelerated its modernization, which is not to be confused with "Westernization."

The Sidels graphically show how current policies have had a major impact on all facets of Chinese life, tracing the change from rural communes and barefoot doctors to urban hospitals. With incentives in the form of profits from agricultural productivity, peasant farmers can now obtain consumer products and can substantially improve their material well being. In medicine, technology is being imported from the West; Chinese scientists and physicians returning from training abroad bring with them modern ideas. Medical schools have upgraded their curricula compared to what was taught during the Cultural Revolution. On the other hand, traditional medicine, including acupuncture, herbal medicine, flexible splinting and other techniques, which flowered during the Cultural Revolution, has continued to thrive. It is being studied intensely and integrated into "scientific medicine" as part of the current modernization program.

The book is organized into eight chapters divided among four sections. The sections are: From Mao to Modernization, Health Care Services, Human Services, and China's and the World's Future. There are in addition seven appendices that provide much useful data and information about diseases, medical education, health manpower, the medical care system and its resources, and medical research. Copious notes keyed to each of the eight chapters provide the reader with additional sources of information, as does the bibliography.

Dramatic change and sharp alterations of course have characterized the Chinese health care system in the 36 years since the People's Republic was established. Westerners are more accustomed to incremental change. Yet the Chinese have opted for quantum leaps and wide swings in direction. This has come about not only out of necessity but also because of sharply different policies promulgated by a changing leadership. There is no guarantee that the current course of "modernization" in China will be steady and long. There are already abundant signs of opposition to Deng Xiaoping's policies in some quarters of China.

The Sidels, however, end their book on an optimistic note. They think that the Chinese will preserve the principles that have brought them so far so fast, while moving off in other directions. As they see it, China can preserve its revolutionary principles and past strengths while simultaneously introducing new technology and raising living standards. Whether or not this happens is a matter of conjecture. But the Sidels' point is well made, for the Chinese have demonstrated an enduring capacity to change some things, modify others, and preserve still others intact.

The Health of China is a fascinating book, written in an easy to read and pleasant style. While its principal focus is on health and medical care, the authors illuminate many important facets of Chinese life. This makes the book must reading for anyone contemplating a trip to China. Those interested in history, modern politics, and developmental problems in the Third World will find it equally useful.

PASCAL JAMES IMPERATO, MD
Deputy Editor

DRUG TREATMENT OF HEART FAILURE

Edited by Jay N. Cohn, MD. 210 pp, illustrated. New York, Yorke Medical Books, 1983. $35.00 (hardcover)

Since the 19th century, the use of digitalis and its derivatives has been the traditional therapy for congestive heart failure. Parenteral and, subsequently, oral diuretics were additional means of therapy. Presently, on the basis of newly defined and amplified physiologic concepts, there has been an elucidation of the mechanisms of action of these drugs. The development of other methods for the treatment of heart failure has also been accelerated. New drugs have been discovered, and existing drugs—the nitrates, for example—have been employed more frequently. In addition, the clinician has scientific reasons, previously unknown, for treating a patient with a particular drug.

This book deals with both the pathophysiology of heart failure and the rationale of the methods used to treat it. The concepts of preload and afterload reduction and inotropic stimulation are explained, and their relation to therapy is detailed. The relation of the renin angiotensin aldosterone system to heart failure is also reviewed. The section on pathophysiology is particularly well written and interesting. The charts, tables, and diagrams are pertinent, clear, and comprehensive. Each chapter is followed by a voluminous bibliography.

Unfortunately, some aspects of the book are less than satisfactory. The editor states in the forward that "the present volume is aimed at providing the clinician with the background necessary to place these new therapeutic efforts in perspective." One has difficulty with some sections, however, in deciding what group of clinicians is being addressed. Some of the data presented are so basic that it is impossible to imagine that a first-year medical student would not be familar with it. For example, there is a detailed description of the method of demonstrating pitting edema: "A finger is applied against the bony structures of the foot or leg; the displacement of fluid results in a dimple or pit." The section on inotropic agents is extremely detailed in relating the biochemical structures of these drugs and the experimental work supporting their use. There is a final chapter on the comprehensive approach to the treatment of heart failure. I found this section to be somewhat brief and superficial, but nonetheless a good summary.

I recommend this volume to clinicians who are interested in the application of laboratory principles to the rapidly changing treatment of the patient with heart failure.

WILLIAM J. SCARPA, MD
The Long Island College Hospital
New York City 11201

INFECTIONS: RECOGNITION, UNDERSTANDING, TREATMENT

Edited by Jean-Claude Pechere, MD and Charles Cherubin, MD. 606 pp, illustrated. Philadelphia, Lea & Febiger, 1984. $32.50 (hardcover)

The editors' stated objective for this volume is to present a "true-to-life" approach to the diagnosis and treatment of infectious diseases. This is a laudatory goal. Unfortunately, not all of the 46 contributors to the 36 chapters have succeeded in meeting the stated objective. Many of the chapters are sketchy in areas that are important for the intellectual and diagnostic approach to the problem at hand.

For example, in the chapter that deals with typhoid fever a single graph of the clinical course of typhoid fever showing the duration of fever, other symptoms, and the periods of bacteremia, bacteriuria, and fecal shedding of typhoid bacilli would have added to the impact of the discussion without increasing the length of the chapter.

An example of an important omission is the absence of a discussion of the clinical characteristics of *Nocardia sp* infection, either in the chapter on pulmonary infections or on infections in immunosuppressed patients. Also omitted is a consideration of the use of acyclovir in the treatment of herpes simplex virus infection. This is an important topic for discussion, especially since acyclovir has been featured in the news media.

There are also many statements that are not technically correct. For example, in the chapter on liver infection, in the paragraph entitled "Treatment of Chronic Hepatitis," it is stated that in patients with hepatitis but lacking markers of hepatitis B virus infection, "most investigators have reported success with prolonged prednisone treatment." This statement is open to argument, because it is only in those patients with signs of active immunologic reactions, such as in lupoid hepatitis, that one can claim some success with prednisone treatment.

Notwithstanding these deficiencies, there are many excellent chapters in the volume, with tables that are replete with clear and concise information. Some of the more outstanding presentations include the chapters on infective endocarditis and urinary tract infections. The chapters on infective endocarditis contain a short, but to the point, consideration of the definition, epidemiology, and pathophysiology of endocarditis. The indications and risks of prophylactic antimicrobial therapy are succinctly presented in a table. The chapter on urinary infections has an excellent discussion of the most common infecting organisms and the vicissitudes of antimicrobial therapy. The section on infections of the eye has excellent tables with clinical data on almost the entire gamut of ocular infections. The photographs were well chosen and illustrate the salient ocular findings in the respective infections.

This volume is not recommended as a basic reference work for the expert in infectious diseases or even for the well-read and experienced specialist in internal medicine. I believe it will appeal to senior medical students, house staff, and physicians in general practice. Furthermore, the volume should be of use to surgical specialists, especially general surgeons and those engaged in the practice of obstetrics and gynecology. This book does not have the breadth and detail of reference works on infectious diseases.

SHALOM HIRSCHMAN, MD
Mount Sinai Medical Center
New York City 10029

BOOKS RECEIVED

ANATOMY

Correlative Neuroanatomy of Computed Tomography and Magnetic Resonance Imaging. By J. de Grott, MD, PHD. 249 pp, illustrated. Philadelphia, PA, Lea & Febiger, 1984. $45.00 (hardcover)

Gray's Anatomy. Thirtieth American Edition. Edited by Carmine D. Clemente, PHD 1676 pp, illustrated. Philadelphia, PA, Lea & Febiger, 1985. $68.50 (hardcover)

CARDIOLOGY

Doppler Echocardiography. By Stanley J. Goldberg, MD, Hugh D. Allen, MD, Gerald R. Marx, MD, Celia J. Flinn, MD. 178 pp, illustrated. Philadelphia, PA, Lea & Febiger, 1985. $30.00 (hardcover)

Doppler Ultrasound in Cardiology. Physical Principles and Clinical Applications. By Liv Hatle, MD, Bjorn Angelsen, Dr Techn. Second Edition. 331 pp, illustrated. Philadelphia, PA, Lea & Febiger, 1985. $30.00 (hardcover)

Life Stress and Coronary Heart Disease. By Ulf De Faire, MD, Tores Theorell, MD. 118 pp, illustrated. St. Louis, MO, Warren H. Green, Inc, 1984. $18.50 (hardcover)

Surviving Your Heart Attack. The Duke University Complete Heart Treatment Program. By James V. Warren, MD and Genell J. Subak-Sharpe. 202 pp. Garden City, NY, Doubleday & Co, Inc, 1984. $13.95 (hardcover)

GERIATRICS

The Aging Musculoskeletal System: Physiological and Pathological Problems. Edited by Carl L. Nelson, MD, Anthony P. Dwyer, MD. 178 pp, illustrated. Lexington, MA, The Collamore Press, 1984. $30.00 (hardcover)

HEALTH ADMINISTRATION

The AMA and U.S. Health Policy: Since 1940. By Frank D. Campion. 301 pp, illustrated. Chicago, IL, Chicago Review Press, 1984. $25.00 (hardcover)

Health Care & The Social Services: Social Work Practice in Health Care. By Richard J. Estes, MSW, DSW. 410 pp. St. Louis, MO, Warren H. Green, Inc., 1984. $42.50 (hardcover)

INFECTIOUS DISEASES

Changing Patterns of Infectious Disease. By Lois M. Bergquist, PHD. 285 pp, illustrated. Philadelphia, PA, Lea & Febiger, 1984. $28.50 (paperback)

Boyd's Introduction to the Study of Disease. Ninth Edition. By Huntington Sheldon, MD. 702 pp, illustrated. Philadelphia, PA, Lea & Febiger, 1984. $28.50 (hardcover)

INTERNAL MEDICINE

Treating Type A Behavior and Your Heart. By Meyer Friedman, MD, and Diane Ulmer, RN, MS. 285 pp. New York, NY, Alfred H. Knopf, 1984. $15.95 (clothcover)

LEGAL MEDICINE

Handbook of Living Will Laws 1981–1984. By Peg Cameron. 100 pp. New York, NY, Society for the Right To Die, 1984. $5.00 (paperback)

MISCELLANEOUS

Clinical Biophysics. Edited by Michael Anbar, PHD, Robert A. Spangler, MD, PHD, Peter Scott, PHD. 747 pp, illustrated. St. Louis, MO, Warren H. Green, Inc, 1985. $75.00 (hardcover)

How To Overcome Dependency On Cocaine, Caffeine, Hallucinogens, Marijuana, Speed and Other Stimulants The Natural and Permanent Way. Getting Over Getting High. By Bernard Green, PHD. 265 pp. New York, NY, William Morrow & Co, 1985. $6.95 (paperback)

How To Save Dollars With Generic Drugs. A Consumer's Guide to High-Quality, Low-Priced Medicines. Why You Pay Too Much for Prescription and Over-the-Counter Drugs and What You Can Do About It. By Max A. Ferm, PHD, and Betty Ferm. 298 pp. New York, NY, William Morrow & Co, 1985. $17.95 (cloth)

Medical Economics Encyclopedia of Practice and Financial Management. Edited by Lawrence Farber. 1280 pp, illustrated. Oradell, NJ, Medical Economics Co, 1985. $69.95 (hardcover)

Medicine and the Management of Living. Taming the Last Great Beast. By William Ray Arney and Bernard J. Bergen. 202 pp. Chicago, IL, The University of Chicago Press, 1984. $19.95 (hardcover)

Medicine in the Mishneh Torah of Maimonides. By Fred Rosner, MD. 325 pp. Hoboken, NJ, KTAV Publishing House, Inc, 1984. $9.95 (paperback)

Physicians' Guide to Professional and Personal Advisers. By Warren Boroson. 248 pp. Oradell, NJ, Medical Economics Books, 1985. $16.95 (paperback)

The Real Pushers. A Critical Analysis of the Canadian Drug Industry. By Joel Lexchin, MD. 272 pp. Vancouver, BC, Canada, New Star Books Ltd, 1984. $8.95 (paperback)

Strong Medicine. By Arthur Hailey. 448 pp. Garden City, NY, Doubleday & Co, 1984. $16.95 (hardcover)

The Vitamin C Connection. Getting Well and Staying Well With Vitamin C. By Dr Emanuel Cheraskin, Dr W. Marshall Ringsdorf, Jr, Dr Emily L. Sisley. 336 pp. New York, NY, Bantam Books, 1983. $3.95 (paperback)

NUTRITION

The Diabetic's Brand-Name Food Exchange Handbook. Food exchanges for over 3,000 supermarket, grocery store, and fast-food products. By Andrea Barrett. 171 pp. Philadelphia, PA, Running Press, 1984. $14.95 (hardcover)

Eating For Life. The Ultimate Diet. By Nathaniel Altman, Ulla Kristiina Laakso, MD. 176 pp, illustrated. New York, NY, Vegetus Books, 1984. $6.95 (paperback)

OPHTHALMOLOGY

Human Cataract Formation. By Ciba Foundation. 284 pp, illustrated. Newark, NJ, Ciba Pharmaceutical Co, 1984. $35.00 (hardcover)

Ocular Differential Diagnosis. Third Edition. By Frederick Hampton Roy, MD. 466 pp. Philadelphia, PA, Lea & Febiger, 1984. $30.00 (hardcover)

Practical Ophthalmic Problems. By Wayne F. March, MD. 91 pp, illustrated. St. Louis, MO, Warren H. Green, Inc, 1984. $12.50 (paperback)

PSYCHIATRY

Psychiatric Medicine. A Handbook. By James L. Mathis, MD. 198 pp. St. Louis, MO, Warren H. Green, Inc, 1984. $22.50 (hardcover)

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Edwin Jerome Adlerman, MD, Bronxville. Died January 22, 1984; age 67. He was a 1940 graduate of Georgetown University School of Medicine. Dr Adlerman was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Urology. His memberships included the American Urological Association, the American Academy of Compensation Medicine, Inc., the American Occupational Medical Association, the New York County Medical Society and the Medical Society of the State of New York.

John T. Alexander, MD, Huntington. Died October 7, 1984; age 65. Dr Alexander was a 1949 graduate of Harvard Medical School. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

William D. Arnold, MD, New York. Died October 4, 1984; age 59. He was a 1948 alumnus of Cornell University Medical College. Dr Arnold was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. His memberships included the Academy of Medicine, the American Academy of Orthopaedic Surgeons, the American Association for the Surgery of Trauma, the New York County Medical Society and the Medical Society of the State of New York.

Clifton H. Berlinghof, MD, Binghamton. Died October 16, 1984; age 86. Dr Berlinghof was a 1925 graduate of the Johns Hopkins University School of Medicine. He was a Fellow of the American College of Physicians, and a member of the New York State Society of Internal Medicine, the Broome County Medical Society and the Medical Society of the State of New York.

Jacob Maurice Bloom, MD, New York. Died August 22, 1984; age 90. Dr Bloom was a 1920 graduate of the New York University College of Medicine. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

David B. Blumenfeld, MD, New York. Died October 6, 1984; age 86. Dr Blumenfeld was a 1923 alumnus of the State University of New York Downstate Medical Center. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Robert Abbe Chait, MD, Rockville Centre. Died September 29, 1984; age 75. Dr Chait was a 1935 alumnus of the Université de Paris Faculté de Médecine. He was a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology, the American Academy of Allergy, the Nassau County Medical Society and the Medical Society of the State of New York.

Simeon O. Chlenoff, MD, New York. Died May 7, 1984; age 89. He was a 1922 alumnus of Universität Bern Medizinische Fakultät. Dr Chlenoff was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the New York Pathological Society, the American Psychiatric Association, the Academy of Psychoanalysis, the New York County Medical Society and the Medical Society of the State of New York.

John Gordon Cole, MD, New York. Died February 6, 1984; age 76. He was a 1932 alumnus of the University of Minnesota Medical School. Dr Cole was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Ophthalmology. His memberships included the Academy of Medicine, the New York Ophthalmology Society, the American Academy of Ophthalmology and Otolaryngology, the American Ophthalmological Society, the New York County Medical Society and the Medical Society of the State of New York.

Henry S. F. Cooper, MD, New York. Died September 10, 1984; age 89. He was a 1924 alumnus of Harvard Medical School. Dr Cooper was a member of the Academy of Medicine, the New York County Medical Society and the Medical Society of the State of New York.

Richard E. Counselman, MD, New Canaan, Connecticut. Died October 21, 1984; age 62. Dr Counselman was a 1951 graduate of the Georgetown University School of Medicine. He was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Emergency Physicians, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

George Dacks, MD, Livonia. Died April 7, 1984; age 81. Dr Dacks was a 1934 alumnus of the University of Rochester School of Medicine and Dentistry. He was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Joseph Theodore Eagan, MD, Oneonta. Died September 7, 1984; age 88. Dr Eagan was a 1921 graduate of the Yale University School of Medicine. He was a member of the American Thoracic Society, the Medical Society of the County of Otsego and the Medical Society of the State of New York.

Max Ellenberg, MD, New York. Died August 18, 1984; age 73. He was a 1935 alumnus of the New York University College of Medicine. Dr Ellenberg was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the Clinical Society of the New York Diabetes Association, the New York County Medical Society and the Medical Society of the State of New York.

Edwin Joaquin Euphrat, MD, Harrisburg, Pennsylvania. Died October 31, 1984; age 77. He was a 1934 alumnus of the State University of New York Downstate Medical Center. Dr Euphrat was a Diplomate of the American Board of Radiology, and a member of the Radiological Society of North America, Inc., the Onondaga County Medical Society and the Medical Society of the State of New York.

Solomon Salmen Fiderer, MD, Brooklyn. Died October 1, 1984; age 91. Dr Fiderer was a 1925 graduate of Medizinische Fakultät der Universität, Wien. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Morris H. Goldenberg, MD, Tamarac, Florida. Died September 28, 1984; age 80. He was a 1929 alumnus of the George Washington University School of Medicine. Dr Goldenberg was a Fellow of the American College of Radiology and a Diplomate of the American Board of Radiology. His memberships included the Radiological Society of New York State, the New York Roentgen Society, the American Public Health Association, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Rudolph Victor Gorsch, MD, Vernon, Connecticut. Died October 13, 1984; age 96. Dr Gorsch was a 1918 alumnus of the Columbia University College of Physicians and Surgeons. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

James Henry Gray, Jr., MD, Ransomville. Died September 23, 1984; age 74. He was a 1935 graduate of the University of Buffalo School of Medicine. Dr Gray was a member of the Medical Society of the County of Niagara and the Medical Society of the State of New York.

William Robert Griffin, MD, Garden City. Died November 3, 1984; age 79. He was a 1936 alumnus of the Georgetown University School of Medicine. Dr Griffin was a member of the Academy of Medicine, the Pan American Association of Ophthalmology, the Nassau County Medical Society and the Medical Society of the State of New York.

Herbert Warner Grover, MD, Altamont. Died October 27, 1984; age 56. He was a 1954 alumnus of the Georgetown University School of Medicine. Dr Grover was a Fellow of the American Academy of Family Physicians and a Diplomate of the American Board of Family Practice. He was a member of the Medical Society of the County of Schenectady and the Medical Society of the State of New York.

Joseph Guccione, MD, Ridgewood. Died September 26, 1984; age 58. Dr Guccione was a 1959 graduate of Università degli Studi di Roma, Facoltà di Medicina e Chirurgia. He was a member of the Academy of Medicine, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Gordon M. Hemmett, MD, Rochester. Died September 15, 1984; age 74. He was a 1938 graduate of the McGill University Faculty of Medicine, Montreal. Dr Hemmett was a Fellow of the American College of Preventive Medicine and a Diplomate of the American Board of Preventive Medicine, Inc. His memberships included the Academy of Medicine, the American Academy of Occupational Medicine, the American Occupational Medical Association, the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Henry Hillel, MD, Syracuse. Died September 20, 1984; age 89. He was a 1921 alumnus of Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin, Prussia. Dr Hillel was a Qualified Fellow of the International College of Surgeons, and a member of the Academy of Medicine, the Onondaga County Medical Society and the Medical Society of the State of New York.

Sidney A. Hollin, MD, New York. Died March 13, 1984; age 54. He was a 1956 graduate of the Temple University School of Medicine. Dr Hollin was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Neurological Surgery. His memberships included the Academy of Medicine, the New York Society of Neurosurgery, the American Association of Neurological Surgeons, the New York County Medical Society and the Medical Society of the State of New York.

Leo Jenkins, MD, Long Island City. Died November 5, 1984; age 72. He was a 1935 alumnus of the New York University College of Medicine. Dr Jenkins was a Diplomate of the American Board of Pediatrics, and a member of the American Academy of Pediatrics, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Murray L. Kaiser, MD, Long Island City. Died October 24, 1983; age 63. Dr Kaiser was a 1946 graduate of the Chicago Medical School. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Abraham Katz, MD, Coconut Creek, Florida. Died October 30, 1984; age 81. He was a 1932 alumnus of the University of Maryland School of Medicine. Dr Katz was a Fellow of the American Academy of Family Physicians and a Diplomate of the American Board of Family Practice. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

William J. Keefe, MD, Wilmington, Delaware. Died June 3, 1984; age 94. He was a 1918 alumnus of the McGill University Faculty of Medicine, Montreal. Dr Keefe was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Morris Kimberg, MD, Lake Worth, Florida. Died October 6, 1984; age 92. Dr Kimberg was a 1915 graduate of the New York University College of Medicine. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Hans Kuehn, MD, New York. Died June 13, 1984; age 83. He was a 1926 alumnus of Medizinische Fakultät der Universität, Wien. Dr Kuehn was a member of the New York County Medical Society and the Medical Society of the State of New York.

Hans Kulka, MD, Wurtsboro. Died September 13, 1984; age 70. Dr Kulka was a 1942 alumnus of Universität Zurich Medizinische Fakultät. He was a member of the Medical Society of the County of Orange and the Medical Society of the State of New York.

Rene Pierre Lacombe, MD, New York. Died March 3, 1984; age 84. Dr Lacombe was a 1936 graduate of the Université de Paris Faculté de Médecine. His memberships included the Academy of Medicine, the American Psychoanalytic Association, the American Psychiatric Association, the New York County Medical Society and the Medical Society of the State of New York.

Richard F. Mascola, MD, Jamaica. Died October 14, 1984; age 76. Dr Mascola was a 1933 alumnus of the University of Vermont College of Medicine. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Joseph Bryant Mathewson, MD, Ithaca. Died September 18, 1984; age 83. He was a 1926 graduate of Cornell University Medical College. Dr Mathewson was a member of the Medical Society of the County of Tompkins and the Medical Society of the State of New York.

Luigi Mazza, MD, Rome, Italy. Died May 23, 1984; age 55. He was a 1951 alumnus of Università degli Studi di Roma, Facoltà di Medicina e Chirurgia. Dr Mazza was a Fellow of the American College of Anesthesiologists and a Diplomate of the American Board of Anesthesiology. He was a member of the American Society of Anesthesiologists, Inc., the New York County Medical Society and the Medical Society of the State of New York.

Edward P. McDonald, MD, Albany. Died September 21, 1984; age 82. He was a 1926 graduate of the Jefferson Medical College of Philadelphia. Dr McDonald was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Medical Society of the County of Albany and the Medical Society of the State of New York.

Milton Mendelson, MD, Central Valley. Died September 16, 1984; age 81. Dr Mendelson was a 1930 alumnus of the State University of New York Upstate Medical Center. He was a member of the Medical Society of the County of Orange and the Medical Society of the State of New York.

William Needles, MD, New York. Died November 12, 1984; age 82. He was a 1925 alumnus of the Columbia University College of Physicians and Surgeons. Dr Needles was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the Academy of Medicine, the New York Neurological Society, the American Psychoanalytic Association, the American Psychiatric Association, the New York County Medical Society and the Medical Society of the State of New York.

Armand Major Patella, MD, Brooklyn. Died May 6, 1984; age 72. Dr Patella was a 1940 alumnus of the State University of New York Downstate Medical Center. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Morris Louis Rakieten, MD, Bay Shore. Died November 7, 1984; age 85. He was a 1931 graduate of the Yale University School of Medicine. Dr Rakieten was a Fellow of the American College of Pathologists and a Diplomate of the American Board of Pathology. He was a member of the Academy of Medicine, the Society of American Bacteriologists, the Suffolk County Medical Society and the Medical Society of the State of New York.

Geremia Rizzuti, MD, Glen Cove. Died October 6, 1984; age 85. Dr Rizzuti was a 1923 graduate of Università di Napoli Facoltà di Medicina e Chirurgia, and was a member of the Clinical Society of the New York Diabetes Association, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Domenico L. Romano, MD, New York. Died August 29, 1984; age 85. Dr Romano was a 1927 alumnus of Università degli Studi di Bologna, Facoltà di Medicina e Chirurgia. He was a Diplomate of the American Board of Dermatology, Inc., and a member of the American Occupational Medical Association, the New York County Medical Society and the Medical Society of the State of New York.

Livia Halasz Rosenfeld, MD, New York. Died August 16, 1984; age 75. Dr Rosenfeld was a 1933 graduate of Medizinische Fakultät der Universität, Wien, and was a member of the American Fertility Society, the New York County Medical Society and the Medical Society of the State of New York.

Felix R. Rosenhain, MD, Forest Hills. Died November 14, 1984; age 56. He was a 1953 alumnus of the Boston University School of Medicine. Dr Rosenhain was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. He was a member of the Academy of Medicine, the American Rheumatism Association, the American Academy of Orthopaedic Surgeons, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Mitchell J. Rubinow, MD, Miami Beach, Florida. Died September 9, 1984; age 83. He was a 1926 graduate of the State University of New York Downstate Medical Center. Dr Rubinow was a member of the American Academy of Family Physicians, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Avram N. Selinger, MD, Cliffside Park, New Jersey. Died August 1, 1984; age 70. Dr Selinger was a 1944 alumnus of Universitatea din Lasi Facultatea de Medicina. He was a member of the New York Cardiological Society, the New York County Medical Society and the Medical Society of the State of New York.

David Raymond Shapiro, MD, Newburgh. Died June 26, 1984; age 63. He was a 1945 alumnus of the McGill University Faculty of Medicine, Montreal. Dr Shapiro was a Fellow of the American College of Anesthesiologists, and a member of the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc., the Medical Society of the County of Orange and the Medical Society of the State of New York.

Dean Davidge Smith, MD, Naples, Florida. Died April 12, 1984; age 79. Dr Smith was a 1934 alumnus of the Johns Hopkins University School of Medicine.

He was a member of the Broome County Medical Society and the Medical Society of the State of New York.

Jack Spivak, MD, Jamaica. Died September 30, 1984; age 73. Dr Spivak was a 1937 graduate of the Université de Genève Faculté de Médecine. He was a Fellow of the American Academy of Family Physicians. His memberships included the American Geriatrics Society, the American Society of Clinical Hypnosis, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Francis G. Velardi, MD, Brooklyn. Died October 28, 1984; age 85. Dr Velardi was a 1924 graduate of the State University of New York Downstate Medical Center. He was a Fellow of the American College of Allergists, and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

David H. Vrooman, MD, Schenectady. Died October 27, 1984; age 84. Dr Vrooman was a 1924 graduate of the Albany Medical College of Union University. He was a Fellow of the American Academy of Family Practice, and a member of the American Occupational Medical Association, the Medical Society of the County of Schenectady and the Medical Society of the State of New York.

Theodore R. Weinstein, MD, Bronx. Died October 1, 1984; age 49. He was a 1960 graduate of Chicago Medical School. Dr Weinstein was a Fellow of the American College of Angiology, and a member of the New York State Society of Internal Medicine, the Bronx County Medical Society and the Medical Society of the State of New York.

Catherine A. Welsh, MD, Schenectady. Died October 28, 1984; age 78. Dr Welsh was a 1935 alumna of the New York University College of Medicine, and a member of the Medical Society of the County of Schenectady and the Medical Society of the State of New York.

Lanpher Chas Weston, MD, Massena. Died September 22, 1984; age 69. He was a 1943 graduate of the State University of New York Upstate Medical Center. Dr Weston was a Qualified Fellow of the International College of Surgeons, and a member of the Medical Society of the County of St. Lawrence and the Medical Society of the State of New York.

George Albert Zak, MD, New York. Died September 2, 1984; age 71. He was a 1937 graduate of Medizinische Fakultät der Universität, Wien. Dr Zak was a Fellow of the American College of Chest Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the American Thoracic Society, the New York County Medical Society and the Medical Society of the State of New York.

Attention MSSNY Delegates:

The 179th Annual Meeting of the Medical Society of the State of New York will be held at the New York Hilton Hotel, New York City, on June 6–9, 1985.

•

Nominating Committee, 1985

(The committee nominates Officers, Councilors, and Trustees of MSSNY, and Delegates to the American Medical Association.)

Member-at-Large—Bernard J. Pisani, M.D., New York, *Chairman*
First District—Edgar P. Berry, M.D., New York
Second District—Samuel M. Gelfand, M.D., Nassau
Third District—Stephen L. Hermele, M.D., Ulster
Fourth District—Robert A. Reid, M.D., Warren
Fifth District—George Lim, M.D., Oneida
Sixth District—Paul M. DeLuca, M.D., Broome
Seventh District—Victor J. Tofany, M.D., Monroe
Eighth District—James J. Kropelin, M.D., Niagara
Ninth District—John A. Ramsdell, M.D., Westchester
Member-at-Large—Ralph M. Schwartz, M.D., Kings

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089–1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), an abbreviated title of no greater than 40 spaces for use as a running foot, and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear on the text. When there are six or fewer authors, all should be listed; where there are seven or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. McGuigan JE. Peptic ulcer. In: Petersdorf RG, Adams RD, Braunwald E, Isselbacher KJ, Martin JB, Wilson JD, eds. *Harrison's Principles of Internal Medicine.* New York, McGraw-Hill, 1983, pp 1697–1712.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Alan Blum, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

Classified Advertising

New York State Journal of Medicine

GENERAL INFORMATION

All classified advertisements are payable in advance, except for government agencies and advertising agencies; a 15% commission to recognized advertising agencies.

Frequency: Monthly
Mailing date: 15th of the month
Closing date: 30 days before mailing date

Circulation: 27,000 (mainly physicians & medical libraries)

DISPLAY ADVERTISING

	1 time	*6 time*	*12 time*	*Width*		*Depth*
1/4-page	$450	$400	$350	3-1/2″	×	4-7/8″
1/6-page	210	190	170	2-1/8″	×	4-7/8″
1/8-page	190	170	150	3-1/2″	×	2-3/8″
1/16-page	95	85	75	3-1/2″	×	1-1/8″
1/18-page	70	60	50	2-1/8″	×	1-1/2″

PARAGRAPH ADVERTISING

The first several words are set in bold face caps.

Rates: $25.00 per insertion for 50 words or less; additional words are 30 cents each. On request, Dept. numbers will be assigned by the Journal for replies to be forwarded to the advertiser. Dept. numbers are 50 cents per insertion. There is no frequency discount for paragraph ads.

Counting Words: Two initials, each abbreviation, figures consisting of 5 numerals or less, are counted as separate words. For replies, your name and address, or telephone number should appear at the end of the ad and be counted in the total number of words.

For further information, please call the Advertising Department at 516-488-6100.

CLASSIFIED ADVERTISING ORDER FORM

New York State Journal of Medicine
Attn: Classified Advertising
420 Lakeville Rd., P.O. Box 5404
Lake Success, N.Y. 11042

Date____________________

Name______________________________________

Address____________________________________

City/State/Zip______________________________

Payment enclosed: $______________

Number of Insertions: ____________

Assign Dept. Number: ____________

Please make check payable to New York State Journal of Medicine.

FOR PHYSICIANS AND SURGEONS WITH A PART-TIME EXPOSURE ONLY

Now offering Medical Malpractice Insurance on an ***occurrence basis*** at drastically reduced rates thru Frontier Insurance Co., Monticello, NY.

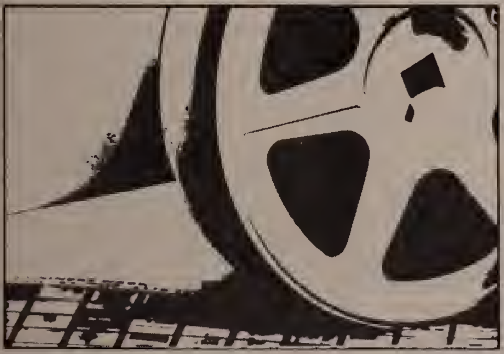
With the tail on claims made policies, you might lose your shirt.

Call or write for further information and application.

MEDICAL
MQ
QUADRANGLE

Medical Quadrangle, Inc.
P. O. Box 456
318 Bedford Road
Bedford Hills, New York 10507
914 666-0170
212 829-3654
"Specializing in Medical Liability Insurance"

This Publication
is available in Microform.



University Microfilms International

Please send additional information
for ______________________
(name of publication)
Name______________________
Institution______________________
Street______________________
City______________________
State__________ Zip__________
300 North Zeeb Road, Dept P.R., Ann Arbor, Mi. 48106

Index to Advertisers

Ayerst Laboratories 9A,10A,11A,12A,25A,26A,27A, 28A,29A,30A,31A,32A,38A,39A,40A
Blue Cross/Blue Shield of Greater NY .... 3rd Cover
Classified Advertising 5A,22A,24A,34A,36A
Eastman Kodak 1A,2A,3A
Eli Lilly and Co. .... 2nd Cover
Geisinger Clinic 21A
Knoll Pharmaceutical 6A,7A,8A
Marion Laboratories 41A,42A
Medical Quadrangle 19A
A.H. Robins 37A
Stuart Pharmaceutical 33A,34A
Upjohn Company .... 4th Cover
Wyeth Laboratories 35A,36A

Mar 22–23. **Infectious Diseases and Human Infertility.** Info: Lauren H. Straub, Dept of Public Relations, Lenox Hill Hospital, 100 E 77th St, New York, NY 10021. Tel: (212) 794-4500.

Mar 22–23. **Ophthalmic Plastic Surgery Workshop: The Eyelids.** Info: Martha Klapp, Manhattan Eye, Ear & Throat Hospital, 210 E 64th St, New York, NY 10021. Tel: (212) 605-3762.

Mar 25–29. **Anesthesiology Comprehensive Review IV.** Info: NYU Medical Center, Post-Graduate Medical School, 550 First Avenue, New York, NY 10016. Tel: (212) 340-5295.

Mar 27. **Learning Disabilities: Treatment as Prevention.** 7 Cat 1 credits. Info: Beverley Baptiste, Beth Israel Medical Center, Nathan D. Perlman Place, New York, NY 10003. Tel: (212) 420-2849.

BROOKLYN

Mar 4. **Comparative Analysis of Inhaled Anesthetics—Factors Governing Selection.** *Mar 5.* **Inhaled vs Fixed Agents for General Anesthesia—Which, When and Why?** Info: Department of Anesthesiology, SUNY Downstate Medical Center, 450 Clarkson Ave, Brooklyn, NY 11203. Tel: (718) 270-1934.

Mar 6. **The Nature of the Immune Defect of AIDS.** *Mar 13.* **Chemotherapy of Brain Tumors.** *Mar 20.* **Breast Imaging Update.** *Mar 27.* **Control of Nausea and Vomiting Caused by Anti-Cancer Drugs.** Info: Harvey Dosik, MD, Interfaith Medical Center, 555 Prospect Place, Brooklyn, NY 11238-9974. Tel: (718) 240-1000.

NASSAU

Mar 1–2. **Babies, Families and Professionals—Clinical Intervention and Research.** Garden City Hotel. Info: Ann J. Boehme, CME, LIJ-Hillside Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-2114.

QUEENS

Mar 29. **Approaches in Sex and Couple Therapy: Beyond the Basics.** Info: Debra Mohr, CME, LIJ-Hillside Medical Center, New Hyde Park, NY 10042. Tel: (718) 470-2114.

SUFFOLK

Mar 13–17. **Family Medicine Update.** Stony Brook. Info: Tamarath K. Yolles, MD, CME, School of Medicine (HSC), 4L, Rm. 177 SUNY at Stony Brook, NY 11794-8437. Tel: (516) 444-2094.

BROOME

Mar 20. **Pulmonary Teaching Day—Update on the Modern Treatment of Respiratory Diseases.** Binghamton. 5 Cat 1 credits. Info: Sandy Cook, CME, United Health Services, Binghamton General Hospital, Mitchell Ave, Binghamton, NY 13903. Tel: (607) 771-2549.

ERIE

Mar 18. **Anesthetic Management of Patients with Multiple Trauma. Multiple Injuries—a Team Approach.** Sheraton Inn. *Mar 19.* **Anesthetic Management of the Patient with Burns.** Erie County Medical Center. *Mar 20.* **Fluid Resuscitation.** Buffalo General Hospital. Info: SUNY at Buffalo, Department of Anesthesiology at Erie County Medical Center, 462 Grider St, Buffalo, NY 14215.

MONROE

Mar 30. **Eighth Annual Spring Seminar in Anesthesiology.** 6 Cat 1 credits. Rochester. Info: Department of Anesthesiology, The Genesee Hospital, 224 Alexander St, Rochester, NY 14607. Tel: (716) 263-5265.

AROUND THE NATION

CALIFORNIA

Mar 7–9. **Brain Insults in Infants and Children: Pathology, Evaluation, Diagnosis and Acute Management.** San Diego. 17 Cat 1 credits. Info: Office of Continuing Medical Education, M-017, UC San Diego School of Medicine, La Jolla, CA 92093. Tel: (619) 452-3940.

Mar 10–13. **New Strategies for Common Clinical Problems.** Tahoe City. *Mar 10–13.* **Office Endocrinology 1985.** *Mar 27–29.* **Symposium on the Use and Standardization of Chemically Defined Antigens.** San Francisco. Info: Extended Programs in Medical Education, Room 569-U, University of California, San Francisco, CA 94143. Tel: (415) 666-4251.

COLORADO

Mar 2–9. **Family Practice Conference.** Vail. 22 Cat 1 credits. *Mar 2–9.* **Cancer Treatment Conference.** Vail. 22 Cat 1 credits. Info: Beth Israel Hospital Conference and Institute Program, PO Box 11338, Denver, CO 80211-0338. Tel: (800) 525-5810.

Mar 9–16. **10th Annual Snow Job in Gynecology and Obstetrics.** Breckenridge. Info: CME, Emory University School of Medicine, 110 WHSCAB, 1440 Clifton Rd, NE, Atlanta, GA 30322.

Mar 9–16. **Decisions in Surgery.** Snowmass. Info: Extended Programs in Medical Education, Room 569-U, University of California, San Francisco, CA 94143. Tel: (415) 666-4251.

Mar 25–29. **Complicated Problems in Hand Surgery.** Snowmass. 30 Cat 1 credits. Info: American Society for Surgery of the Hand, 3025 South Parker Road, Suite 65, Aurora, CO 80014. Tel: (303) 755-4588.

FLORIDA

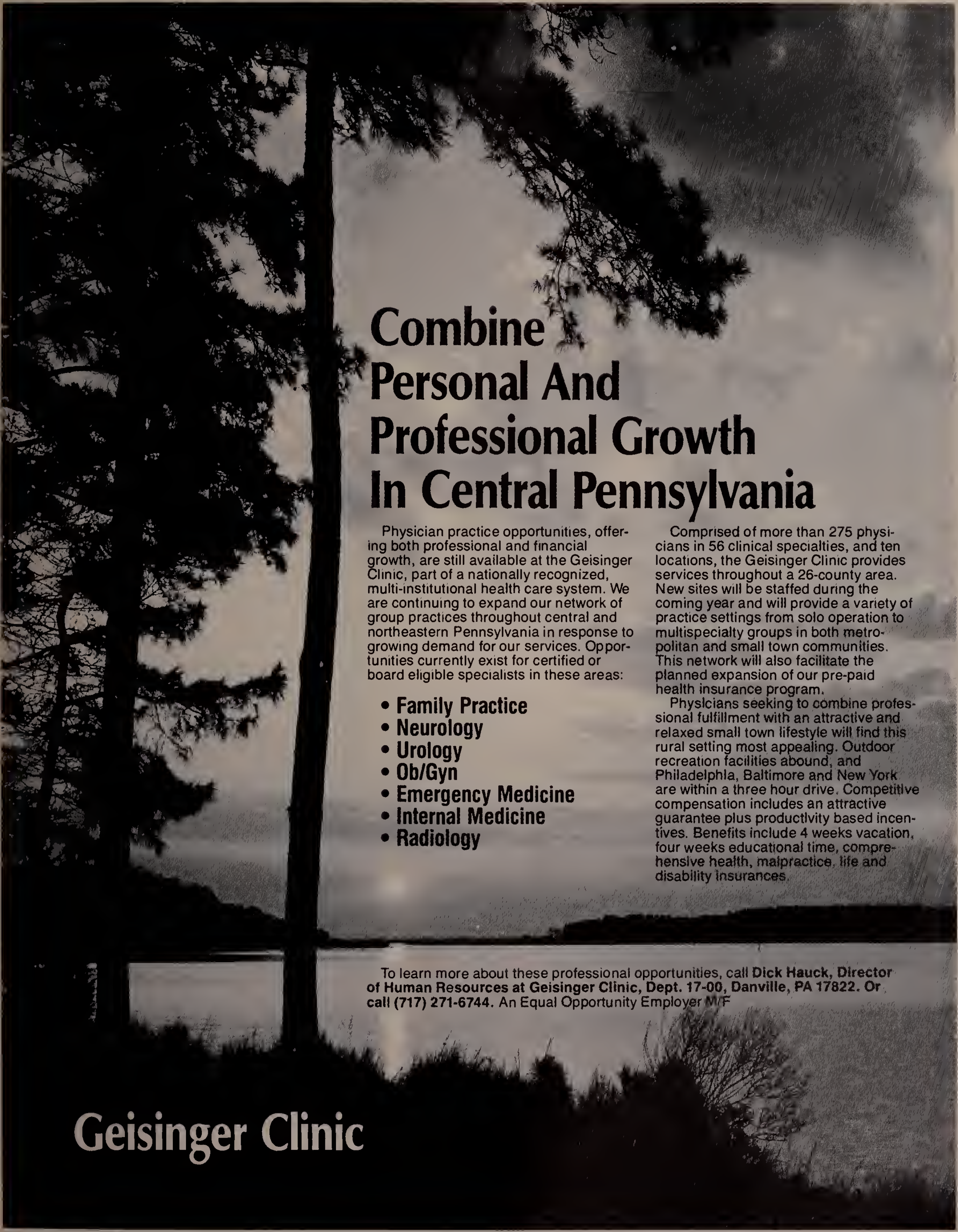
Mar 7–9. **Legal and Ethical Aspects of Health Care for the Elderly.** St. Petersburg Beach. 14 Cat 1 credits. Info: Conference Registrar, American Society of Law & Medicine, 765 Commonwealth Ave, Boston, MA 02215. Tel: (617) 262-4990.

Mar 9–12. **New Concepts in Obstetrics & Gynecology.** Kissimmee. Info: Perinatal Resources, Inc. 2410 Coventry Rd, Columbus, OH 43221. Tel: (800) 424-6903.

Mar 10–14. **Magnetic Resonance Imaging in Medical Practice.** Bal Harbour. 24 Cat 1 credits. Info: Lucy R. Kelley, Radiology Seminars, Inc. Coral Gables, FL 33134. Tel: (305) 674-2680.

Mar 15–17. **National Conference on Pediatric Lung Diseases.** Sarasota. 20 Cat 1 credits. Info: Rayna Saville, CME, Children's Hospital, 219 Bryant St, Buffalo, NY 14222. Tel: (716) 878-7630.

Mar 25–28. **A Collaborative Seminar on Practical Decisions in Surgery.** Orlando. 20 Cat 1 credits. Info: United States Section, International College of Surgeons, 1516 North Lake Shore Drive, Chicago, IL 60610.



Combine Personal And Professional Growth In Central Pennsylvania

Physician practice opportunities, offering both professional and financial growth, are still available at the Geisinger Clinic, part of a nationally recognized, multi-institutional health care system. We are continuing to expand our network of group practices throughout central and northeastern Pennsylvania in response to growing demand for our services. Opportunities currently exist for certified or board eligible specialists in these areas:

- **Family Practice**
- **Neurology**
- **Urology**
- **Ob/Gyn**
- **Emergency Medicine**
- **Internal Medicine**
- **Radiology**

Comprised of more than 275 physicians in 56 clinical specialties, and ten locations, the Geisinger Clinic provides services throughout a 26-county area. New sites will be staffed during the coming year and will provide a variety of practice settings from solo operation to multispecialty groups in both metropolitan and small town communities. This network will also facilitate the planned expansion of our pre-paid health insurance program.

Physicians seeking to combine professional fulfillment with an attractive and relaxed small town lifestyle will find this rural setting most appealing. Outdoor recreation facilities abound, and Philadelphia, Baltimore and New York are within a three hour drive. Competitive compensation includes an attractive guarantee plus productivity based incentives. Benefits include 4 weeks vacation, four weeks educational time, comprehensive health, malpractice, life and disability insurances.

To learn more about these professional opportunities, call **Dick Hauck, Director of Human Resources at Geisinger Clinic, Dept. 17-00, Danville, PA 17822. Or call (717) 271-6744.** An Equal Opportunity Employer M/F

Geisinger Clinic

Need A Temporary Physician?
During: vacations, CME's, illness, recruiting.
Want Free Time While You Practice Medicine?
Join CompHealth's Locum Tenens Physician Group.
For further information, call 412-741-3310.

CompHealth A Physician Group
WILSON ROSS, Regional Administrator
114 Centennial Ave., Sewickley, PA 15143

MANHATTAN MEDICAL
Medical professional office suites built to suit.
From 1,000 sq. ft. to 25,000 sq. ft.
For further information call: **Erwin Stern**
(212) 759-7825

IN-OFFICE SURGERY

Surgical suite design and outfitting
Staffing
Practice management

GGC Consultants
126 Bay Ridge Avenue Brooklyn, NY 11220
718-680-8166

NEEMA EMERGENCY MEDICAL
—a professional association—

Emergency Medicine Positions—available with emergency physician group in PA, NY, NJ, VA, WVA, MD, KY and throughout New England, the Southeast, the Midwest, including all suburban, rural and metropolitan areas. Fee-for-service with minimum guarantee provided. Malpractice paid. Practice credits toward board certification. Physician department directors also desired. Please send resume to: NEEMA Emergency Medical, Suite 400, 399 Market Street, Phila., PA 19106 or phone 215-925-3511 in PA, or 800-523-0776 outside PA.

Medical Society of the State of New York
ANNUAL CME ASSEMBLY
JUNE 7-9, 1985
The New York Hilton, New York City
Combined With The Annual Meeting of
The House of Delegates
JUNE 6-9

NATIONAL EMERGENCY SERVICES, INC. (N.E.S.)

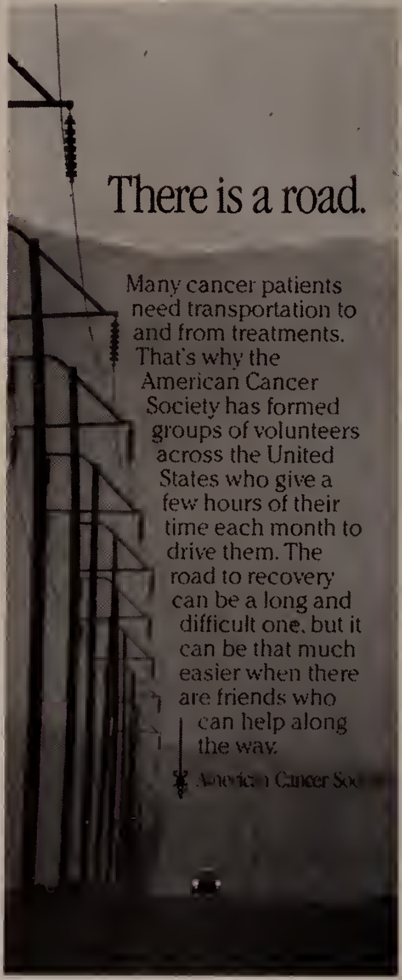
Directorships, full- and part-time emergency department opportunities are available throughout New York State and the country for primary care physicians with ER experience. Enjoy a variety of practice settings, competitive remuneration, paid malpractice Insurance, and flexible scheduling. For information and application materials contact the recruitment department at (516) 365-4010, (Outside N.Y. State 1-800-645-4848) or send C.V. to N.E.S., One Hollow Lane, Suite 304, Lake Success, N.Y. 11042.

ORTHOPEDIC SURGEON

Orthopod needed full time for my clinics in Rochester and Buffalo to perform Orthopedic Disability Evaluations.

Kick the hassle of practice, hospitals, etc. Enjoy banker's hours and maintain high income.

Write with CV to H. Lewis, M.D., 3641 Quail Hollow, Bloomfield Hills, Michigan, 48013.



Many cancer patients need transportation to and from treatments. That's why the American Cancer Society has formed groups of volunteers across the United States who give a few hours of their time each month to drive them. The road to recovery can be a long and difficult one, but it can be that much easier when there are friends who can help along the way.

This space contributed as a public service.

INDIANA

Mar 25–29. **Electrocardiographic Interpretation of Complex Arrhythmias: A Physiological Approach.** Indiana University Medical Center. Info: American College of Cardiology, Extramural Programs Department, 9111 Old Georgetown Road, Bethesda, MD 20814. Tel: (301) 897-5400, ext 230.

MASSACHUSETTS

Mar 25–29. **Fundamentals of Industrial Hygiene.** *Mar 25–29.* **Occupational and Environmental Radiation Protection.** *Mar 26–28.* **Risk Analysis in Occupational Health.** Boston. Info: Office of Continuing Education, Dept A, Harvard School of Public Health, 677 Huntington Ave, Boston, MA 02115. Tel: (617) 732-1171.

NEVADA

Mar 3–6. **Maxillofacial Trauma Symposium.** Lake Tahoe. 16 Cat 1 credits. Info: Extended Programs in Medical Education, Room 569-U, University of California, San Francisco, CA 94143. Tel: (415) 666-4251.

PENNSYLVANIA

Mar 24–29. **Family Practice Review.** Lancaster. Info: Office for CME, Temple University School of Medicine, 3400 North Broad St, Philadelphia, PA 19140. Tel: (215) 221-4787.

RHODE ISLAND

Mar 24–27. **Northeastern Conference on Alcoholism and Drug Dependence.** Newport. 15 Cat 1 credits. Info: Jane A. Drury, Edgehill Newport Foundation, Beacon Hill Rd, Newport, RI 02840. Tel: (401) 849-5700. ext 304.

ST LAWRENCE

Mar 14. **Pulmonary Function Testing.** *Mar 21.* **Calcium Blockers in Heart Disease.** Info: Sebastian A. Mazzotta, MD, Canton-Potsdam Hospital, 50 Leroy St. Potsdam, NY 13676. Tel: (315) 265-3300.

TEXAS

Mar 28–31. **Family Practice Recertification Review.** San Antonio. Info: The University of Texas Health Science Center at San Antonio Medical School, CME, 7730 Floyd Curl Drive, San Antonio, TX 78284.

AROUND THE WORLD

CANADA

Mar 11–15. **Annual Meeting of the International Academy of Pathology.** Toronto. Info: United States-Canadian Division of the International Academy of Pathology, 1003 Chafee Ave, Augusta, GA 30904. Tel: (404) 724-2973.

Mar 25–27. **Cardiovascular Conference at Lake Louise.** Alberta. 14 Cat 1 credits. Info: American College of Cardiology, Extramural Programs Dept, 9111 Old Georgetown Rd, Bethesda, MD 20814. Tel: (301) 897-5400. ext 230.

MONACO

Mar 16–24. **Pediatric Update 1985.** Monte Carlo. Info: Debra Mohr, CME, Long Island Jewish-Hillside Medical Center, New Hyde Park, NY 10040. Tel: 718-470-2114.

PUERTO RICO

Mar 8–12. **Digestive Disease Review V and Advances in Nutrition Therapy II.** San Juan. Info: Department of Medicine, Norwalk Hospital, Norwalk CT 06856.

Mar 11–15. **Current Topics in Diagnostic Radiology.** Dorado Beach. *Mar 11–15.* **Managing Clinical Problems in the Elderly.** Dorado Beach. *Mar 17–24.* **Recent Advances in Clinical Psychiatry.** Humacao. Info: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

DON'T COUNT ON THIS ALONE TO SAVE YOUR LIFE.

First-aid kits are fine for some emergencies. But what if you were choking? Having a heart attack? Or a stroke? This kit would be useless. Call the Red Cross. We'll teach you and your employees how to save a life. From work-safety to CPR, you can count on the Red Cross. **We'll help. Will you?**

A Public Service of This Publication Ad Council

YOU CAN'T FLY IF YOU'RE HIGH.

Don't let your lungs go to pot.

When you reach for a dream, whether it's in school, in sports, or on stage, you've got to be on your toes. That's why you should take a second look at marijuana and think twice before you smoke.

New studies show that smoking pot can hurt your lungs much more than you think. If you're young and growing, it's a lot more dangerous than you imagined. And a lot less cool.

Healthy lungs can mean a longer life. And a longer life means more time to make your dreams come true. Don't let your lungs to to pot.

AMERICAN LUNG ASSOCIATION
The Christmas Seal People®

Space contributed by the publisher as a public service

NEW LAW CUTS TAX BREAKS FOR LUXURY CARS

If the car you want costs more than $16,667.00 don't spend your money, use ours—we still pass the investment tax credit to you.

If you're in the 50% tax bracket no longer can you save half the depreciation on anything over $16,677.00 for the first 3 years:

HOWEVER,

When you lease you can continue to deduct your monthly rental payments in excess of $16,000.00 *provided* you conform to the limits and rules set by the I.R.S. regarding usage. The leasee must use the vehicle more than 50% of the time for his business or profession.

SO DOESN'T IT MAKE SENSE TO LEASE NOW!

ALL MAKES & MODELS AVAILABLE.

CALL CAROL LEASING INC.

(516) 222-9041

ROOSEVELT RACEWAY, WESTBURY, NY 11590

PRACTICES WANTED

OPHTHALMOLOGIST, BOARD CERTIFIED desires to purchase practice in New York City or suburban area. Confidence maintained. Please contact Dept. 168, c/o NYSJM.

YOUNG INTERNIST, wishes to purchase Nassau County office based or nursing home based private practice. Available immediately. Strictly confidential. Reply Dept. 365, c/o NYSJM.

REAL ESTATE FOR SALE OR RENT

SUBLEASE-FURNISHED MEDICAL OFFICE in a Medical Building in Eastchester, NY. Easily accessible to hospitals and parkways. Reasonable. Call (914) 779-8004.

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

REAL ESTATE FOR SALE OR RENT—CONT'D

WANTED: MEDICAL OFFICE BUILDINGS. As principals we are seeking "existing buildings"—fully tennanted by physicians, dentists and health care services. "To be built" medical facilities as a joint venture with either an institution or prominent area physician. Contact: Paul Gellert, Gelco Associates, 155 West 68th St., N.Y., N.Y. 10023; (212) 223-1130.

PSYCHIATRIST-CONSULTATION ROOM. Available full time. Evening space for group therapy. East 60's New York City. (212) 879-4355.

MEDICAL CENTER, 20 MILES NORTH OF TAMPA. Gross potential unlimited. Two or more doctors. 14,000 active patients. Laboratory, X-ray, emergency room, physical therapy. Variable terms. $450,000. New Hospital in near future. Call 1-813-985-3038 recorder.

OFFICE SPACE FOR RENT: Enjoy Adirondack Mountains; convenient to New York City, Boston and Montreal. New multi-doctor professional office soon to be design completed in growing Saratoga County, NY. Preventative oriented physician/specialist(s) preferred. Direct inquiries to Box 8801, Albany, NY 12208.

MARCO ISLAND, FLORIDA. Two-bedroom condominium, eleventh floor; on Gulf. Beautifully decorated and furnished. For lease. Call (513) 891-1263.

LAKE SUCCESS RANCH HOUSE: Golf club membership guaranteed. Near hospitals, Northern State Parkway and L.I.E., Great Neck Schools, village tennis and pool, 2-3 bedrooms, 2 baths, large eat-in kitchen, air conditioners, sprinklers, burglar alarm, fire place, 2 car garage. ¼ acre. $320,000. Owner Mr. Friedman (516) 626-1211.

RONKONKOMA AT MACARTHUR AIRPORT. 10,000 sq. ft. office building opening in June 1985. Will subdivide to 1,250 sq. ft., 2,500 sq. ft. and up. Call (516) 422-1067.

Buy Bonds

Take Stock in America.

Evidence
that all beta blockers
are not created equal.

New studies uncover the potassium effects of beta-2 blockade

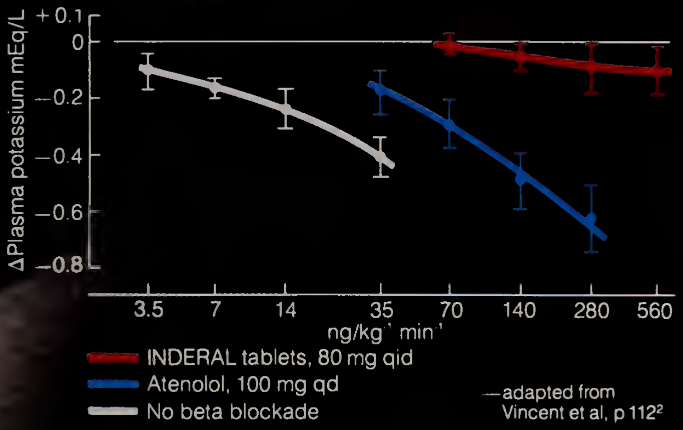
Clinical pharmacology data from the *New England Journal of Medicine:*

"...when normal young men are given infusions of epinephrine at levels such as those that circulate in patients with myocardial infarction, their serum potassium concentrations fall by about 0.8 mmol per liter. Hypokalemia is prevented by...beta-2 blockade."[1]

INDERAL (propranolol HCl) prevented beta-2 mediated hypokalemia

In a pharmacological study comparing INDERAL with atenolol, 10 hypertensive patients were infused with the nonselective beta agonist, isoproterenol, which also stimulates beta-2 mediated hypokalemia. At doses high enough to overcome beta-1 mediated heart rate reductions, the hypokalemia caused by isoproterenol was blunted by INDERAL, but not by atenolol.[2]

INDERAL compared with atenolol: change in plasma potassium after beta-agonist infusion[2]



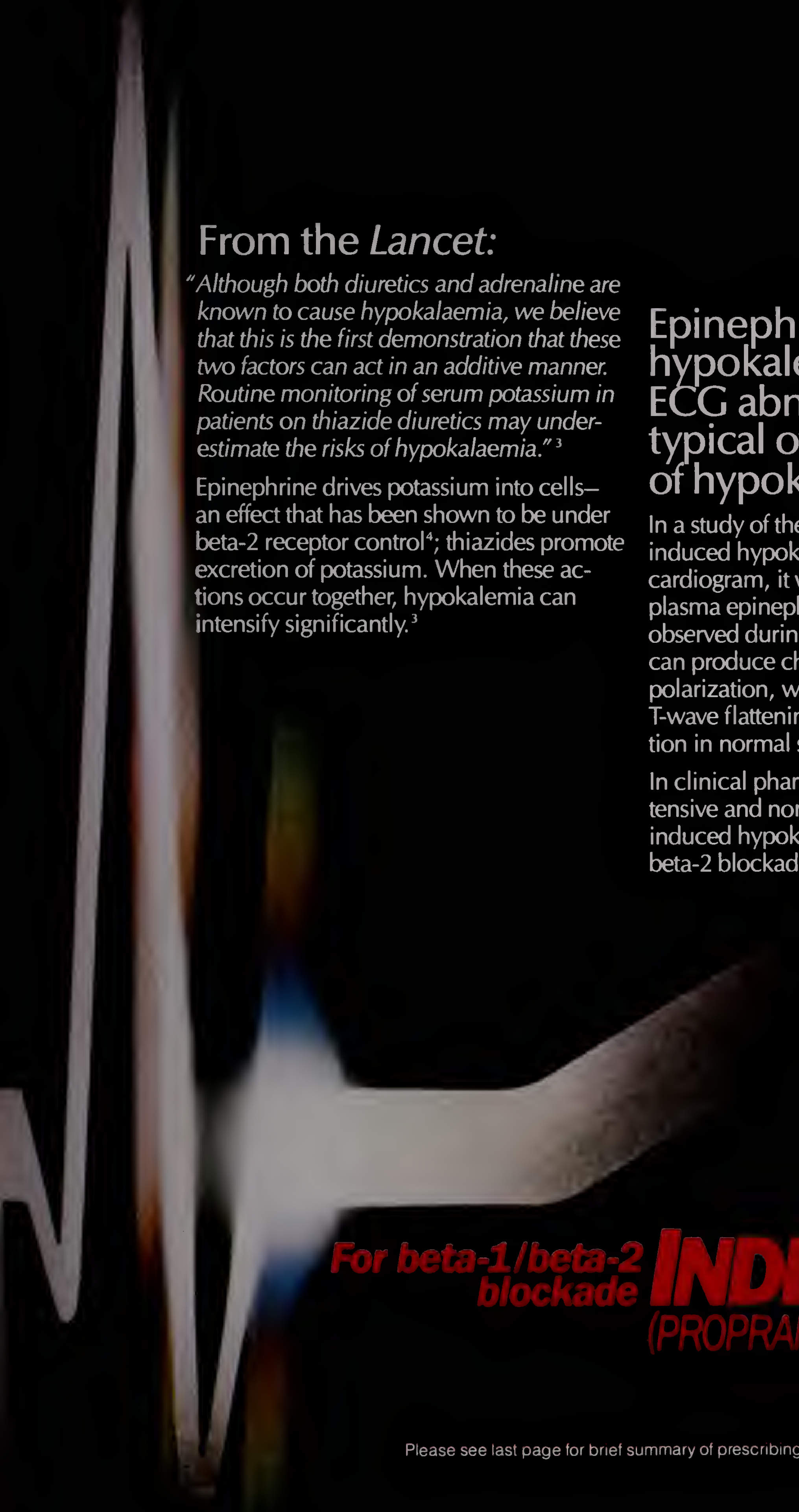
—adapted from Vincent et al, p 112[2]

For beta-1/beta-2 blockade INDERAL®

(PROPRANOLOL HCl) TABLETS

Please see last page for brief summary of prescribing information.

Epinephrine-induced hypokalemia can intensify diuretic-induced hypokalemia



From the *Lancet:*

"Although both diuretics and adrenaline are known to cause hypokalaemia, we believe that this is the first demonstration that these two factors can act in an additive manner. Routine monitoring of serum potassium in patients on thiazide diuretics may underestimate the risks of hypokalaemia." [3]

Epinephrine drives potassium into cells—an effect that has been shown to be under beta-2 receptor control[4]; thiazides promote excretion of potassium. When these actions occur together, hypokalemia can intensify significantly.[3]

Epinephrine-induced hypokalemia can cause ECG abnormalities typical of other forms of hypokalemia

In a study of the effects of epinephrine-induced hypokalemia on the electrocardiogram, it was shown that levels of plasma epinephrine similar to those observed during myocardial infarction can produce changes in ventricular repolarization, which are reflected in T-wave flattening and QT prolongation in normal subjects.[5]

In clinical pharmacology studies of hypertensive and normal patients, epinephrine-induced hypokalemia was prevented by beta-2 blockade.[2,4]

For beta-1/beta-2 blockade

INDERAL

(PROPRANOLOL HCl) TABLETS

Please see last page for brief summary of prescribing information.

Right from the start
in hypertension...

Once-daily INDERAL LA (propranolol HCl) for smooth blood pressure control without the potassium problems of diuretics

Patients with "uncomplicated" hypertension may develop ventricular arrhythmias in the presence of hypokalemia.[6] Once-daily INDERAL LA maintains smooth blood pressure reductions without a negative effect on serum potassium and provides patients with broad cardiovascular benefits—all in a convenient daily dose.

Like conventional INDERAL tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.

And for lifetime benefits in foundation treatment of angina

Once-daily INDERAL LA provides 24-hour control of angina symptoms—plus the cardiovascular benefits of the world's leading beta blocker. And unlike calcium channel blockers, INDERAL LA, alone or with a nitrate, is recommended for first-line treatment of stable angina.

Simply start new patients on 80 mg INDERAL LA once daily. Dosage may be increased to 160 mg once daily to achieve maximal control.

Once-daily

INDERAL® LA

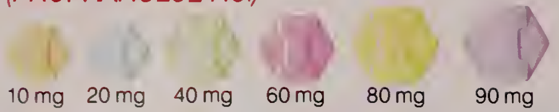
(PROPRANOLOL HCl) LONG ACTING CAPSULES

In HYPERTENSION

and ANGINA

The appearance of these capsules is a registered trademark of Ayerst Laboratories.
Please see last page for brief summary of prescribing information.

INDERAL®
(PROPRANOLOL HCl) TABLETS



The appearance of these tablets is a registered trademark of Ayerst Laboratories.

Once-daily
INDERAL® LA
(PROPRANOLOL HCl) LONG ACTING CAPSULES

The appearance of these capsules is a registered trademark of Ayerst Laboratories.

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION. SEE PACKAGE CIRCULARS.)*

INDERAL® (propranolol hydrochloride) **Tablets and Injectable**

INDERAL® LA (propranolol hydrochloride) **Long Acting Capsules**

INDICATIONS AND USAGE—INDERAL Tablets

Hypertension: INDERAL (propranolol hydrochloride) is indicated in the management of hypertension. It may be used alone or used in combination with other antihypertensive agents particularly a thiazide diuretic. INDERAL is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL is indicated for the long-term management of patients with angina pectoris.

Cardiac Arrhythmias:

1.) Supraventricular arrhythmias

a) Paroxysmal atrial tachycardias, particularly those arrhythmias induced by catecholamines or digitalis or associated with the Wolff-Parkinson-White syndrome. (See W-P-W under WARNINGS.) b) Persistent sinus tachycardia which is noncompensatory and impairs the well-being of the patient. c) Tachycardias and arrhythmias due to thyrotoxicosis when causing distress or increased hazard and when immediate effect is necessary as adjunctive, short term (2-4 weeks) therapy. May be used with, but not in place of, specific therapy. (See Thyrotoxicosis under WARNINGS.) d) Persistent atrial extrasystoles which impair the well-being of the patient and do not respond to conventional measures. e) Atrial flutter and fibrillation when ventricular rate cannot be controlled by digitalis alone, or when digitalis is contraindicated.

2.) Ventricular tachycardias

Ventricular arrhythmias do not respond to propranolol as predictably as do the supraventricular arrhythmias. a) Ventricular tachycardias. With the exception of those induced by catecholamines or digitalis, INDERAL is not the drug of first choice. In critical situations when cardioversion technics or other drugs are not indicated or are not effective, INDERAL may be considered. If, after consideration of the risks involved, INDERAL is used, it should be given intravenously in low dosage and very slowly. (See DOSAGE AND ADMINISTRATION.) *Care in the administration of INDERAL with constant electrocardiographic monitoring is essential as the failing heart requires some sympathetic drive for maintenance of myocardial tone.* b) Persistent premature ventricular extrasystoles which do not respond to conventional measures and impair the well-being of the patient.

3.) Tachyarrhythmias of digitalis intoxication

If digitalis-induced tachyarrhythmias persist following discontinuance of digitalis and correction of electrolyte abnormalities, they are usually reversible with *oral* INDERAL. Severe bradycardia may occur. (See OVERDOSAGE.) Intravenous propranolol hydrochloride is reserved for life-threatening arrhythmias. Temporary maintenance with oral therapy may be indicated. (See DOSAGE AND ADMINISTRATION in the package circulars.)

4.) Resistant tachyarrhythmias due to excessive catecholamine action during anesthesia. Tachyarrhythmias due to excessive catecholamine action during anesthesia may sometimes arise because of release of endogenous catecholamines or administration of catecholamines. When usual measures fail in such arrhythmias, INDERAL may be given intravenously to abolish them. All general inhalation anesthetics produce some degree of myocardial depression. Therefore, when INDERAL (propranolol hydrochloride) is used to treat arrhythmias during anesthesia, it should be used with extreme caution and constant ECG and central venous pressure monitoring. (See WARNINGS.)

Myocardial Infarction: INDERAL is indicated to reduce cardiovascular mortality in patients who have survived the acute phase of myocardial infarction and are clinically stable.

Migraine: INDERAL is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta receptor stimulation. Clinical improvement may be temporary.

Pheochromocytoma: After primary treatment with an alpha-adrenergic blocking agent has been instituted, INDERAL may be useful as *adjunctive* therapy if the control of tachycardia becomes necessary before or during surgery.

It is hazardous to use INDERAL unless alpha-adrenergic blocking drugs are already in use, since this would predispose to serious blood pressure elevation. Blocking only the peripheral dilator (beta) action of epinephrine leaves its constrictor (alpha) action unopposed.

In the event of hemorrhage or shock, there is a disadvantage in having both beta and alpha blockade since the combination prevents the increase in heart rate and peripheral vasoconstriction needed to maintain blood pressure.

With inoperable or metastatic pheochromocytoma, INDERAL may be useful as an adjunct to the management of symptoms due to excessive beta receptor stimulation.

INDICATIONS AND USAGE—INDERAL LA Long Acting Capsules

Hypertension: INDERAL LA (propranolol hydrochloride) is indicated in the management of hypertension. It may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

CONTRAINDICATIONS. INDERAL and INDERAL LA are contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block; 3) bronchial asthma. 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL.

WARNINGS. CARDIAC FAILURE. Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL (propranolol hydrochloride) therapy. Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)— PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL, like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e.g., dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA: Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin.

THYROTOXICOSIS. Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

PRECAUTIONS. *General:* Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL is not indicated for the treatment of hypertensive emergencies.

Beta adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

Clinical Laboratory Tests. Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

Pregnancy: Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: bradycardia, congestive heart failure; intensification of AV block; hypotension; paresthesia of hands; thrombocytopenic purpura; arterial insufficiency, usually of the Raynaud type.

Central Nervous System: Lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances; hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics.

Gastrointestinal: nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: bronchospasm.

Hematologic: agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-Immune. In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

REFERENCES.

1. Epstein FH, Rosa RM. Adrenergic control of serum potassium. *N Engl J Med* 1983;309 1450-1451. **2.** Vincent HH, Boomsma F, Man in't Veld AJ, et al: Effects of selective and nonselective β-agonists on plasma potassium and norepinephrine. *J Cardiovasc Pharmacol* 1984;6:107-114. **3.** Struthers AD, Whitesmith R, Reid JL: Prior thiazide diuretic treatment increases adrenaline-induced hypokalaemia. *Lancet* 1983;1.1358-1361. **4.** Brown MJ, Brown DC, Murphy MB: Hypokalemia from beta$_2$-receptor stimulation by circulating epinephrine. *N Engl J Med* 1983;309:1414-1419. **5.** Struthers AD, Reid JL, Whitesmith R, et al: Effect of intravenous adrenaline on electrocardiogram, blood pressure, and serum potassium. *Br Heart J* 1983;49.90-93. **6.** Holland OB, Nixon JV, Kuhnert L: Diuretic-induced ventricular ectopic activity. *Am J Med* 1981;70:762-768.

9410/1184

Ayerst®
AYERST LABORATORIES
New York, N.Y. 10017

Copyright © 1984 AYERST LABORATORIES
Division of AMERICAN HOME PRODUCTS CORPORATION

Angina comes in many forms...

So does SORBITRATE®

(ISOSORBIDE DINITRATE)

Unsurpassed flexibility in nitrate therapy.

© 1985 ICI AMERICAS INC.

See following page for brief summary of prescribing information.

SORBITRATE®
(ISOSORBIDE DINITRATE)

Please consult full prescribing information before use. A summary follows:

INDICATIONS AND USAGE: SORBITRATE (isosorbide dinitrate) is indicated for the treatment and prevention of angina pectoris All dosage forms of isosorbide dinitrate may be used prophylactically to decrease frequency and severity of anginal attacks and can be expected to decrease the need for sublingual nitroglycerin

The sublingual and chewable forms of the drug are indicated for acute prophylaxis of angina pectoris when taken a few minutes before situations likely to provoke anginal attacks Because of a slower onset of effect, the oral forms of isosorbide dinitrate are not indicated for acute prophylaxis

CONTRAINDICATIONS: SORBITRATE is contraindicated in patients who have shown purported hypersensitivity or idiosyncrasy to it or other nitrates or nitrites Epinephrine and related compounds are ineffective in reversing the severe hypotensive events associated with overdose and are contraindicated in this situation.

WARNINGS: The benefits of SORBITRATE during the early days of an acute myocardial infarction have not been established If one elects to use organic nitrates in early infarction, hemodynamic monitoring and frequent clinical assessment should be used because of the potential deleterious effects of hypotension.

PRECAUTIONS: General: Severe hypotensive response, particularly with upright posture, may occur with even small doses of SORBITRATE The drug should therefore be used with caution in subjects who may have blood volume depletion from diuretic therapy or in subjects who have low systolic blood pressure (eg, below 90 mmHg). Paradoxical bradycardia and increased angina pectoris may accompany nitrate-induced hypotension. Nitrate therapy may aggravate the angina caused by hypertrophic cardiomyopathy

Marked symptomatic, orthostatic hypotension has been reported when calcium channel blockers and organic nitrates were used in combination Dose adjustment of either class of agents may be necessary

Tolerance to this drug and cross-tolerance to other nitrates and nitrites may occur Tolerance to the vascular and antianginal effects of isosorbide dinitrate or nitroglycerin has been demonstrated in clinical trials, experience through occupational exposure, and in isolated tissue experiments in the laboratory The importance of tolerance to the appropriate use of isosorbide dinitrate in the management of patients with angina pectoris has not been determined However, one clinical trial using treadmill exercise tolerance (as an end point) found an 8-hour duration of action of oral isosorbide dinitrate following the first dose (after a 2-week placebo washout) and only a 2-hour duration of effect of the same dose after 1 week of repetitive dosing at conventional dosing intervals. On the other hand, several trials have been able to differentiate isosorbide dinitrate from placebo after 4 weeks of therapy and, in open trials, an effect seems detectable for as long as several months.

Tolerance clearly occurs in industrial workers continuously exposed to nitroglycerin. Moreover, physical dependence also occurs since chest pain, acute myocardial infarction, and even sudden death have occurred during temporary withdrawal of nitroglycerin from the workers In clinical trials in angina patients, there are reports of anginal attacks being more easily provoked and of rebound in the hemodynamic effects soon after nitrate withdrawal. The relative importance of these observations to the routine, clinical use of isosorbide dinitrate is not known However, it seems prudent to gradually withdraw patients from isosorbide dinitrate when the therapy is being terminated, rather than stopping the drug abruptly

Information for Patients: Headache may occur during initial therapy with SORBITRATE Headache is usually relieved by the use of standard headache remedies or by lowering the dose and tends to disappear after the first week or two of use

Drug Interactions: Alcohol may enhance any marked sensitivity to the hypotensive effect of nitrates.

Isosorbide dinitrate acts directly on vascular smooth muscle, therefore, any other agent that depends on vascular smooth muscle as the final common path can be expected to have decreased or increased effect depending on the agent

Carcinogenesis, Mutagenesis, Impairment of Fertility: No long-term studies in animals have been performed to evaluate the carcinogenic potential of this drug A modified two-litter reproduction study in rats fed isosorbide dinitrate at 25 or 100 mg/kg/day did not reveal any effects on fertility or gestation or any remarkable gross pathology in any parent or offspring fed isosorbide dinitrate as compared with rats fed a basal-controlled diet

Pregnancy Category C: Isosorbide dinitrate has been shown to cause a dose-related increase in embryotoxicity (increase in mummified pups) in rabbits at oral doses 35 and 150 times the maximum recommended human daily dose There are no adequate and well-controlled studies in pregnant women. SORBITRATE should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: It is not known whether this drug is excreted in human milk Because many drugs are excreted in human milk, caution should be exercised when SORBITRATE is administered to a nursing woman

Pediatric Use: The safety and effectiveness of SORBITRATE in children has not been established

ADVERSE REACTIONS: Adverse reactions, particularly headache and hypotension, are dose-related In clinical trials at various doses, the following have been observed

Headache is the most common (reported incidence varies widely, apparently being dose-related, with an average occurrence of about 25%) adverse reaction and may be severe and persistent Cutaneous vasodilation with flushing may occur Transient episodes of dizziness and weakness, as well as other signs of cerebral ischemia associated with postural hypotension, may occasionally develop (the incidence of reported symptomatic hypotension ranges from 2% to 36%) An occasional individual will exhibit marked sensitivity to the hypotensive effects of nitrates and severe responses (nausea, vomiting, weakness, restlessness, pallor, perspiration, and collapse) may occur even with the usual therapeutic dose. Drug rash and/or exfoliative dermatitis may occasionally occur Nausea and vomiting appear to be uncommon. Case reports of clinically significant methemoglobinemia are rare at conventional doses of organic nitrates The formation of methemoglobin is dose-related and, in the case of genetic abnormalities of hemoglobin that favor methemoglobin formation, even conventional doses of organic nitrate could produce harmful concentrations of methemoglobin.

DOSAGE AND ADMINISTRATION: For the treatment of angina pectoris, the usual starting dose for sublingual SORBITRATE is 2.5 to 5 mg, for chewable tablets, 5 mg, for oral (swallowed) tablets, 5 to 20 mg and for controlled-release forms, 40 mg

SORBITRATE should be titrated upward until angina is relieved or side effects limit the dose In ambulatory patients, the magnitude of the incremental dose increase should be guided by measurements of standing blood pressure

The initial dosage of sublingual or chewable SORBITRATE for prophylactic therapy in angina pectoris patients is generally 5 or 10 mg every 2 to 3 hours Adequate controlled clinical studies demonstrating the effectiveness of chronic maintenance therapy with these dosage forms have not been reported

SORBITRATE in oral doses of 10 to 40 mg given every 6 hours or in oral controlled-release doses of 40 to 80 mg given every 8 to 12 hours is generally recommended The extent to which development of tolerance should modify the dosage program has not been defined *The oral controlled-release forms of isosorbide dinitrate should not be chewed*

DOSAGE FORMS AVAILABLE: Sublingual Tablets (2 5, 5, 10 mg), Chewable Tablets (5, 10 mg): Oral Tablets (5, 10, 20, 30, 40 mg), Sustained Action Tablets (40 mg)

STUART PHARMACEUTICALS
Division of ICI Americas Inc.
Wilmington, DE 19897

STR-2282

PRACTICES AVAILABLE

ESTABLISHED SOLO FAMILY PRACTICE: Opportunity to assume established private practice in pleasant college community in Northwestern Pennsylvania. Fully equipped office and family residence available. Admitting privileges at major Medical Center, which will assist with relocation and placement of qualified Board Eligible/Certified Family Practice Physician. Call Tina Carrigan (215) 896-5080.

FOR SALE-ACTIVE PRACTICE INTERNAL MEDICINE. Established, solo, since 1954, Garden City, Long Island. Several local hospitals nearby. Negotiable. Retiring for personal reasons. Reply Dept. 378 c/o NYSJM.

INTERNAL MEDICINE practice for sale. Well-established in Nassau County. Home-office, fully equipped for one or two physicians. Telephone (516) 747-3072.

MEDICAL PRACTICES FOR SALE—Are you contemplating selling or buying a medical practice? We can help. PRACTICE FINDERS, INC. 45 East 89th Street, New York, NY 10028, Phone (212) 860-2206.

ESTABLISHED INTERNAL MEDICINE PRACTICE FOR SALE: Semi-rural area, 2 hours from Philadelphia, Baltimore and Washington, D.C. Unique buy out opportunity. Details of sale provided upon request. Contact: Edward F. Quinn, III, M.D. or Catherine Messick, 800 North duPont Highway, Milford, Delaware 19963. Phone (302) 422-3071.

MISCELLANEOUS

PATIENT NEWSLETTER: Show patients you care by keeping in touch! Send them our quarterly health promotion newsletter. As low as $1 per year. Customized with your name, address and optional office column. Mailing service. Free sample. RX: LIVE WELL, 6809 Santa Maria Lane, Dallas, Texas 75214-2847, or call (214) 821-5218.

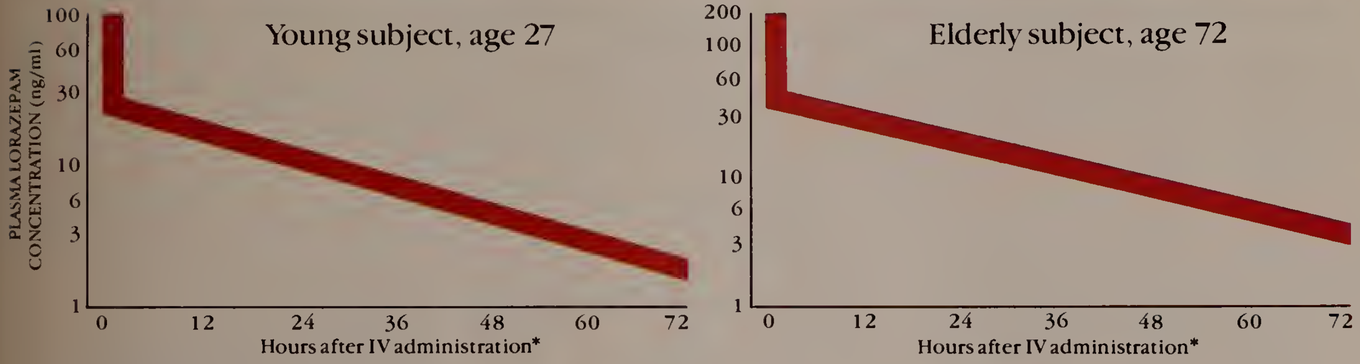
ARCHITECTURE & PLANNING, Comprehensive services from analysis of alternative office locations through design and construction. Experienced in both new construction and renovation. Lewis & Gould (212) 807-6588.

"CONSERVATIVE OPTION STRATEGIES FOR THE PRUDENT INVESTOR" Writing options on stocks or government securities can be utilized to enhance portfolio yields and provide protection in a declining market. Learn how advanced computer techniques can be applied in developing attractive conservative strategies for your portfolio. To discuss your requirements and to find out if options are an appropriate element for your investment strategy, call David Turetsky, Prudential-Bache Securities, at 800-223-2750 or (212) 697-6400 for further information and the Basic Options Disclosure Document which discusses the risks and uses of listed options.

TAX ACCOUNTANT-ATTORNEY, formerly with I.R.S., announces his availability to prepare taxes, accounting services, advise on tax shelters, pension plans and represent buyers or sellers of medical practices. I have other doctor clients. Gerald Goldberg, (212) 406-1156 and (516) 365-5679.

Among leading benzodiazepines, only Ativan has proof... pharmacokinetics not significantly altered by age.[1]

Representative charts of comparison testing



Lorazepam is nearly 100 percent bioavailable by the intramuscular and oral routes, as compared to the intravenous. Therefore, data for clearance of intravenous lorazepam are equally applicable to oral lorazepam.

- **Clearance not significantly delayed by age, liver or kidney dysfunction**
- **Cumulative sedative effects seldom a problem**
- **Available in 0.5-mg tablets to facilitate the recommended geriatric starting dosage**

1. Greenblatt DJ: Clinical study, pharmacokinetics and bioavailability in the elderly, Ativan® (lorazepam). Data on file, Wyeth Laboratories.

*Fourteen subjects, aged 60 to 84 years, participated in the study. Twelve subjects, aged 19 to 32 years, served as "young controls." Subject dosage was adjusted for body weight and ranged from 1.5 mg to 3.0 mg of lorazepam. Within the study, lorazepam clearance was monitored following IV, IM and oral administration in the elderly group and following IV administration in the control group. The effect of aging on total clearance of lorazepam was relatively small and not statistically significant. Half-life values following the three different routes of administration were essentially identical.

Only
Ativan®
(lorazepam) CIV

Wyeth Laboratories
Philadelphia, PA 19101

© 1985, Wyeth Laboratories.

See important information on following page.

The appearance of these tablets is a registered trademark of Wyeth Laboratories

® DISTINCTIVE DESIGN 0.5, 1.0 and 2.0 mg

Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, wam patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Transient amnesia or memory impairment has been reported in association with the use of benzodiazepines.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® Ⓒ for (lorazepam) Anxiety

DOSAGE: Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.

HOW SUPPLIED: 0.5, 1.0 and 2.0mg tablets.

Wyeth Laboratories
Philadelphia, PA 19101
™

MISCELLANEOUS—CONT'D

DOCTOR, YOU CAN'T BEAT the quality or the price! Holter Monitor Scanning Service. Physician owned, trained and supervised. No contracts to sign. We can arrange for lease/purchase of Holter equipment. Why are you paying more and getting less? Now using UP Service for faster turn around time. DCG Interpretation (313) 879-8860.

TAX ATTORNEY AND PENSION PLAN Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professional incorporation—No insurance required—references upon request—Jack Wachstock, PC, 3000 Marcus Ave., Lake Success, N.Y. 11530 (516) 437-4848.

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate, with no points, fees or charges of any kind. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

MEDSTAR: THE CUSTOMIZED COMPUTER SYSTEM for medical office management. Designed by a physician and his staff. Completely customized for each practice. Affordable, Reliable, Easy to use. Leasing available. Medstar, 363 Route 111, Smithtown, NY 11787. (516) 360-1422.

ACUPUNCTURE REVIEW FOR FIRST NATIONAL CERTIFICATION and New Jersey Certification Examinations. February 23–March 3, 1985, conducted by Dr. Ralph Alan Dale in New Jersey. Course approved toward Certification by New York Boards for Medicine and Dentistry. Contact Nancy Whelan, R.P.T., 4216 D Palm Bay Circle, West Palm Beach, Florida 33406 or call (305) 683-0815.

ARCHITECTURAL DESIGN AND CONSTRUCTION: Designers, builders of custom professional offices. Consulting services available. Whether you're opening a new office, relocating, remodeling or expanding an existing office in New York City area and adjacent New Jersey counties. Donald Young, Inc. Tel: (718) 948-5191.

HAVING A PROBLEM WITH ALCOHOL AND/OR DRUGS. Special AA Meeting for Doctors April 11th to April 15th, 1985, Govenor Morris Inn, Morristown, N.J. For information write: Secretary, 86 Miller Road, Morristown, N.J. 07906. Anonymity Preserved!

You know Robitussin®, and the "DAC" formula gives you just what you need to manage those severe coughs complicated with nasal congestion . . . Decongestant And Codeine!

Robitussin®-DAC Ⓥ

Each 5 ml (1 teaspoonful) contains:

Guaifenesin, USP	100 mg
Pseudoephedrine Hydrochloride, USP	30 mg
Codeine Phosphate, USP (Warning: May be habit forming)	10 mg

Alcohol 1.4 percent

℞ ONLY

A-H-ROBINS
Pharmaceutical Division
Richmond, Virginia 23261-6609

Angina Protection with Benefits for a Lifetime

ONCE-DAILY CONTROL WITH HEART-SAVING BENEFITS

By reducing heart rate and cardiac contractility, INDERAL LA helps protect the heart from the potentially serious and debilitating consequences of ischemia. A highly effective antianginal agent for around-the-clock control of symptoms, INDERAL LA also provides cardiovascular protection for a sense of security in the years ahead.

PROTECTION AND EXPERIENCE NO CALCIUM BLOCKER CAN MATCH

Unlike calcium blockers, INDERAL LA—either alone or with a nitrate—is recommended for early treatment of angina in the majority of patients. Equally important, INDERAL LA delivers the proven performance and safety profile of INDERAL tablets—confirmed by millions of patients during 16 years of clinical use.

START WITH 80 MG ONCE DAILY

Dosage may be increased to 160 mg once daily, as needed, to achieve optimal control. INDERAL LA should not be used in congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma. Please see next page for further details and brief summary of prescribing information.

ONCE-DAILY **INDERAL LA**
(PROPRANOLOL HCl) *LONG ACTING CAPSULES*

The appearance of INDERAL LA capsules is a registered trademark of Ayerst Laboratories

Ayerst.

JUST ONCE EACH DAY
FOR SIMPLIFIED CORE
THERAPY IN ANGINA

ONCE-DAILY
INDERAL® LA
(PROPRANOLOL HCl) LONG ACTING CAPSULES

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*
INDERAL*® LA brand of propranolol hydrochloride **(Long Acting Capsules)**
DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride. Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules.
CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period.

The mechanism of the antihypertensive effect of INDERAL has not been established Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain. Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use. Effects on plasma volume appear to be minor and somewhat variable. INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients.

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction. Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period. The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential The significance of the membrane action in the treatment of arrhythmias is uncertain.

The mechanism of the antimigraine effect of propranolol has not been established Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, as a result of pathologic or functional changes, sympathetic activity is detrimental to the patient. But there are also situations in which sympathetic stimulation is vital For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction. Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

Propranolol is not significantly dialyzable.
INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary
CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL
WARNINGS. CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g , dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA. Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes In these patients, it may be more difficult to adjust the dosage of insulin.

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case this resulted after an initial dose of 5 mg propranolol
PRECAUTIONS. *General.* Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL is not indicated for the treatment of hypertensive emergencies.

Beta adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure.

Clinical Laboratory Tests: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy: Pregnancy Category C INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose There are no adequate and well-controlled studies in pregnant women INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman

Pediatric Use: Safety and effectiveness in children have not been established
ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: bradycardia, congestive heart failure; intensification of AV block, hypotension; paresthesia of hands; thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type.

Central Nervous System: lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances; hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics

Gastrointestinal: nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic: pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory: bronchospasm.

Hematologic: agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.
DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels. Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION—*Dosage must be individualized* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS).

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis. If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

*The appearance of INDERAL LA capsules is a registered trademark of Ayerst Laboratories

8833/384

Ayerst.
AYERST LABORATORIES
New York, N.Y. 10017

BALANCED CALCIUM CHANNEL BLOCKADE!

CARDIZEM®
(diltiazem HCl)
balances potent coronary vasodilation with a low incidence of side effects

Low incidence of side effects
CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

Reduces angina attack frequency*
42% to 46% decrease reported in multicenter study.[1]

Increases exercise tolerance*
In Bruce exercise test,[2] control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes ($P<.005$).

CARDIZEM®
(diltiazem HCl)

THE BALANCED CALCIUM CHANNEL BLOCKER

Please see full prescribing information on following page.

2/84

---

*Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

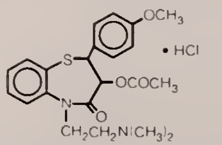
References:
1. Strauss WE, McIntyre KM, Parisi AF, et al: Safety and efficacy of diltiazem hydrochloride for the treatment of stable angina pectoris: Report of a cooperative clinical trial. Am J Cardiol 49:560-566, 1982.
2. Pool PE, Seagren SC, Bonanno JA, et al: The treatment of exercise-inducible chronic stable angina with diltiazem: Effect on treadmill exercise. Chest 78 (July suppl):234-238, 1980.

PROFESSIONAL USE INFORMATION

cardizem®
(diltiazem HCl)
30 mg and 60 mg tablets

DESCRIPTION

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one,3-(acetyloxy)-5-[2-(dimethylamino)ethyl]-2,3-dihydro-2-(4-methoxyphenyl)-, monohydrochloride,(+)-cis-. The chemical structure is:



Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform. It has a molecular weight of 450.98. Each tablet of CARDIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration.

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth muscle.

Mechanisms of Action. Although precise mechanisms of its antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways:

1. Angina Due to Coronary Artery Spasm: CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial and subendocardial. Spontaneous and ergonovine-induced coronary artery spasm are inhibited by CARDIZEM.
2. Exertional Angina: CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal and maximal exercise work loads.

In animal models, diltiazem interferes with the slow inward (depolarizing) current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Diltiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance.

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricular conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load. Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect, cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of diltiazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by diltiazem.

Intravenous diltiazem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Diltiazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, diltiazem significantly prolongs sinus cycle length (up to 50% in some cases).

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Diltiazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40%. CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 60 mg are given; a 120-mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem.

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm.** CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina).** CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia.
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing.
Cardiac Failure	Administer inotropic agents (isoproterenol, dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

The oral/LD_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort acute anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy**—CARDIZEM may be safely coadministered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

Cardizem 30-mg tablets are supplied in bottles of 100 (NDC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1771-49). Each green tablet is engraved with MARION on one side and 1771 engraved on the other. CARDIZEM 60-mg scored tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1772-49). Each yellow tablet is engraved with MARION on one side and 1772 on the other.

Issued 4/1/84

Another patient benefit product from
PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MISSOURI 64137

From Blue Cross and Blue Shield of Greater N.Y.

The last claim form!

Available with the new IBM® PC-AT!

Introducing

Amicus 1™

MEDICAL OFFICE MANAGEMENT SYSTEM

A computer system that does something you could never do before

For over 50 years, Blue Cross and Blue Shield of Greater New York has been the most consistently innovative force in helping physicians and patients meet the financial demands of progressive medical care. Now, with advances in the technology of computer networking, we can help you do something never before possible. Amicus 1 lets you transmit claims directly by phone to our computer for faster, more efficient processing of our medical and Medicare B* claims.

Direct communication provides better service

Just as direct communication between people is preferred for accuracy, computer-to-computer communication reduces the potential for errors and delays. When you purchase Amicus 1, you gain significant benefits. Faster collections and fewer errors. Greater productivity. And significant relief of cash flow pain.

Handles most of your accounting and financial management functions

Of course, Amicus 1 does more than just send claims to our computer. It assists you in all your billing and accounts receivable needs. It provides up to the minute financial reports on your practice. And more. All with just a few short entries.

A complete system featuring the IBM® Personal Computer XT

We've developed a ready-to-use system featuring an IBM Personal Computer XT. In addition, Amicus 1 includes a high speed printer; a tape backup storage unit; and a fast modem for telephone data transmission and access to medical and financial services. The Amicus 1 system includes installation with a three-day training program for your office staff. It also includes an on-site service contract plus a special telephone response line available during business hours.

Fill out your last claim form.

Just complete and mail the coupon below. Or, call us at **212 490-4180** to learn how Amicus 1 can help reduce your paperwork, increase your productivity and improve your cash flow.

Tell me how Amicus 1 can work for my practice.
☐ Yes, I'd like to see a personal demonstration.
☐ Please send me more information. NYSJM

1. NAME

2. SPECIALTY

3. ADDRESS *(Street, city, state, ZIP code)*

4. TELEPHONE ( )

5. OFFICE MANAGER

Send completed form to: **Medical Business Services**
P.O. Box 1707 Grand Central Station
New York, N.Y. 10163

Medical Business Services
Div. of Blue Cross. Blue Shield. of Greater New York

*Applies to the 16 counties where Blue Cross and Blue Shield of Greater N.Y. is the Medicare carrier
IBM® is a registered trademark of International Business Machines Corporation
Amicus 1™ is a trademark of Blue Cross and Blue Shield of Greater New York

Motrin®

ibuprofen, Upjohn

600 mg Tablets

Upjohn

©1984 The Upjohn Company

The Upjohn Company • Kalamazoo, Michigan 49001 USA

J 4044 January 1984

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

MARCH 1985 • Volume 85, Number 3

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

AN APPRECIATION

Milton Rosenberg, MD, 1923–1985
An Indefatigable Spirit

M.D.S.

Contents

COMMENTARIES

Hodgkin's disease in siblings 91
NICHOLAS J. VIANNA, MD

Adult-onset Still's disease 92
DANIEL G. BAKER, MD

Arteriovenous malformation of the stomach 93
LAWRENCE M. SHERMAN, MD

"Gerioscopy" 94
LYNN P. CARMICHAEL, MD

RESEARCH PAPERS

Tubal sterilization with the Falope ring in an ambulatory-care surgical facility 98
STEVEN G. KAALI, MD, ROBERT LANDESMAN, MD

Chlorpromazine in experimental gastric ulcers induced by restraint and cold stress 101
CHARLES C. CANVER, MD

REVIEW ARTICLE

The negligible role of ethylene dibromide in overall human cancer risk 103
JOHN H. WEISBURGER, PHD, MD

CORRELATION CONFERENCE IN RADIATION AND PATHOLOGY

A pelvic mass with periosteal reaction 106

CASE REPORTS

Hodgkin's disease in half-siblings 109
RICHARD J. HIRSCHMAN, MD, MYRON P. NOBLER, MD

Adult-onset Still's disease and inflammatory orbital pseudotumor 110
JOHN J. CUSH, MD, IAN H. LEIBOWITZ, MD, SANDOR A. FRIEDMAN, MD

Massive gastric hemorrhage from the rupture of arterial malformation 112
PIN KIE TENG, MD, SAM CHO, MD

Amylase in serous ovarian neoplasms 115
GITA RAMASWAMY, MD, VENKITA JAGADHA, MD, VICTOR TCHERTKOFF, MD

Gruntzig balloon dilation for fibromuscular dysplasia of the internal carotid arteries 113
ETHAN L. WELCH, MD, JOYCE A. LEMKIN, MD, PHD, JOSEPH E. GEARY, MD

LETTERS TO THE EDITOR

Recurrent malignant melanoma following a 46-year disease-free interval 95
LEON ROSENKRANZ, MD, CONRAD SCHROEDER, MD

Solitary nonparasitic liver cysts 95
STEVEN M. MILLER, MD, ALISAN B. GOLDFARB, MD, ZVI ZISBROD, MD

Psychiatric approach to drug abuse 96
DAVID W. BROOK, MD

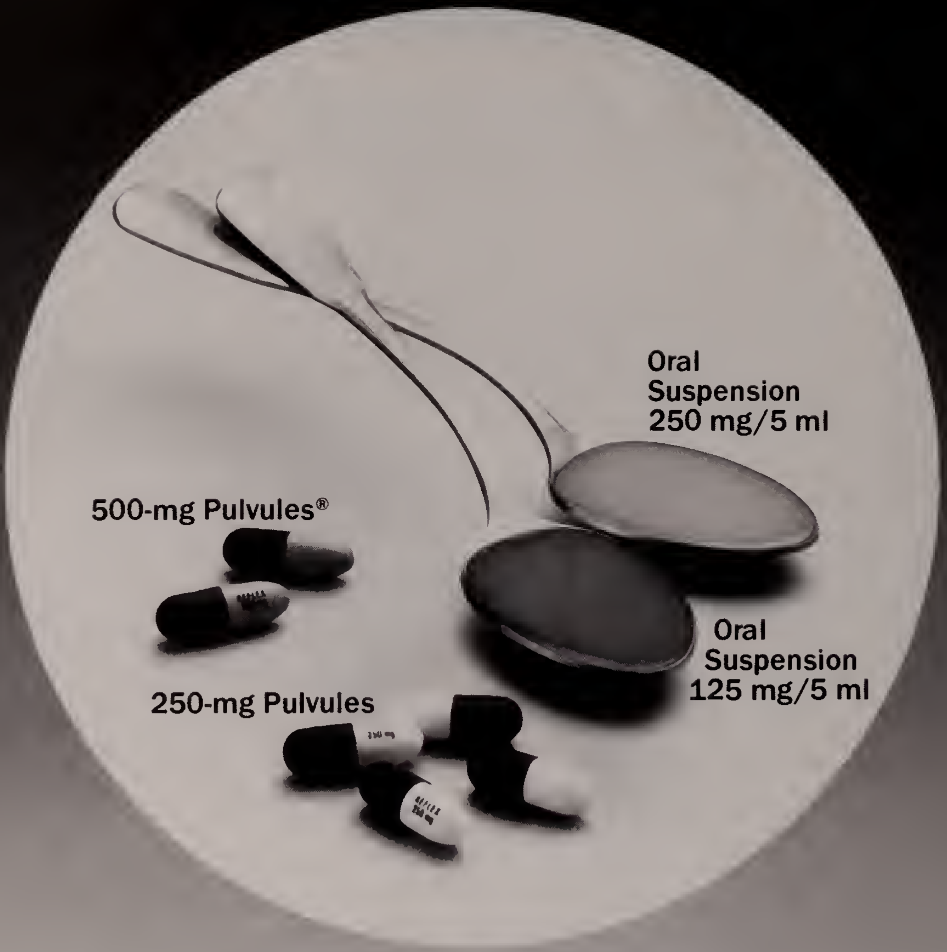
***Candida tropicalis* papillary necrosis treated with ketoconazole** 97
PAUL S. COHEN, MD, MICHAEL J. BLOOM, MD, GARY J. KOLANCHIK, MD, JAMES B. TUCKER, MD, ROBERT L. SCHEER, MD

BOOK REVIEWS 118

OBITUARIES 122

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
APR 1 - 1985

Easy To Take



Keflex®
cephalexin

Additional information
available to the profession
on request.

DISTA
420113

Dista Products Company
Division of Eli Lilly and Company
Indianapolis, Indiana 46285
Mfd. by Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

BALANCED CALCIUM CHANNEL BLOCKADE!

CARDIZEM®
(diltiazem HCl)
balances potent coronary vasodilation with a low incidence of side effects

Low incidence of side effects
CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

Reduces angina attack frequency*
42% to 46% decrease reported in multicenter study.[1]

Increases exercise tolerance*
In Bruce exercise test,[2] control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes (*P*<.005).

CARDIZEM®
(diltiazem HCl)

THE BALANCED CALCIUM CHANNEL BLOCKER

Please see full prescribing information on following page. 2/84

---

*Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

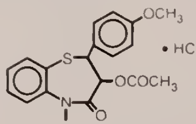
References:
1. Strauss WE, McIntyre KM, Parisi AF, et al: Safety and efficacy of diltiazem hydrochloride for the treatment of stable angina pectoris: Report of a cooperative clinical trial. Am J Cardiol 49:560-566, 1982.
2. Pool PE, Seagren SC, Bonanno JA, et al: The treatment of exercise-inducible chronic stable angina with diltiazem: Effect on treadmill exercise. Chest 78 (July suppl):234-238, 1980.

PROFESSIONAL USE INFORMATION

cardizem
(diltiazem HCl)
30 mg and 60 mg tablets

DESCRIPTION

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one,3-(acetyloxy) -5-[2-(dimethylamino)ethyl]-2,3-dihydro-2-(4-methoxyphenyl)-, monohydrochloride,(+)-cis-. The chemical structure is:



Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform. It has a molecular weight of 450.98. Each tablet of CARDIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration.

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth muscle.

Mechanisms of Action. Although precise mechanisms of its antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways:

1. Angina Due to Coronary Artery Spasm: CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial and subendocardial. Spontaneous and ergonovine-induced coronary artery spasm are inhibited by CARDIZEM.
2. Exertional Angina: CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal and maximal exercise work loads.

In animal models, diltiazem interferes with the slow inward (depolarizing) current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Diltiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance.

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricular conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load. Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect; cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of diltiazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by diltiazem.

Intravenous diltiazem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Diltiazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, diltiazem significantly prolongs sinus cycle length (up to 50% in some cases).

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Diltiazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40%. CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 60 mg are given; a 120-mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem.

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm.** CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina).** CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS).

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia.
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing.
Cardiac Failure	Administer inotropic agents (isoproterenol dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarteren bitartrate).

Actual treatment and dosage should depend on the severity of th clinical situation and the judgment and experience of the treati physician.

The oral/LD_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort ac anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy**—CARDIZEM may be safely coadministered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

Cardizem 30-mg tablets are supplied in bottles of 100 (NDC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1771-49). Each green tablet is engraved with MARION on one side and 1771 engraved on the other. CARDIZEM 60-mg scored tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1772-49). Each yellow tablet is engraved with MARION on one side and 1772 on the other.

Issued 4/1/84

Another patient benefit product from

PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MISSOURI 64137

Kodak presents...

New KODAK EKTACHEM DT60 Analyzer

A timely invest

The KODAK EKTACHEM DT60 Analyzer creates an extra service for your patients without extra investment in labor. And because it can pay for itself in three months, it's a timely investment in your future.

The chemistry tests you need

With the DT60 Analyzer you perform key chemistry tests in your own office instead of using an outside laboratory. Available tests include glucose, cholesterol, triglycerides, BUN, uric acid, sodium, and potassium, with total hemoglobin and bilirubin coming soon.

The time you need

Get test results in five minutes or less; perform up to 75 tests an hour. Save time waiting for results to assist in your diagnosis, and on follow-up phone calls.

The accuracy you need

The DT60 Analyzer uses proven technology and methodology from the KODAK EKTACHEM 400 and 700 Analyzers, which

© Eastman Kodak Company, 1984

ment for your office.

provide millions of accurate, precise results to clinical laboratories nationwide.

The simplicity you need

The DT60 Analyzer, compact as a personal computer, features dry slide technology to eliminate wet reagents. It is automated to free up your staff, and training takes only minutes. From the finger-stick sample to results printout, the DT60 Analyzer is simplicity itself.

To see what the DT60 Analyzer can do for you, write Eastman Kodak Company, Dept. 740-B, 343 State Street, Rochester, NY 14650, or call **1 800 44KODAK,** Ext 423 (1 800 445-6325, Ext 423) today.

Leading the way in healthcare technology for over 100 years.

KODAK EKTACHEM
Clinical Chemistry Products

MAY NOT BE AVAILABLE IN ALL AREAS

NEW YORK STATE JOURNAL OF MEDICINE

LIBRARY, ARCHIVES, AND PUBLICATIONS COMMITTEE

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
ELIZA H. CALDWELL, MD
URSULA MATULONIS (Medical Student)
GERALD P. MURPHY, MD
RICHARD B. NOLAN, MD
CARL E. POCHEDLY, MD
JOHN T. PRIOR, MD

Editor	ALAN BLUM, MD
Deputy Editor and Book Review Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Managing Editor	VICKI GLASER
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Administrative Assistant	IMELDA MORRISSEY
Editorial Assistant	JEANNE CONNERY
Librarian	ELLA ABNEY
Library Assistant	ELEANOR BURNS
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1985

RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MARK C. HOMONOFF, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. Synergistic Media Sales, 50 East 42nd St., Suite 504, New York, N.Y. 10017. Telephone: (212) 682-2200.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

ALLISON B. LANDOLT, MD, Westchester	*President*
BERNARD J. PISANI, MD, New York	*Past-President*
DANIEL F. O'KEEFFE, MD, Warren	*President-Elect*
JOHN A. FINKBEINER, MD, New York	*Vice-President*
JOHN H. CARTER, MD, Albany	*Secretary*
GEORGE LIM, MD, Oneida	*Assistant Secretary*
CHARLES D. SHERMAN, JR, MD, Monroe	*Treasurer*
MORTON KURTZ, MD, Queens	*Assistant Treasurer*
JOHN L. CLOWE, MD, Schenectady	*Speaker*
CHARLES N. ASWAD, MD, Broome	*Vice-Speaker*

Councilors

Term Expires 1985	JAMES H. COSGRIFF, JR, MD, Erie SAMUEL M. GELFAND, MD, Nassau RICHARD A. HUGHES, MD, Warren ANTONIO F. LASORTE, MD, Broome
Term Expires 1986	ROBERT A. MAYERS, MD, Westchester THOMAS D. PEMRICK, MD, Rensselaer RALPH E. SCHLOSSMAN, MD, Queens STANLEY A. STECKLER, MD, Suffolk
Term Expires 1987	DAVID M. BENFORD, MD, New York DUANE M. CADY, MD, Onondaga JAMES M. FLANAGAN, MD, Wayne M. THEODORE TANENHAUS, MD, Brooklyn

Trustees

CARL GOLDMARK, JR, MD, New York, *Chairman*
G. REHMI DENTON, MD, Albany
RALPH M. SCHWARTZ, MD, New York
EDGAR P. BERRY, MD, New York
RICHARD D. EBERLE, MD, Onondaga
JOSEPH F. SHANAPHY, MD, Richmond
VICTOR J. TOFANY, MD, Monroe

Executive Vice-President	MILTON ROSENBERG, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Associate	IRMA A. ERICKSON
Director, Division of Scientific Publications	ALAN BLUM, MD
Director, Division of Management Services	GEORGE FORREST, JR
Assistant Director, Division of Governmental Affairs	JERRY S. HOFFMAN, ESQ
Director, Division of Communications	EDWARD HYNES
Director, Division of Insurance and Membership Benefits	BERNARD M. JACKSON
Director, Division of Membership	EUNICE SKELLY
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Scientific Activities	GEORGE J. LAWRENCE, JR, MD
Director, Division of Advertising-Sales and Meeting Services	ROBERT MILLER
General Counsel	DONALD R. MOY, JD
Director, Division of Computer Services	IVAN H. NAJMAN
Director, Division of Medical Services	ROBERT J. O'CONNOR, MD
Director, Division of Long-Range Planning	DEBORAH R. SIEGEL
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. Editorial material published in the *Journal* is covered by copyright. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

Classified Advertising

PHYSICIANS WANTED

DIRECTOR OF HEALTH SERVICES: Ithaca College, a private, coeducational institution of 5,300 students in the Finger Lakes region of New York State, offers academic programs in the liberal arts as well as several professional areas. The College is largely residential with approximately 3,500 students living on campus in a variety of living arrangements. Position requires a full time physician to administer the College Health Service. The Health Center is a primary care facility with laboratory and inpatient beds. In addition to the Director there is a second physician, three PA's, and additional nursing and support staff. Formal duties are during the two major academic semesters. A college maintained house is provided as the expected residence. Liberal fringe benefits. Malpractice insurance. Salary competitive. Position begins August 15, 1985. Applications will be accepted until the position is filled. Applications received by April 15 will be given first consideration. Please submit applications with resumes to Richard J. Correnti, Vice President for Student affairs, Ithaca College, Ithaca, New York 14850.

PHYSICIAN FOR CHILDREN'S SUMMER CAMP. Located in lower NYS Catskills, CAMP ECHO seeks Physicians for July and August 1985. Physicains for two or four week periods, with or without camp age children. Excellent medical facility, 2 R.N.'s + aide, private rooms and facilities for M.D. Camp accommodates 350 boys and girls from 6-16 yrs. of age. Write CAMP ECHO, P.O. Box 255, Roslyn, NY 11576 or call (516) 742-2679.

GENERAL/FAMILY PRACTITIONERS, if you are looking for an opportunity to be in the forefront of medical care, practice preventive medicine, work with other innovative professionals, and earn a comfortable living in pleasant surroundings, send your curriculum vitae to Physician Placement Dept.—38. An equal opportunity employer. CIGNA Healthplans of California, 700 N. Brand Blvd., Suite 500, Glendale, CA 91203.

OBSTETRICIAN/GYNECOLOGIST: Second OB/GYN needed to join this rural group practice of 10 physicians. Primary care is emphasized. Formal tertiary linkages to Rochester General Hospital for high risk. Base salary, then income tied to productivity. Contact: Richard F. Endres, M.D., Sodus Health Center, P.O. Box A, Sodus, NY 14551. EOE.

FAMILY PRACTICE GROUP seeks BC/BE Family Practitioner. Early Partnership, five weeks vacation. Excellent hospitals. Beautiful college town in the Hudson Valley vacation area 90 miles north of N.Y.C. Address CV to Administrator, Medical Associates of New Paltz, PC, 40 Sunset Ridge, New Paltz, NY 12561.

PHYSICIANS WANTED—CONT'D

PHYSICIANS needed for 75 residential children's camps located in Northeast. July and/or August. Contact: Association of Independent Camps (M), 60 Madison Ave., New York, NY 10010. (212) 679-3230.

BOARD CERTIFIED INTERNIST: Preventive & Occupational Health—a leader in field offers varied opportunities (full-time, half-time or per diem) at our clinics and corporate medical units. Send C.V. to Executive Health Examiner, 777 Third Ave., N.Y., N.Y. 10017, Attn: Personnel Director N. [EOE M/F].

WOODSTOCK, NEW YORK: BC OB/GYN seeks BC/BE female or male for rapidly expanding progressive general OB/GYN practice. Curently rich artists community 20 minutes from all mountain recreation. Rural area with diverse patient population. Practice emphasises patient education, responsibility, and choice and offers services at Community Hospital and Free-Standing (in office) Birthing Room with Midwife. Flexible creative call schedule with possibility of 3-4 monthes off per year to pursue other interests or participate in medical work exchange on Carribean Island. Our office hours, patient load and philosophy reflect the value we place on our own health and family life. Type of association is negotiable with potential for early partnership. Please send replys to Lawrence M. Perl, M.D., RD#1 Box 57, Athens, NY 12015.

ACTIVE GENERAL INTERNIST seeks board cert. or eligible cardiologist also willing to do general internal medicine. No invasive cardiology. Modern, luxurious office equipped with Holter Monitor, automative lab partially computerized. Very desirable area Eastern L.I. 1½ hours from N.Y.C. Excellent boating, sailing, fishing, serenity. Salary, Malpractice, early partnership. Send CV to Dept. 379 c/o NYSJM.

PHYSICIAN FOR CHILDREN'S SUMMER CAMP JULY AND/OR AUGUST 1985. Camp located at Beach Lake, Pennsylvania, accommodates 350 campers, ages 6–16. Complete modern health center; 2 R.N.'s in attendance. Will accept one M.D. for each month, no children accepted who are of camp age. Camp opens June 29th and closes August 23rd. Private room and facilities. Write to: Trail's End Camp, c/o Beach Lake, Inc., 215 Adams Street, Brooklyn, NY 11201. Please include your phone number.

INTERNAL MEDICINE: Full-time opportunities available for board eligible or board certified physicians for busy inpatient internal medicine department of south Florida hospital. Clinic positions also available in Tampa, Palm Beach and Miami. Send C.V. to Fern Blum, EMSA, 8200 W. Sunrise Blvd., Building C, Plantation, Florida 33322 or call (305) 472-6922.

FREESTANDING EMERGENCY CENTERS seeks high quality physicians with emergency or family practice experience to work full-time and part-time in progressive, people oriented FEC's located in Western and Northern New York communities. Medical Directorship opportunities also available. Reply with C.V. and cover letter stating availability and salary requirements to: Medical Center Consultants, 2279 Grand Island Blvd., Grand Island, New York 14072.

PHYSICIANS WANTED—CONT'D

MANHATTAN EYE, EAR & THROAT HOSPITAL Fellowship in Ophthalmic Plastic Reconstructive and Orbital Surgery. Preceptors: Byron Smith, M.D., Richard D. Lisman, M.D., Murray Meltzer, M.D. Available: July 1985–June 1986. An extensive one year fellowship with full salary, malpractice insurance, heavy surgical load, general plastic surgery and head and neck exposure, direct patient responsibility, including primary surgical cases. Inquires and C.V. to: Administrator, Department of Ophthalmology, Manhattan Eye, Ear & Throat Hospital, 210 East 64th Street, New York, NY 10021.

MEDICAL CARE CLINIC in Sun City, Arizona, multi-speciality clinic seeks Neurologist, Gynecologist, Ophthalmologist, Internal Medicine, Gastroenterologist and Dermatologist. Area served has approximately 100,000 population, and three full-service hospitals. Retirement community surrounded by rapidly growing communities. Terms negotiable. Contact Barry Horst, 10448 W. Coggins Drive, Sun City, Arizona 85351, (602) 933-5457.

PRIMARY CARE PHYSICIAN: Physician needed for Skilled Nursing Facility located in Queens. Should have some experience with acute and long term care. Residents of the nursing home are particularly interested in a female practitioner. Contact: Arthur T. Risbrook, M.D., Medical Director at (718) 739-2523 ext. 15.

PRACTICE OPPORTUNITY: Family Practitioner, well established, multi-specialty group, with a developing Family Practice Department, is looking for a Family Practitioner for its satellite offices. Competitive salary. Liberal fringe benefits. Please send inquiry and Curriculum Vitae to Executive Director, Slocum-Dickson Medical Group P.C., 430 Court Street, Utica, NY 13502. Telephone area code 315-797-0500.

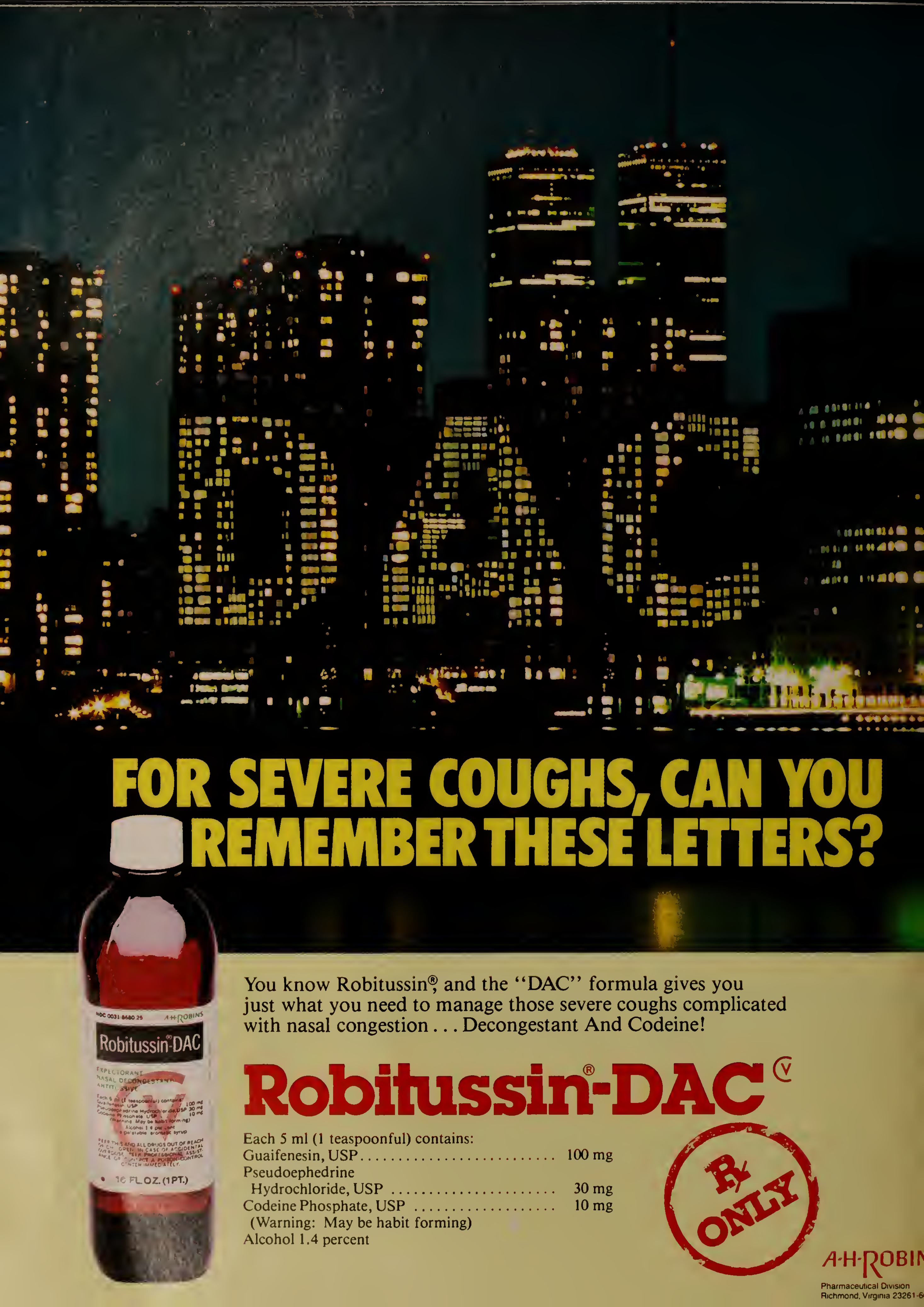
PHYSICIAN WANTED to share office space with an older general practitioner with intention of sale on retirement. Pleasant, rural community in Upstate New York. Office next to hospital. Terms negotiable. Call (315) 536-4456 or nights (315) 536-8084 or write H.A. Mikk, M.D., Med. Arts Bldg., Penn Yan, New York 14527.

POSITIONS WANTED

GENERAL SURGEON, Board certified, aggressive young and recently graduated from an excellent New York residency program is seeking a position in a group practice in New York State. CV on request. J. Hyacinthe, M.D., 930 East 101 Street, Brooklyn, NY 11236.

PRACTICES WANTED

YOUNG INTERNIST, wishes to purchase Nassau County office based or nursing home based private practice. Available immediately. Strictly confidential. Reply Dept. 365, c/o NYSJM.



FOR SEVERE COUGHS, CAN YOU REMEMBER THESE LETTERS?

You know Robitussin®, and the "DAC" formula gives you just what you need to manage those severe coughs complicated with nasal congestion . . . Decongestant And Codeine!

Robitussin®-DAC Ⓥ

Each 5 ml (1 teaspoonful) contains:

Guaifenesin, USP	100 mg
Pseudoephedrine Hydrochloride, USP	30 mg
Codeine Phosphate, USP	10 mg

(Warning: May be habit forming)
Alcohol 1.4 percent

℞ ONLY

A·H·ROBI
Pharmaceutical Division
Richmond, Virginia 23261-

DON'T COUNT ON THIS ALONE TO SAVE YOUR LIFE.

First-aid kits are fine for some emergencies. But what if you were choking? Having a heart attack? Or a stroke? This kit would be useless.
Call the Red Cross. We'll teach you and your employees how to save a life.
From work-safety to CPR, you can count on the Red Cross.
We'll help. Will you?

A Public Service of This Publication Ad Council

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meetings and lectures unless the information is submitted at least two months prior to the event.*

APRIL 1985

AROUND THE STATE

MANHATTAN

Apr 2. **The Hearing Impaired Child. Diagnosis and Management.** *Apr 3.* **How to Develop a Research Project.** *Apr 20.* **Psychodynamic Basis of Supportive Psychotherapy.** *Apr 26.* **Common Problems in Pediatric Surgery Update 1985.** Info: Post-Graduate Medical Education, Beth Israel Medical Center, 10 Nathan D. Perlman Pl, New York, NY 10029. Tel: (212) 420-2849.

Apr 10–13. **Alcoholism & Drug Abuse: Problems in Clinical Decision-Making.** Smithers Alcoholism Treatment & Training Center of the St. Luke's-Roosevelt Hospital Center. 25 Cat 1 credits. *Apr 26.* **Contraception: Issues & Controversies.** 7 Cat 1 credit. Info: Dr Elizabeth C. Gerst, Director, CME, College of Physicians & Surgeons, Columbia University, 630 W. 168th St, New York, NY 10032. Tel: (212) 694-3682.

Apr 11–13. **Lasers in Ophthalmology.** 18 Cat 1 credits. Info: Janet M. Moskow, Manhattan Eye, Ear & Throat Hospital, 210 East 64th St, New York, NY 10021. Tel: (212) 838-9200.

Apr 12–13. **Ureteropyeloscopy Seminar.** Memorial Sloan-Kettering Cancer Center. 12 Cat 1 credits. Info: CME Conference Planning Office, C-180, MSKCC, 1275 York Ave, New York, NY 10021. Tel: (212) 794-6754.

Apr 15–19. **Intensive Care Medicine.** Info: American College of Physicians, 4200 Pine St, Philadelphia, PA 19104. Tel: (800) 523-1546.

Apr 15–19. **Comprehensive Review of Physical Medicine and Rehabilitation.** 42 Cat 1 credits. *Apr 25–27.* **Advances in Dermatology.** 17 Cat 1 credits. *Apr 27 & 28.* **Review of Basic Sciences in Urology.** 14 Cat 1 credits. *Apr 29–May 2.* **Interventional Neuroradiology.** 29 Cat 1 credits. Info: NYU Medical Center Post-Graduate Medical School, 550 First Avenue, New York, NY 10016. Tel: (212) 340-5295.

Apr 16. **Family Therapy with Chronic Physical Illness.** Lenox Hill Hospital. Info: Mrs V. Matthes, 542 Baldwin Ave, Baldwin, NY 11510. Tel: (516) 379-1787.

Apr 17. **Monthly Slide Seminar—Diseases of the Breast.** 2 Cat 1 credits. Info: Victor Tchertkoff, MD, Moderator, Metropolitan Hospital Center, 1901 First Ave, New York, NY 10029. Tel: (212) 360-6253.

Apr 17–19. **Neurochemical Analysis of the Conscious Brain: Voltammetry & Push-Pull Perfusion.** Info: New York Academy of Sciences, 2 E 63rd St, New York, NY 10021.

Apr 19. **Perspectives on Changing Reimbursements—The New Economics of Medical Care.** Warwick Hotel. Info: Ann J. Boehme, CME, Long Island Jewish-Hillside Medical Center, New Hyde Park, NY 11042. Tel: (212) 470-2114.

Apr 20. **Recent Advances in Anorectal Surgery.** *Apr 26.* **Common Problems in Pediatric Surgery—Update 1985.** *Apr 27.* **Biofeedback Medicine.** Info: Page & William Black Post-Graduate School of Medicine, Mount Sinai School of Medicine, One Gustave L. Levy Pl, New York, NY 10029. Tel: (212) 650-6737.

Apr 24–27. **The Interspeciality Facial Surgery Congress for Aesthetic, Cancer & Reconstructive Surgery.** Waldorf-Astroia. Info: James F. Pawlak, c/o Pierre Guibor, MD, 630 Park Ave, New York, NY 10021. Tel: (212) 734-1010 or (800) 223-4500.

BROOKLYN

Apr 9. **Update on Obstetric Anesthesia.** *Apr 10.* **Resuscitation of the Newborn.** Info: Abraham I. Raskob, MD, CME, SUNY-Downstate Medical Center, Department of Anesthesiology, Box #6, 450 Clarkson Ave, Brooklyn, NY 11203, Tel: (212) 270-1934.

Apr 3. **Esophageal Carcinoma.** *Apr 17.* **Childhood Thrombocytopenic Purpura**

Motrin®
ibuprofen, Upjohn
600 mg Tablets

More convenient for your patients.

Upjohn

©1984 The Upjohn Company

The Upjohn Company • Kalamazoo, Michigan 49001 USA

J-4044 January 1984

New studies uncover the potassium effects of beta-2 blockade

Clinical pharmacology data from *The New England Journal of Medicine:*

"...when normal young men are given infusions of epinephrine at levels such as those that circulate in patients with myocardial infarction, their serum potassium concentrations fall by about 0.8 [mmol] per liter. Hypokalemia is prevented by selective beta-2 blockade."[1]

Evidence that all beta blockers are not created equal.

Right from the start in hypertension...

Once-daily INDERAL LA (propranolol HCl) for smooth blood pressure control without the potassium problems of diuretics

Once-daily INDERAL LA (propranolol HCl) avoids the risk of diuretic-induced ECG abnormalities due to hypokalemia.[2,3] In addition, INDERAL LA preserves potassium balance without additive agents or supplements while providing simple, well-tolerated therapy with broad cardiovascular benefits.

Once-daily INDERAL LA for the cardiovascular benefits of the world's leading beta blocker

Simply start with 80 mg once daily. Dosage may be increased to 120 mg to 160 mg once daily as needed to achieve additional control.

Like conventional INDERAL tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.

For beta-1/beta-2 blockade

Once-daily

INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES

The appearance of these capsules is a registered trademark of Ayerst Laboratories.

80 mg 120 mg 160 mg

Please see brief summary of prescribing information on the next page for further details.

Once-daily
For beta-1/beta-2 blockade INDERAL® LA
(PROPRANOLOL HCl) LONG ACTING CAPSULES

The appearance of these capsules is a registered trademark of Ayerst Laboratories

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION SEE PACKAGE CIRCULAR.)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use Effects on plasma volume appear to be minor and somewhat variable INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients.

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential The significance of the membrane action in the treatment of arrhythmias is uncertain

The mechanism of the antimigraine effect of propranolol has not been established Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm

Propranolol is not significantly dialyzable.

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e.g., dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes In these patients, it may be more difficult to adjust the dosage of insulin

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General* Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure.

Clinical Laboratory Tests Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy Pregnancy Category C INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman

Pediatric Use Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular bradycardia, congestive heart failure, intensification of AV block; hypotension; paresthesia of hands; thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type

Central Nervous System lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances, hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics

Gastrointestinal nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: bronchospasm.

Hematologic agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily. If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval.

HYPERTENSION—*Dosage must be individualized* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood pressure control is achieved. The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE—*Dosage must be individualized.* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis. If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

REFERENCES

1. Epstein FH, Rosa RM Adrenergic control of serum potassium *N Engl J Med* 1983, 309 1450-1451 **2.** Holland OB, Nixon JV, Kuhnert L. Diuretic-induced ventricular ectopic activity *Am J Med* 1981;70 762-768 **3.** Holme I, Helgeland A, Hjermann I, et al Treatment of mild hypertension with diuretics The importance of ECG abnormalities in the Oslo study and in MRFIT. *JAMA* 1984,251 1298-1299.

9411/1184

Ayerst® AYERST LABORATORIES
New York, N Y 10017

Copyright © 1984 AYERST LABORATORIES
Division of AMERICAN HOME PRODUCTS CORPORATION

Angina comes in many forms...

So does SORBITRATE®

(ISOSORBIDE DINITRATE)

Unsurpassed flexibility in nitrate therapy.

2.5 mg 5 mg 10 mg
Sublingual Tablets

5 mg 10 mg
Chewable Tablets

5 mg 10 mg 20 mg 30 mg 40 mg
Oral "Swallow" Tablets

40 mg
Sustained Action "Swallow" Tablets

© 1985 ICI AMERICAS INC.

See following page for brief summary of prescribing information.

SORBITRATE®
(ISOSORBIDE DINITRATE)

Please consult full prescribing information before use. A summary follows:

INDICATIONS AND USAGE: SORBITRATE (isosorbide dinitrate) is indicated for the treatment and prevention of angina pectoris All dosage forms of isosorbide dinitrate may be used prophylactically to decrease frequency and severity of anginal attacks and can be expected to decrease the need for sublingual nitroglycerin

The sublingual and chewable forms of the drug are indicated for acute prophylaxis of angina pectoris when taken a few minutes before situations likely to provoke anginal attacks Because of a slower onset of effect, the oral forms of isosorbide dinitrate are not indicated for acute prophylaxis

CONTRAINDICATIONS: SORBITRATE is contraindicated in patients who have shown purported hypersensitivity or idiosyncrasy to it or other nitrates or nitrites Epinephrine and related compounds are ineffective in reversing the severe hypotensive events associated with overdose and are contraindicated in this situation.

WARNINGS: The benefits of SORBITRATE during the early days of an acute myocardial infarction have not been established If one elects to use organic nitrates in early infarction, hemodynamic monitoring and frequent clinical assessment should be used because of the potential deleterious effects of hypotension

PRECAUTIONS: General: Severe hypotensive response, particularly with upright posture, may occur with even small doses of SORBITRATE The drug should therefore be used with caution in subjects who may have blood volume depletion from diuretic therapy or in subjects who have low systolic blood pressure (eg, below 90 mmHg) Paradoxical bradycardia and increased angina pectoris may accompany nitrate-induced hypotension. Nitrate therapy may aggravate the angina caused by hypertrophic cardiomyopathy

Marked symptomatic, orthostatic hypotension has been reported when calcium channel blockers and organic nitrates were used in combination Dose adjustment of either class of agents may be necessary

Tolerance to this drug and cross-tolerance to other nitrates and nitrites may occur Tolerance to the vascular and antianginal effects of isosorbide dinitrate or nitroglycerin has been demonstrated in clinical trials, experience through occupational exposure, and in isolated tissue experiments in the laboratory. The importance of tolerance to the appropriate use of isosorbide dinitrate in the management of patients with angina pectoris has not been determined However, one clinical trial using treadmill exercise tolerance (as an end point) found an 8-hour duration of action of oral isosorbide dinitrate following the first dose (after a 2-week placebo washout) and only a 2-hour duration of effect of the same dose after 1 week of repetitive dosing at conventional dosing intervals On the other hand, several trials have been able to differentiate isosorbide dinitrate from placebo after 4 weeks of therapy and, in open trials, an effect seems detectable for as long as several months

Tolerance clearly occurs in industrial workers continuously exposed to nitroglycerin. Moreover, physical dependence also occurs since chest pain, acute myocardial infarction, and even sudden death have occurred during temporary withdrawal of nitroglycerin from the workers In clinical trials in angina patients, there are reports of anginal attacks being more easily provoked and of rebound in the hemodynamic effects soon after nitrate withdrawal The relative importance of these observations to the routine, clinical use of isosorbide dinitrate is not known However, it seems prudent to gradually withdraw patients from isosorbide dinitrate when the therapy is being terminated, rather than stopping the drug abruptly

Information for Patients: Headache may occur during initial therapy with SORBITRATE Headache is usually relieved by the use of standard headache remedies or by lowering the dose and tends to disappear after the first week or two of use

Drug Interactions: Alcohol may enhance any marked sensitivity to the hypotensive effect of nitrates

Isosorbide dinitrate acts directly on vascular smooth muscle, therefore, any other agent that depends on vascular smooth muscle as the final common path can be expected to have decreased or increased effect depending on the agent

Carcinogenesis, Mutagenesis, Impairment of Fertility: No long-term studies in animals have been performed to evaluate the carcinogenic potential of this drug A modified two-litter reproduction study in rats fed isosorbide dinitrate at 25 or 100 mg/kg/day did not reveal any effects on fertility or gestation or any remarkable gross pathology in any parent or offspring fed isosorbide dinitrate as compared with rats fed a basal-controlled diet

Pregnancy Category C: Isosorbide dinitrate has been shown to cause a dose-related increase in embryotoxicity (increase in mummified pups) in rabbits at oral doses 35 and 150 times the maximum recommended human daily dose There are no adequate and well-controlled studies in pregnant women SORBITRATE should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: It is not known whether this drug is excreted in human milk Because many drugs are excreted in human milk, caution should be exercised when SORBITRATE is administered to a nursing woman

Pediatric Use: The safety and effectiveness of SORBITRATE in children has not been established

ADVERSE REACTIONS: Adverse reactions, particularly headache and hypotension, are dose-related In clinical trials at various doses, the following have been observed

Headache is the most common (reported incidence varies widely, apparently being dose-related, with an average occurrence of about 25%) adverse reaction and may be severe and persistent Cutaneous vasodilation with flushing may occur Transient episodes of dizziness and weakness, as well as other signs of cerebral ischemia associated with postural hypotension, may occasionally develop (the incidence of reported symptomatic hypotension ranges from 2% to 36%). An occasional individual will exhibit marked sensitivity to the hypotensive effects of nitrates and severe responses (nausea, vomiting, weakness, restlessness, pallor, perspiration, and collapse) may occur even with the usual therapeutic dose Drug rash and/or exfoliative dermatitis may occasionally occur Nausea and vomiting appear to be uncommon Case reports of clinically significant methemoglobinemia are rare at conventional doses of organic nitrates The formation of methemoglobin is dose-related and, in the case of genetic abnormalities of hemoglobin that favor methemoglobin formation, even conventional doses of organic nitrate could produce harmful concentrations of methemoglobin.

DOSAGE AND ADMINISTRATION: For the treatment of angina pectoris, the usual starting dose for sublingual SORBITRATE is 2 5 to 5 mg; for chewable tablets, 5 mg, for oral (swallowed) tablets, 5 to 20 mg, and for controlled-release forms, 40 mg

SORBITRATE should be titrated upward until angina is relieved or side effects limit the dose In ambulatory patients, the magnitude of the incremental dose increase should be guided by measurements of standing blood pressure

The initial dosage of sublingual or chewable SORBITRATE for prophylactic therapy in angina pectoris patients is generally 5 or 10 mg every 2 to 3 hours Adequate controlled clinical studies demonstrating the effectiveness of chronic maintenance therapy with these dosage forms have not been reported

SORBITRATE in oral doses of 10 to 40 mg given every 6 hours or in oral controlled-release doses of 40 to 80 mg given every 8 to 12 hours is generally recommended The extent to which development of tolerance should modify the dosage program has not been defined *The oral controlled-release forms of isosorbide dinitrate should not be chewed*

DOSAGE FORMS AVAILABLE: Sublingual Tablets (2 5, 5, 10 mg), Chewable Tablets (5, 10 mg), Oral Tablets (5, 10, 20, 30, 40 mg), Sustained Action Tablets (40 mg)

STUART PHARMACEUTICALS
Division of ICI Americas Inc.
Wilmington DE 19897

STR-2282

(ITP)—Controversies in Management. Info: Harvey Dosik, MS, Interfaith Medical Center, 555 Prospect Pl, Brooklyn, NY 11238. Tel: (718) 240-1000.

NASSAU

Apr 19–21. **Thermography in Neuromusculoskeletal Disorders.** Glen Cove. 10 Cat 1 credits. Info: Page & William Black Postgraduate School of Medicine, Mount Sinai School of Medicine, One Gustave L. Levy Pl, New York, NY 10029. Tel: (212) 650-6737.

Apr 24–26. **Low Back Pain & Sciatica Syndrome Revisited.** Nassau Country Medical Center. 18 Cat 1 credits. Info: Arminius Cassvan, MD, Department of Physical Medicine & Rehabilitation, Nassau County Medical Center, East Meadow, NY 11554. Tel: (516) 542-2525.

SUFFOLK

Apr 2. **Re-examining "Normal Routine" care of the newborn.** *Apr 9.* **Monitoring Blood Gases in the Sick Neonate.** *Apr 16.* **Neonatal Medicine.** Info: Arthur Schwager, MD, CME, Huntington Hospital, 270 Park Ave, Huntington, NY 11743. Tel: (516) 351-2000.

Apr 8. **Clinical Applications of Micro-Surgical Reconstruction and Replantation.** Info: Donna C. Joslin, CME, Southampton Hospital, 240 Meeting House Lane, Southampton, NY 11968. Tel: (516) 283-2600, ext 505.

Apr 19–20. **Current Concepts in the Design of Hip Implants.** The Garden City Hotel. Info: Tamarath K. Yolles, MD, CME, School of Medicine (HSC), 4L, Rm. 177 SUNY at Stony Brook, NY 11794-8437. Tel: (516) 444-2094.

BROOME

Apr 27. **Organizing a Multidisciplinary Team Approach to Trauma.** Binghamton. 6 Cat 1 credit. Info: Sandy Cook, CME, Binghamton General Hospital, Mitchell Ave, Binghamton, NY 13903. Tel: (607) 771-2549.

MONROE

Apr 16–19. **Changing Scenes in the Laboratory.** Rochester. Info: Empire State Association for Medical Technology, 133 Somershire Drive, Rochester, NY 14617. Tel: (716) 338-4425.

Only Ativan® (lorazepam) Ⓒ IV

offers all these benefits in addition to rapid relief of anxiety:

- relief of anxiety associated with depressive symptoms
- clearance not significantly delayed by age, liver or kidney dysfunction
- cumulative sedative effects seldom a problem
- little likelihood of drug interaction (all benzodiazepines produce additive effects when taken with alcohol or other CNS depressants)
- no significant changes in vital signs in cardiovascular patients*
- short duration of action, simple metabolism

*Benzodiazepines have not been shown to be of benefit in treating the cardiovascular component.

See important information on following page.

© 1985, Wyeth Laboratories

Wyeth Laboratories
Philadelphia, PA 19101

The appearance of these tablets is a registered trademark of Wyeth Laboratories

DISTINCTIVE DESIGN® 0.5, 1.0 and 2.0 mg

Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Transient amnesia or memory impairment has been reported in association with the use of benzodiazepines.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® Ⓒ
for (lorazepam)
Anxiety

DOSAGE: Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.

HOW SUPPLIED: 0.5, 1.0 and 2.0mg tablets.

Wyeth Laboratories
Philadelphia, PA 19101

NEW LAW CUTS TAX BREAKS FOR LUXURY CARS

If the car you want costs more than $16,667.00 don't spend your money, use ours—we still pass the investment tax credit to you.

If you're in the 50% tax bracket no longer can you save half the depreciation on anything over $16,677.00 for the first 3 years:

HOWEVER,

When you lease you can continue to deduct your monthly rental payments in excess of $16,000.00 *provided* you conform to the limits and rules set by the I.R.S. regarding usage. The leasee must use the vehicle more than 50% of the time for his business or profession.

SO DOESN'T IT MAKE SENSE TO LEASE NOW!

ALL MAKES & MODELS AVAILABLE.

CALL CAROL LEASING INC.

(516) 222-9041

ROOSEVELT RACEWAY, WESTBURY, NY 11590

Need A Temporary Physician?
During: vacations, CME's, illness, recruiting.
Want Free Time While You Practice Medicine?
Join CompHealth's Locum Tenens Physician Group.
For further information, call 412-741-3310.

CompHealth A Physician Group
WILSON ROSS, Regional Administrator
114 Centennial Ave., Sewickley, PA 15143

NEEMA EMERGENCY MEDICAL
—a professional association—

Emergency Medicine Positions—available with emergency physician group in PA, NY, NJ, VA, WVA, MD, KY and throughout New England, the Southeast, the Midwest, including all suburban, rural and metropolitan areas. Fee-for-service with minimum guarantee provided. Malpractice paid. Practice credits toward board certification. Physician department directors also desired. Please send resume to: NEEMA Emergency Medical, Suite 400, 399 Market Street, Phila., PA 19106 or phone 215-925-3511 in PA, or 800-523-0776 outside PA.

NEW YORK STATE
JOURNAL OF MEDICINE

March 1985 • Volume 85, Number 3

COMMENTARIES

Hodgkin's disease in siblings

One of the unique features about Hodgkin's disease is that at least three different age incidence patterns exist for this disorder. In industrialized countries the disease appears to occur in a bimodal pattern with peaks in young adulthood and older ages.[1,2] In poor countries in Africa[3] and in Latin America[4] this disease is most common in children and the elderly. Developing areas, such as Puerto Rico, appear to have an intermediate pattern, with a high proportion of childhood cases and an increased frequency among young adults.[5] Different epidemiologic patterns are also observed within countries, such as in the United States. For example, the age incidence curves for the black population in New York State (excluding New York City) are most similar to those observed in developing countries.[5]

The changes in the incidence patterns of Hodgkin's disease noted at various levels of socioeconomic development strongly suggest that environmental factors are important in the etiology of the disorder.[1,6] Familial studies of this lymphoma would also appear to support such a hypothesis. In one of the first extensive studies of familial Hodgkin's disease, Razis et al[7] found a threefold excess among families with a member who had this disorder. The investigators interpreted this finding as being consistent with an infectious cause. Other investigators[7-11] have suggested that the most common familial pattern of this lymphoma is in siblings. In this issue of the *Journal*, Hirschman and Nobler[12] report on nodular sclerosing Hodgkin's disease that occurred in a half-brother and half-sister. They had the same mother and the condition was diagnosed nine years apart. The authors raise the possibility that an infectious agent requiring intense contact at an early age might be involved. These suggestions are consistent with much of the current knowledge of Hodgkin's disease.

There does appear to be a significant excess of the lymphoma among the siblings of affected individuals, particularly if they are of the same sex[10] and close in age. In addition, residence in the same household prior to and at the time of diagnosis of the first sibling appears to be an important factor.[11] Both observations suggest that prolonged close contact might be a key element.

In contrast to these findings, Hodgkin's disease in spouses is rare.[10,11] If either prolonged or close interpersonal contact, or both, is important, additional factors must be involved. Genetic mechanisms might influence the predisposition to developing this malignancy, as might also be the case with Burkitt's tumor[13] and the X-linked syndrome described by Purtilo.[14] Indeed, HLA genotypes might play an important role in the occurrence of Hodgkin's disease.[15]

Even if this is true, the excessive frequency in siblings must be considered in light of the compelling evidence to suggest that age-related environmental factors are important in the etiology of this lymphoma in the family setting. Large sibships (≥ 5) appear to be at increased risk for this disorder.[16] Furthermore, when one examines the birth order distribution of childhood patients from these large sibships, the excess occurs in birth orders one and two; a significant decrease in risk is evident for birth order five and greater.[16] These observations suggest that familial factors might influence the age of initial exposure and the host response to an environmental agent. The very low risk of Hodgkin's disease among siblings of birth order five and greater might be due to exposure at a very early age which results in a high naturally acquired immunity to disease ratio. In contrast, children of birth order one have a higher risk, possibly because exposure is delayed until the beginning of their school years. The same phenomenon appears to be true for certain viral disorders.[17]

Clearly there is a need for additional studies on the influence of sibship size, birth order, sexual concordance, and types of contact (intensity, duration) on observed incidence patterns of Hodgkin's disease. This is true in both the family setting and for student aggregates, where close repetitive contact also appears to be important.[18] Family factors may also influence the different age-incidence patterns of this lymphoma. Could we possibly be describing the epidemiologic activity of a viral agent in Hodgkin's disease?

NICHOLAS J. VIANNA, MD
Director
Division of Health Risk Control
New York State Department of Health
Corning Tower
Empire State Plaza
Albany, NY 12237

1. O'Conor GT, Correa P, Christine B, Axtell L, Myers M: Hodgkin's disease in Connecticut: histology and age distribution, in *International Symposium on Hodgkin's Disease*, National Cancer Institute Monograph, No. 36. US Department of Health, Education and Welfare, 1973, pp 3-8.
2. Stalsberg LT: Hodgkin's disease in Western Europe: a review, in *International Symposium on Hodgkin's Disease*, National Cancer Institute Monograph, No. 36. US Department of Health, Education and Welfare, 1973, pp 31-36.
3. Davies JNP: Hodgkin's disease in childhood, in *International Symposium on Hodgkin's Disease*, National Cancer Institute Monograph, No. 36. US Department of Health, Education and Welfare, 1973, pp 45-47.
4. Correa P: Hodgkin's disease in Latin America, in *International Symposium on Hodgkin's Disease*, National Cancer Institute Monograph, No. 36, US Department of Health, Education and Welfare, 1973, pp 9-14.
5. Correa P, O'Conor GT: Epidemiologic patterns of Hodgkin's disease. *Int J Cancer* 1971; 8:192-201.
6. Vianna NJ, Thind IS, Louria DB, Polan A, Kirmss V, Davies JNP: Epidemiologic and histologic patterns of Hodgkin's disease in blacks. *Cancer* 1977; 40: 3133-3139.
7. Razis DV, Diamond HD, Carver LF: Familial Hodgkin's disease: its significance and implications. *Ann Intern Med* 1959; 51:933.
8. DeVore JW, Doan CA: A study of the Hodgkin's syndrome XII. Hereditary and epidemiological aspects. *Ann Intern Med* 1957; 47:300.
9. MacMahon B: Epidemiology of Hodgkin's disease. *Cancer Res* 1966; 26: 1189-1201.
10. Gufferman S, Cole P, Smith PG, Lukes RJ: Hodgkin's disease in siblings. *N Engl J Med* 1977; 296:248-250.
11. Vianna NJ, Davies JNP, Polan AK, Wolfgang P: Familial Hodgkin's disease: an environmental and genetic disorder. *Lancet* 1974; 2:854-857.
12. Hirschman RJ, Nobler MP: Hodgkin's disease in half-siblings. *NY State J Med* 1985;85:109.
13. Rowley JD: Do human tumors show a chromosome pattern specific for each etiologic agent? *J Natl Cancer Inst* 1974; 52:315-320.
14. Purtilo DT, Yang JPS, Cassel CK, Harper R: X-linked recessive progressive combined variable immunodeficiency (Duncan's disease). *Lancet* 1975; 1:935-940.
15. Hors J, Steinberg G, Andrieu JM, et al: HLA genotypes in familial Hodgkin's disease. Excess of HLA identical affected sibs. *Eur J Cancer* 1980; 16:809-815.
16. Vianna NJ, Polan AK: Immunity in Hodgkin's disease: Importance of age at exposure. *Ann Intern Med* 1978; 89:550-556.
17. Fox JP, Hall CE, Elveback LR: *Epidemiology: Man and Disease.* London, The MacMillan Co, 1970, p 200.
18. Vianna NJ, Greenwald P, Brady J, et al: Hodgkin's disease: cases with features of a community outbreak. *Ann Intern Med* 1972; 77:169-180.

Adult-onset Still's disease

Adult onset Still's disease is an uncommon disease with an almost mystical quality. In adults it is seen as a severe febrile illness and is most often mistaken for an infectious disease. Because of its systemic nature, this disease presents clinicians with a difficult challenge.

Classic signs of adult-onset Still's disease include a quotidian fever pattern associated with an evanescent, pruritic rash usually found in areas of skin contact with clothing or sheets. There is severe malaise associated with the fever, and arthralgias are prominent. Arthritis is a part of the disease but may not be present early in its course, and occasionally is absent altogether.[1] Sore throat is a consistent symptom in recent series[2] but does not have a recognizable basis on examination. Other symptoms, including abdominal pain, neurologic involvement, and polyserositis have been reported.[1,3,4] The report by Cush, Leibowitz and Friedman[5] in this issue of the *Journal* of ocular involvement with an inflammatory orbital pseudotumor emphasizes the ubiquitous inflammatory nature of the disease. The consistent lack of serious renal involvement is of interest, although some investigators have reported inflammatory lesions on renal biopsy.[1]

In the acute situation, laboratory studies are useful for eliminating other possible ailments. There are, however, several consistent and helpful findings: peripheral blood leukocyte counts are almost always elevated and frequently are in the 30,000 to 50,000 range. This one laboratory finding in the absence of bacterial infection should signal the possibility of adult-onset Still's disease, since systemic lupus erythematosus classically presents with leukopenia. The erythrocyte sedimentation rate is also almost always elevated but is a much less helpful finding in general. Abnormal liver enzyme levels are common and should not dissuade a physician from this diagnosis.

The differential diagnosis guides the clinician to the use of the appropriate laboratory tests. Diseases that should be considered include systemic lupus erythematosus (with tests for antinuclear antibodies and anti-DNA antibodies), acute viral illnesses (viral titers), subacute bacterial endocarditis (blood cultures) or other acute infections including tuberculosis. Rheumatoid arthritis, rheumatic fever, and acute leukemia or lymphoma must also be considered. Extensive studies are frequently necessary to rule out these possibilities and to detect the extent of organ involvement. Unfortunately, pathologic specimens obtained from skin, muscle, kidney, lymph node, liver, synovium, and bone marrow are also nonspecific.[1] Response to aspirin therapy or steroids (or both) together with follow-up are additional keys to diagnosis. This is especially true in light of the classic fusion of the carpal metacarpal joint described by Medsgar and Christy.[6]

Clinicians who care for patients with early stage adult-onset Still's disease are faced with a myriad of symptoms and a paucity of helpful laboratory tests. The seriousness of the clinical picture most often leads to a trial of antibiotic therapy while the laboratory studies proceed. It is hard to argue against such therapy, but when cultures for bacteria are negative the drugs should be stopped. Treatment with high doses of aspirin has been proposed to avoid the expense of extensive laboratory testing. Such a trial should not be attempted until acute infection, systemic lupus erythematosus, leukemia, and tuberculosis have been ruled out with reasonable certainty, and special care should be taken if hepatic enzyme levels are elevated.[2,7].

Although adult-onset Still's disease should not be at the top of the list of causes of an acute febrile illness, it should be considered when other more likely possibilities are eliminated. Its multisystemic nature and cyclic character, together with a void of information as to possible pathologic mechanisms, make it a fascinating illness.

DANIEL G. BAKER, MD
Staff Rheumatologist
Arthritis-Immunology Center
Philadelphia Veterans
Administration Medical Center
University and Woodland Aves
Philadelphia, PA 19104

1. Bujak JS, Aptekar RG, Decker JL, Wolff SM: Juvenile rheumatoid arthritis presenting in the adult as a fever of unknown origin. *Medicine* 1973; 52:431–444.
2. Baker DG, Schumacher HR, Reginato AJ: Fifteen patients with adults Still's disease: life threatening liver failure in two. *Arthritis Rheum* 1979; 22:590.
3. Bywaters EGL: Still's disease in the adult. *Ann Rheum Dis* 1971; 30:121–133.
4. Elkon KB, Hughes GRV, Bywaters EGL, et al: Adult-onset Still's disease: Twenty-year follow-up and further studies of patients with active disease. *Arthritis Rheum* 1982; 25:647–654.
5. Cush JJ, Leibowitz IH, Friedman SA: Adult-onset Still's disease and inflammatory orbital pseudotumor. *NY State J Med* 1985; 85:110–111.
6. Medsgar Jr TA, Christy WC: Carpal arthritis with ankylosis in late onset Still's disease. *Arthritis Rheum* 1976; 19:232–242.
7. Esdaile JM, Tannenbaum H, Hawkins D: Adult Still's disease. *Am J Med* 1980; 68:825–830.

Arteriovenous malformation of the stomach

The article by Teng and Cho[1] in this issue of the *Journal* is deserving of attention not only because it reminds us of an obscure but potentially deadly anatomic aberration, but also because it permits us to review the methods of diagnosis and therapy for acute gastrointestinal bleeding. Arteriovenous malformation of the stomach was first described in 1884, but received little attention and was rarely reported until Goldman's review of the subject 80 years later.[2] The typical case—a middle-aged patient with massive upper gastrointestinal bleeding, having no previous ulcer diathesis or hematologic abnormality—fits perfectly the present case description. Unfortunately, the clinical course is also paralleled. A blind surgical procedure is performed, following which massive bleeding recurs, often leading to the patient's demise.[2-4] Many of the original cases were not diagnosed until postmortem examination. Endoscopy has seldom proved useful in identifying a bleeding point or establishing the diagnosis; barium studies have not been helpful. However, since the advent of visceral angiography many more cases have been reported prior to operation.

The problem of detecting an arteriovenous malformation of the stomach provides a small but striking example of the difficulty in detecting the source of obscure gastrointestinal bleeding from any condition. An excellent review of possible diagnostic methods and a guide to their proper sequential use in the diagnosis of gastrointestinal bleeding was written by Steer and Silen.[5] Endoscopy as an initial diagnostic maneuver is safe and accurate, especially in cases of upper gastrointestinal bleeding. Radionuclide imaging in a relatively stable patient may be helpful in defining the general area of bleeding and in demonstrating that the bleeding remains active, thus improving the chances of a positive visceral angiogram. Visceral angiography, particularly in cases of active bleeding or in those cases in which endoscopy has failed to establish a source, may be helpful and carries a low rate of major complications. The use of therapeutic visceral angiography, through which embolization or vasopressin infusion is carried out, is a promising approach in many cases.[6] Barium contrast studies, on the other hand, obscure the field, precluding angiographic or endoscopic examination, and may cloud the clinical picture by demonstrating other concurrent lesions that may not themselves be the source of bleeding. It appears that the use of barium contrast studies is limited to those cases in which there is no active or acute hemorrhage.[7]

In cases of an arteriovenous malformation of the stomach, surgery remains the sole effective treatment. Because the blood supply to the stomach is so extensive, attempts to devascularize the lesion via endoscopy or angiography are both hazardous and futile. While either of these therapeutic endeavors may be of temporary benefit, excision of the lesion after precise angiographic localization appears to be the only way to eliminate the problem. Fortunately, multiple arteriovenous malformations of the stomach are rare. In only one case were multiple lesions noted,[8] and that patient has remained free from recurrent bleeding for six years after surgery (personal follow-up).

The case presented by Teng and Cho is typical of arteriovenous malformation of the stomach, including unsuccessful initial surgical management, with patient death as at least an indirect consequence. As these authors and Steer and Silen point out, in patients in whom endoscopy is either not feasible or has failed to establish a diagnosis, visceral angiography should be performed. This allows for a rational plan of management in acute gastrointestinal bleeding.

LAWRENCE M. SHERMAN, MD
Clinical Instructor
Department of Surgery
SUNY at Buffalo
School of Medicine
Buffalo, NY 14214

1. Teng TK, Cho S: Massive gastric hemorrhage from rupture of arterial malformation. *NY State J Med* 1985; 85:112-113.
2. Goldman RL: Submucosal arterial malformation ("aneurysm") of the stomach with fatal hemorrhage. *Gastroenterology* 1964; 46:589-594.
3. Gough MH: Submucosal arterial malformation of the stomach as a probable cause of recurrent severe hematemesis in a 16-year-old girl. *Br J Surg* 1977; 64: 522-524.
4. Richter RM: Massive gastric hemorrhage from submucosal arterial malformation. *Am J Gastroenterol* 1975; 64:324-326.
5. Steer ML, Silen W: Diagnostic procedures in gastrointestinal hemorrhage. *N Engl J Med* 1983; 309:646-650.
6. Athanasoulis CA: Angiographic methods for the control of gastric hemorrhage. *Am J Dig Dis* 1976; 21:174-181.
7. Allen HM, Block MA, Schuman BM: Gastroduodenal endoscopy. Management of acute upper gastrointestinal hemorrhage. *Arch Surg* 1973; 106:450-455.
8. Sherman L, Shenoy SS, Satchidanand SK, Neumann PR, Barrios GG, Peer RM: Arteriovenous malformation of the stomach. *Am J Gastroenterol* 1979; 72: 160-164.

GERIOSCOPY

(AN UNPAID ANNOUNCEMENT)

Geriatrics has lagged in development compared to other clinical disciplines. Notwithstanding the acute need, there are few educational programs, research is limited, and the field lacks a specific technology applicable to the medical diagnosis and treatment of older persons. All this may change due to a breakthrough by biomedical scientists and marketing executives at a major medical center who are perfecting the technique of gerioscopy. This completely invasive procedure, affording minimal risk to the hospital, involves the evaluation of a patient by a board certified gerioscopy specialist utilizing a flexible six-foot binocular panendogeriscope.

Using gerioscopy the specialist is able to perform any of the following: accurately predict the patient's length of stay (on earth) [LOS(oe)]; precisely determine the biopsychosocial requirements of the patient; conveniently plug into a Banzai Personal Computer and through the use of a transcoupler provide a daily digital reduction printout of the projected daily cost-benefit ratio.

Gerioscopy replaces the prior outmoded procedures performed by nurses, social workers, physical therapists, psychologists, and many physicians. These savings almost offset the costs of required technicians and sales managers, and the "hands off, mouth off" feature of the instrument relieves the physician of the need to touch or talk to the patient.

Gerioscopy is fully reimbursable by Medicare and qualifies the patient for a four-week stay at an extended care facility. Gerioscopy II, with a remote teaching head, will be available in mid-1985 and will be user-friendly. Maintenance service contracts will be available. For information write or call:

LYNN P. CARMICHAEL, MD
Department of Family Medicine
PO Box 016700
Miami, FL 33101
(305) 547-6604

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042.*

Recurrent malignant melanoma following a 46-year disease-free interval

TO THE EDITOR: Malignant melanoma can run a strikingly unpredictable and erratic course.[1] A patient was seen at Staten Island Hospital with recurrent melanoma 46 years following treatment for Stage II disease.

Case Report. This patient was first seen in 1927 at age 32 with a melanoma underneath the nail of the right index finger. Infraclavicular adenopathy and a nodule on the right chest wall were discovered. External radiation was administered, with disappearance of the masses. The right index finger was subsequently amputated in 1931. Records still in existence do not further describe the histopathologic features of the primary lesion.

The patient's course was reported by George Pack, MD in a series on subungual melanoma published in 1939 (case 10).[2] The case is also mentioned in a more recent book by Ariel.[3]

The patient remained well until 1977, when he was admitted to Staten Island Hospital for evaluation of anemia and splenomegaly. There was no evidence of cutaneous melanoma, and the patient did not recall any previous skin lesion which had regressed. A radionuclide scan of the spleen showed a filling defect. At laparotomy, the spleen was found to be infiltrated by melanoma. The patient died in 1978. An autopsy showed recurrent melanoma invading the abdominal viscera. No evidence of a new primary lesion was found.

Discussion. There have been several published series describing large groups of patients with melanoma that have attempted to correlate clinicopathologic staging with different treatment methods and survival statistics.[3-5] Follow-up data concerning patients with disease-free intervals of more than 15 years are restricted to anecdotal case reports. Briele et al[6] reported three patients with melanoma involving the extremities who had disease-free intervals of 20 to 23 years before recurrence in regional lymph nodes. Hendrix[1] reported on four patients with melanoma who had stage I disease and then developed metastases five or more years following initial treatment. One of these patients was free of disease for 20 years before developing a non-fatal recurrence in regional lymph nodes. Koh et al[7] described two patients with stage I melanoma who had disease-free intervals of 14 and 18 years before recurrence.

Melanoma is one of the few tumors that frequently involves the spleen.[8] In one series, 36% of the patients were found to have splenic metastases at autopsy.[9]

Except for the chronology of events, the clinical course of this patient was not atypical. He initially had a subungual melanoma, which, in retrospect, most likely involved regional nodes, followed by infiltration of the spleen and death due to disseminated intraabdominal metastases. However, the 46-year disease-free interval is extraordinary, and carries with it a significant lesson. No patient with treated stage II melanoma can be considered cured, even if the patient remains well for five or even 10 years following therapy; such patients require lifelong follow-up for signs of recurrence.

LEON ROSENKRANZ, MD
395 South End Ave-Apt 12L
New York, NY 10280
CONRAD SCHROEDER, MD
Department of Medicine
Staten Island Hospital

1. Hendrix RC: Unusual survival patterns of patients with metastatic melanoma. *Cancer* 1969; 24: 574–576.
2. Pack GT, Adair FR: Subungual melanoma. *Surgery* 1939; 5:47.
3. Ariel IM: *Malignant Melanoma.* New York, Appleton-Century-Crofts, 1981.
4. Callery C, Cochran AJ, Roe DJ et al: Factors prognostic for survival in patients with malignant melanoma spread to the regional lymph nodes. *Ann Surg* 1982; 196:69–75.
5. Shaw HM, McGovern VJ, Milton GW, Farago GA, McCarthy WH: The female superiority in survival in clinical stage II cutaneous malignant melanoma. *Cancer* 1982; 49:1941–1944.
6. Briele HA, Beattie CW, Ronan SG, Chaudhuri PK, Das Gupta TK: Late recurrence of cutaneous melanoma. *Arch Surg* 1983; 118:800–803.
7. Koh HK, Sober AJ, Fitzpatrick TB: Late recurrence (beyond ten years) of cutaneous malignant melanoma. *JAMA* 1984; 251:1859–1862.
8. DeVita VT, Fisher RI: Natural history of malignant melanoma as related to therapy. *Cancer Treat Rep* 1976; 60:153–157.
9. Nathanson L, Hall TC, Farber S: Biological aspects of human malignant melanoma. *Cancer* 1967; 20:650–655.

Solitary nonparasitic liver cysts

TO THE EDITOR: Solitary nonparasitic cysts of the liver are rare. The first report has been attributed to Michel,[1] who in 1856 documented a large cyst in the liver of a 60-year-old man. The majority of cysts are without symptoms; when symptoms do arise, they mimic more common diseases. A diagnosis may be established prior to surgery in the majority of cases by physical examination, ultrasonography, and negative echinococcal and amebic serum titers.

Case Report. A 27-year-old woman sought treatment for anorexia for one day and increasingly severe pain in the right upper quadrant. The pain was associated with chills, but she denied any nausea, vomiting, diarrhea, or change in the color of her stools or urine.

Three years previously a similar episode had been diagnosed as acute cholecystitis. The patient had no history of hepatitis, jaundice, or trauma. She was not taking any medications and had never traveled outside the continental United States.

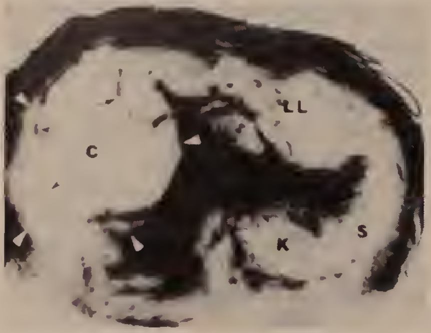
When first evaluated, the patient was in obvious distress. Her temperature was 36.8°C; blood pressure, 120/70 mm Hg; pulse, 108/min. The skin and sclera were anicteric. The abdomen was tender, and Murphy's sign was present. No masses were palpable.

The white blood cell count was 20,200/mm^3, with 79% polymorphonuclear leukocytes, 9% band forms, 5% lymphocytes, and 7% monocytes. The results of serum chemistry studies were as follows: total bilirubin, 1.0 mg%; SGPT, 24 mU/mL; SGOT, 18 mU/mL; alkaline phosphatase, 89 mU/mL. A chest film demonstrated an elevated right hemidiaphragm. Results of urinalysis were within normal limits. A tentative diagnosis of acute cholecystitis was made, and intravenous ampicillin was administered.

The following day the patient was febrile, with temperatures to 38.6°C. An ultrasound revealed a thick-walled cyst, 17 cm in diameter, compatible with echinococcal infection (Fig). Intravenous metronidazole was added to the antibiotic regimen. The patient's fever and pain diminished. Amebic and echinococcal serum titers were reported as negative. A computed tomographic (CT) scan demonstrated the presence of a cyst replacing the right lobe of the liver.

On celiotomy, a cyst containing loculated straw-colored fluid was noted. There were multiple septa within the cyst, and a pouch was formed by resecting the anterior wall and suturing the cut edges to the adjacent liver. Drains were removed on the 10th day after surgery, and the patient was discharged from the hospital four days later.

Cultures of the cystic fluid did not contain bacteria or parasites. A paraffin section of the cyst wall demonstrated fibrotic tissue with no cellular lining, compatible with the diagnosis of a solitary nonparasitic liver cyst.



Abdominal ultrasound demonstrates a cyst in the right lobe of the liver. C, cyst; LL, left lobe of liver; K, kidney; S, spleen. Light arrows depict cyst border; dark arrows indicate septa within the cyst.

Discussion. Solitary nonparasitic liver cysts occur infrequently, with less than 500 documented cases appearing in the literature.[1,2] Necropsy statistics indicate that these cysts are usually asymptomatic. Eliason and Smith[3] reported only two cases among 211,046 hospital admissions, while at the same institution 28 such cysts were identified in 20,000 autopsies.[3]

The pathogenesis of solitary nonparasitic liver cysts remains obscure. Moschcowitz[4] described the presence of aberrant bile ducts in cystic livers and ascribed the formation of cysts to obstruction or inflammatory hyperplasia of these ducts.

Solitary nonparasitic liver cysts are reported more frequently in women than in men, and the majority of lesions are detected in the fourth through seventh decades.[1,2,5] The epithelial lining of the cyst is usually comprised of cuboidal cells; however, as in this patient, there may be no cellular element present.[1,5] The diameter of these cysts may vary from 2 cm to 3 cm, or they can fill the entire abdomen.[2,5] Recently, an association between solitary nonparasitic liver cysts and benign bile duct polyps has been reported.[6]

The symptoms caused by uncomplicated solitary nonparasitic liver cysts are often related to cyst size. Distention, nausea and vomiting, and upper abdominal pain are common complaints.[2,5] A physical examination will usually reveal an anicteric patient with an abdominal mass.[1,2,5]

Abdominal ultrasonography is the best diagnostic tool to establish the presence of liver cysts. Serologic tests are necessary for differentiation among echinococcal cysts, amebic abscesses, and solitary nonparasitic liver cysts. The advent of CT scanning has occurred after the majority of reported cases, and its application awaits further evaluation. Angiography does not aid in determining cyst etiology, but may be helpful in planning therapy if hepatic resection is contemplated.

Complications arising from solitary nonparasitic liver cysts are unusual. Although patients may have fever and chills, bacterial superinfection has not been seen. However, hemorrhage into the cyst, intra-abdominal rupture, torsion of the cyst pedicle, biliary obstruction, and cyst-enteric fistulas have been reported.[1,3,5] Additionally, squamous cell carcinoma arising in the cyst lining has been documented.[7]

The treatment of symptomatic solitary nonparasitic liver cysts ranges from aspiration to liver resection; options include extirpation with or without hepatic resection, marsupialization, incision and drainage, and cyst-enterostomy.[1,2,5] Percutaneous needle drainage has been advocated for small (1–3 cm) and moderate (3–10 cm) size cysts.[1,5] Larger cysts require surgical intervention. Bile-containing cysts should be drained internally with a Roux loop.[1,2] Asymptomatic cysts need not be treated.

STEVEN M. KELLER, MD
ALISAN B. GOLDFARB, MD
Department of Surgery
The Mount Sinai Hospital
One Gustave L. Levy Place
New York, NY 10029

ZVI ZISBROD, MD
Department of Cardiothoracic Surgery
Maimonides Medical Center
4802 10th Ave
New York, NY 11219

1. Hadad AR, Westbrook KC, Graham GG, Morris WD, Campbell GS: Symptomatic nonparasitic liver cysts. *Am J. Surg* 1977; 134:739–744.

2. Sanfelippo PM, Beahrs OH, Weiland LH: Cystic disease of the liver. *Ann Surg* 1974; 179:922–925.

3. Eliason EC, Smith DC: Solitary nonparasitic cyst of the liver. *Clinics* 1944; 3:607–621.

4. Moschcowitz E: Nonparasitic cysts (congenital) of the liver, with a study of aberrant bile ducts. *Am J Med Sci* 1906; 131:679–699.

5. Flagg RS, Robinson DW: Solitary nonparasitic hepatic cysts: Report of the oldest known case and review of the literature. *Arch Surg* 1967; 96:964–973.

6. Austin EH, Mitchell GE, Oliphant M, et al: Solitary hepatic cyst and benign bile duct polyp: a heretofore unheralded association. *Surgery* 1981; 89:359–363.

7. Bloustein PA, Silverberg SG: Squamous cell carcinoma originating in an hepatic cyst: Case report with a review of the hepatic cyst-carcinoma association. *Cancer* 1976; 38:2002–2005.

Psychiatric approach to drug abuse

TO THE EDITOR: In response to the commentary by Wetli (*NY State J Med* 1984; 84:587) on drug abuse prevention, attention should be called to another facet of the drug abuse issue. The prevention of drug abuse through education and the recognition of its medical complications are important aspects of a medical approach. Psychiatric treatment is also an approach to this problem not mentioned by Wetli, which may be useful.

Many, if not most, drug abusers, and some occasional users, have psychiatric problems; and the recognition of drug abuse as possibly indicating underlying psychiatric illness is essential for helping these patients. Although psychiatric treatment of such patients is often difficult and frustrating, selected patients can respond to therapeutic intervention.[1] The referral of such patients for psychiatric evaluation and treatment, if indicated, should be regarded as an integral part of a medical approach to drug abuse. More and more psychiatric training programs are including training in this field, and new drug and other forms of therapy can be helpful in selected cases. Decreased drug use and morbidity and improved functioning can be a result of successful psychiatric treatment.

Many researchers have found a number of psychosocial and psychiatric correlates of drug abuse.[2,3] Since the etiology of drug abuse is probably multifactorial, a multifactorial approach to dealing with this issue is likely to be the most productive.

The prevention of drug abuse is indeed an important approach to a major societal problem, and therapeutic intervention by psychiatrists with experience in this area may be of value in dealing with this difficult medical-psychiatric challenge.

DAVID W. BROOK, MD
Assistant Clinical Professor
Department of Psychiatry
The Mount Sinai Medical Center
One Gustave L. Levy Place
New York, NY 10029

1. O'Brien CP, Woody GE, McLellan AT: Psychiatric disorders in opioid-dependent patients. *J Clin Psychiatry* 1984; 45:9–13.
2. Kandel DB: Drugs and drinking behavior among youth. *Ann Rev Sociol* 1980; 6:235–285.
3. Brook JS, Whiteman M, Gordon AS, Brook DW: Paternal determinants of female adolescents' marijuana use. *Dev Psychol* 1984; 20:1032–1043.

Candida tropicalis papillary necrosis treated with ketoconazole

TO THE EDITOR: The spectrum of candidal disease in the genito-urinary tract is described in the medical literature.[1-7] Diabetes mellitus, corticosteroids, antineoplastic agents, and antibiotics are the most frequent predisposing factors. Genito-urinary manifestations of the disease are primary or secondary pyelonephritis, papillary necrosis, cortical abscess, fungal ball formation with potential obstruction, cystitis, or asymptomatic candiduria. Intravenous amphotericin B has been the therapeutic mainstay of fungal upper urinary tract infections. We report on a diabetic man with a persistent candidal papillary necrosis who was cured with the oral agent ketoconazole.

Case Report. A 37-year-old insulin-dependent man was admitted to the hospital with fever, right flank pain, pyuria, and hematuria. The physical examination showed a chronically ill man with bilateral flame-shaped hemorrhages and tenderness in the right upper quadrant. The patient's blood urea nitrogen level was 31 mg/dL; creatinine, 1.4 mg/dL; and random blood sugar, 322 mg/dL. An initial urine culture grew mixed urethral flora. Cultures of blood taken on admission grew no bacteria. Two urine cultures grew *Candida tropicalis.* A renal sonogram revealed right renomegaly, and retrograde pylography indicated mild dilation of the right collecting system without obstruction.

One week later, the patient passed debris which was identified as fungi by a methenamine stain. *Candida tropicalis* was repeatedly cultured from the urine. It was resistant to miconazole but sensitive to 5-flucytosine, which was administered at 1.0 g orally four times a day. A four-day course of amphotericin B bladder irrigation was also instituted. Repeat retrograde pyelography revealed papillary necrosis. The patient was discharged one week later.

The patient sought treatment for right flank pain three weeks after discharge, having discontinued his medication. Urine cultures again grew *Candida tropicalis*, which was documented to be resistant to 5-flucytosine. He was discharged without antifungal therapy after spontaneous resolution of the symptoms.

The patient was readmitted to the hospital six weeks later with worsening right flank pain and *Candida tropicalis* in his urine. Renal sonography again demonstrated right hydronephrosis. Subsequent analysis of the urinary debris revealed necrotic tissue with large numbers of budding yeast and hyphae. A diagnosis of *Candida tropicalis* papillary necrosis was made. It was decided to avoid parenteral amphotericin B because this patient had pre-existing renal disease. Four weeks of oral ketoconazole therapy was begun at a dosage of 400 mg daily. His condition improved rapidly. There were no further urine cultures positive for *Candida tropicalis* after treatment with ketoconazole was commenced. Follow-up sonography showed gradual resolution of the right hydronephrosis.

Discussion. This is an unusual case of renal papillary necrosis associated with *Candida tropicalis*, which has been cured with oral ketoconazole.[8-11] Severe infection or vascular disease of the renal pyramids may result in papillary necrosis. Papillary necrosis is most frequently associated with pyelonephritis in the setting of diabetes mellitus. Other less common predisposing conditions include analgesic abuse, macroglobulinemia, obstructive uropathy, sickle cell disease, and cirrhosis.[12] The clinical spectra of papillary necrosis and acute pyelonephritis may be indistinguishable. An indolent course for months to years punctuated by episodes of acute illness or a fulminant process with shock may occur with papillary necrosis.

Five-flucytosine was initially administered to this patient, but the organism became resistant to this agent during therapy. This is a frequently reported phenomenon. Monitoring of serum levels is recommended in patients with renal disease in order to reduce the likelihood of dose-related bone marrow depression and hepatotoxicity. Renal candidiasis is suspected when funguria and pyuria with large numbers of mycelial forms persist. Diabetes mellitus, urinary catheters, preceding antibiotic therapy, coexistent bacterial urinary tract infections, and corticosteroid therapy are common predisposing factors to funguria. We presume that this patient's enlarged kidney was due to obstruction by "aggregated fungus" and sloughed tissue. The candidiasis was likely limited to the collecting system.

It may be difficult to differentiate candidal infection of the renal pelvis from lower urinary tract involvement. Serologic tests are of limited value. When funguria does not resolve and the above coincident factors are controlled or eliminated, an upper tract infection must be suspected. A radiographic or sonographic evaluation is then indicated.

Amphotericin B results in a high frequency of renal failure as well as other serious toxicity. Its usage could have resulted in the need for chronic dialysis in this patient, who had chronic renal failure. A nephrectomy would have been considered if the ketoconazole failed to eradicate the fungal infection.

Effective treatment of this infection resulted in the avoidance of dialysis and, possibly, a nephrectomy. In addition, ketoconazole therapy precluded the need for prolonged intravenous therapy with amphotericin B, with or without admission to the hospital.

PAUL S. COHEN, MD
ROBERT L. SCHEER, MD
Department of Internal Medicine

MICHAEL J. BLOOM, MD
GARY J. KOLANCHIK, MD
JAMES B. TUCKER, MD
Department of Family Practice
St. Joseph's Hospital Health Center
301 Prospect Ave
Syracuse, NY 13203

1. Rovinescu I, Belanger PM, Lapalme R: Monolial infection of the urinary bladder. *Urol Int* 1968; 23:428–435.
2. Schönebeck J, Winblad B: Primary renal Candida infection. *Scand J Urol Nephrol* 1971; 5:281–284.
3. Margolin HN: Fungus infections of the urinary tract. *Semin Roentgenol* 1971; 6:323–330.
4. Fisher J, Mayhall G, Duma R, Shadomy S, Shadomy J, Watlington C: Fungus balls of the urinary tract. *South Med J* 1979; 72:1281–1284, 1287.
5. Janosko EO, McRoberts JW: Evaluation and treatment of urinary candidiasis. *South Med J* 1979; 72:1578–1581.
6. Tennant Jr FS, Remmers Jr AR, Perry JE: Primary renal candidiasis. Associated perinephric abscess and passage of fungus balls in the urine. *Arch Intern Med* 1968; 122:435–440.
7. Harbach LB, Burkholder GV, Goodwin WE: Renal candidiasis: a cause of anuria. *Br J Urol* 1970; 42:258–264.
8. Goodwin LS, Gilman A: *The Pharmacological Basis of Therapeutics*, 6th ed. New York, Macmillan, p 1237.
9. World Medical Press and Review: Drug treatment, one new drug in brief, ketoconazole. *Hosp Phys* 1981; 17:24.
10. Abramowicz M: Drugs for treatment of systemic fungal infections. *The Medical Letter*. April 2, 1982, p 38.
11. Graybill JR, Drutz DJ: Ketoconazole: a major innovation for treatment of fungal disease. *Ann Intern Med* 1980; 93:921–923.
12. Eknoyan G, Qunibi WY, Grissom RT, Tuma SN, Ayus JC: Renal papillary necrosis: an update. *Medicine* 1982; 61:55–73.

ADDITION

Arthur H. Wolintz, MD, author of "Neuro-ophthalmologic aspects of occlusive cerebrovascular disease" (*NY State J Med* 1985; 85:18–23), is also the Director of the Department of Ophthalmology and Neuroophthalmology, Kingsbrook Jewish Medical Center, Brooklyn, NY.

Tubal sterilization with the Falope ring in an ambulatory-care surgical facility

Steven G. Kaali, MD, Robert Landesman, MD

ABSTRACT. Five hundred and twenty laparoscopic tubal sterilizations with Falope rings were performed at the Women's Medical Pavilion in Dobbs Ferry, NY, a freestanding surgical unit. Three hundred and eighty elective and 140 post-abortal sterilizations over a four-year period are reviewed. Meticulously maintained laparoscopy equipment, well trained surgeons, supporting anesthesia, and operating and recovery room personnel were available; no major complications were noted. Three subsequent intrauterine pregnancies, one ectopic pregnancy, and 12 "technical failures" occurred in this series. This study indicated that outpatient sterilization using the Silastic banding technique compares favorably with other sterilization procedures. Suggestions for patient selection, counseling, and surgical technique, and guidelines for organizing a similar ambulatory surgical unit are discussed.

(NY State J Med 1985; 85:98–100)

INTRODUCTION

In the United States, tubal sterilization is the most commonly performed elective, intra-abdominal surgical procedure of reproductive aged women.[1] More than 750,000 patients annually have some form of tubal ligation. Therefore, any complications or morbidity associated with this procedure could have a major impact on public health. There is a growing concern in this country about the rapid rise in the cost of health care. It is estimated that 10% of the gross national product is spent on health related services, and this figure is expected to exceed 20% by the end of this century.[2] Ambulatory surgery is one mechanism for achieving significant cost containment. This report presents our experience with 520 consecutive female sterilizations utilizing the Falope ring on an ambulatory basis.

PATIENTS AND METHODS

During the period January 1, 1980 to December 31, 1983, 520 consecutive patients requesting sterilization underwent Falope ring sterilization at this freestanding surgical unit. The patients were of reproductive age, were healthy, and desired voluntary sterilization. During the initial visit a history, physical examination, hematocrit evaluation, urinalysis, blood type and Rh factor, pregnancy test, and Papanicolaou smear were required for each patient. All candidates received a 30-minute counseling session and signed an informed consent after having read a detailed pamphlet describing the procedure. After surgery, the patients returned for a follow-up visit in one week and were referred back to their private physicians with written instructions to contact the surgical unit if any unexpected event developed (pregnancy, menstrual disorders, or persistent pain). Eighty-nine patients who had had previous abdominal surgery (17.1%) were also included.

Four hundred and thirty-five patients requested general anesthesia (83.6%) with endotracheal intubation and assisted respiration; 85 procedures were performed under local stand-by anesthesia (16.4%). The patients were admitted to the surgical unit the same day; they underwent an anesthetic evaluation and their desire for sterilization was reaffirmed. Three hundred and two patients (58%) required a dilation and curettage; 140 patients (26.9%) requested, at the same time, first trimester pregnancy termination by suction curettage. Under general anesthesia, pelvic examination was performed, and the patient was placed in the steep Trendelenburg position. The Wisap intrauterine manipulator was then attached to the cervix by 60 mm Hg of suction. The Verres needle was inserted through a 1-cm incision at the lower rim of the navel. A minimum of one liter of CO_2 was delivered prior to introducing the 12-mm metal trocar cannula. After inserting the laprocator (a laparoscopic single incision ring applicator) the pelvic organs were visualized. The uterus was elevated and the thinnest portion of the tube was grasped by the tong and drawn into the central cylinder using a to-and-fro motion. The outer cylinder was pushed forward, thereby placing the Falope ring onto the knuckle of the tube. The instrument was withdrawn, and the tube was checked for blanching and to make certain that the length of the knuckle was acceptable (Figs). After releasing the CO_2 from the abdominal cavity, the periumbilical incision required for the trocar was closed with two interrupted chromic sutures.

RESULTS

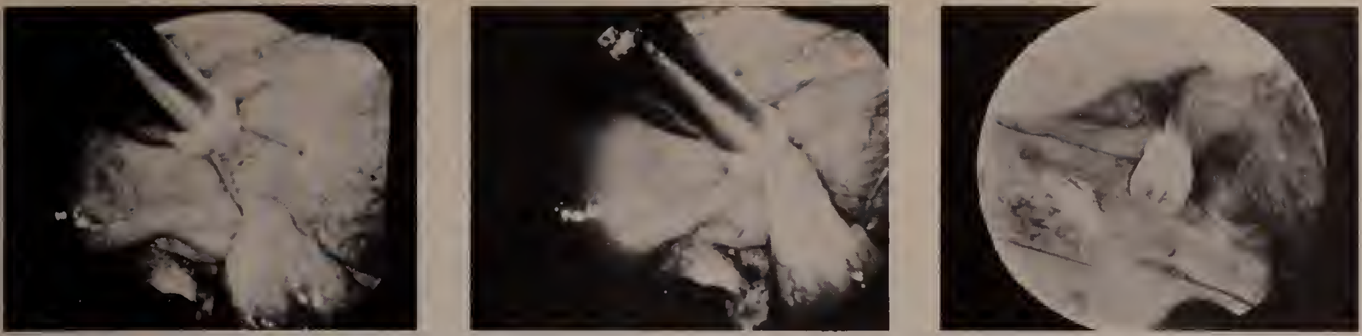
The patients ranged in age from 21 to 50 years of age. Of the 520 patients, 309 (59%) were between 31 and 40 years old. The age, parity, and weight are listed in Table I. In the age group between 20 and 26 years all 13 patients were multiparous and could not be dissuaded from the operation. Two of the six women between 46 and 50 years of age were first seen with an early pregnancy and requested concomitant first trimester abortion. The other four women in the oldest age category either had ovulatory cycles, a history of pregnancy within 3 years, or had a family history of menstruation into the middle of the sixth decade.

Pain after surgery requiring narcotics was noted in 125 patients (24%) in the recovery room. No patients were hospitalized, and 450 women (86.5%) spent between one and six hours in the recovery room before being discharged from the hospital (Table II). There were no major complications during or after surgery, as defined by the stan-

---

From the Departments of Obstetrics and Gynecology, College of Physicians and Surgeons of Columbia University (Dr Kaali) and The New York Hospital-Cornell Medical Center (Dr Landesman), New York, NY.

Address correspondence to Dr Landesman, 530 E 72nd St, New York, NY 10021.



Left, the fallopian tube is identified; Center, the tube is drawn into the central cylinder; Right, the outer cylinder is pushed forward, placing the Falope ring onto the tube.

dard categories of the Centers for Disease Control[8]—unintended major surgery, transfusion, febrile morbidity, life threatening event, hospitalization, or death. Twelve "technical failures" were encountered in this series. This terminology refers to patients in whom the originally planned procedure could not be completed for a variety of reasons. Four patients had tubes that were too thick or adhered tubes (0.7%), and it was impossible to draw them into the central cylinder of the laprocator. Through a second incision, Secu-clips (Cryomedics, Inc, Bridgeport, CT) were placed on both fallopian tubes. On one occasion, mini-laparotomy (0.2%) was required to free omental adhesions from the left adnexal area after successfully applying the Falope ring to the right side. Seven patients had accidental tubal transection (1.3%). In two patients, a small (3 cm) contained broad ligament hematoma resulted. One patient required electrocauterization of the torn tube for bleeding; and in six cases the Falope ring was placed on each end of the transected tubes. The hematomas remained limited after observation for 15 minutes. Three uterine perforations (0.6%) were recorded: one was inflicted by the Wisap intrauterine cannula; two resulted from currettage.

At the time of laparoscopy no bleeding or hematomas were noted, and management consisted of the usual recovery room observation. There were four subsequent pregnancies (0.8%) in this study group. One patient had an ectopic pregnancy (0.2%) 14 months after sterilization. The patient was hospitalized and a left salpingectomy was performed. Three years after the original procedure, large myomata with an eight-week gestational sac developed in one patient. The patient elected to undergo a hysterectomy. Review of the operative report of the sterilization revealed difficulties in applying the Falope ring to the right side.

Two patients were re-admitted to this unit for first trimester abortions and to undergo repeat laparoscopic tubal ligation. At the time of laparoscopy, the tubal segments were 5 mm apart. The ring was located between the separated segments and was covered by peritoneum. Falope rings were applied to the proximal tubal segments. These cases constituted true failures and probably were due to recanalization or fistula formation. Twenty-two intrauterine devices were removed and 140 first trimester abortions were performed at the time of laparoscopy. If infection was evident, antibiotic therapy was begun and surgery was postponed. In this group, pelvic abscess, peritonitis, or infected hematoma did not occur. There were no complications due to anesthesia; however, mild thrombophlebitis developed in the forearm of one patient at the intravenous site; it was self limited.

Discussion

Methods of female sterilization include suprapubic minilaparotomy,[3] laparoscopy either by Falope ring,[4] spring clip,[5] bipolar coagulation,[6] or hysteroscopic directed silicone rubber plugs.[7] Minilaparotomy entails an abdominal incision and, in selected cases, may be performed in an outpatient setting. However, it involves laparotomy and is poorly suited for the retroverted displaced uterus. The recovery period is usually longer than for laparoscopy. Bipolar cauterization or any type of high frequency current may result in burns and usually destroys the tube and its blood supply, making reanastomosis more difficult. In our experience, use of the Falope ring and the Hulka clip are the best techniques. If the Falope ring is unsuitable in a thickened tube, the Hulka clip may be applied. Silicone plugs, which are directed to the tubal ostium via hysteroscopic guidance, are in the experimental phase. Reed and Erb[7] have been successful in occluding the tubes with this technique in 90% of the cases. Because our experience and expertise was primarily with the Falope ring, it was used in this series. Since described by Yoon,[4] during the past decade one million procedures with the Falope ring have been reported. The use of Silastic bands (Kli, Inc, Ivyland, PA) in hospital settings to occlude the fallopian tubes offers a safe and effective method for permanent birth control.

The results of this study indicate that laparoscopic tubal ligation with the Falope ring is a suitable procedure for outpatient female sterilization. The procedure should be offered to mature women with no further desire of pregnancy. In this series of 520 patients, 31 (6%) were nulliparous. All these women were career oriented and had been

TABLE I. Patients Undergoing Falope Ring Sterilization

Age (yr)	No. of patients		Parity	No. of patients		Weight (lbs)	No. of patients	
20-26	13	(2.5%)	0	31	(6.0%)	100-120	51	(9.8%)
26-30	152	(29.2%)	1	112	(21.5%)	121-140	192	(36.9%)
31-35	136	(26.1%)	2	270	(51.9%)	141-160	144	(27.7%)
36-40	172	(33.1%)	3	72	(13.8%)	161-180	65	(12.5%)
41-45	41	(7.9%)	4	30	(5.8%)	181-200	50	(9.6%)
46-50	6	(1.2%)	5	5	(1%)	over 200	18	(3.5%)
Total	520	100.0%		520	100.0%		520	100.0%

TABLE II. Time in Recovery Room Before Discharge

Time (hr)	No. of Patients	Percent
0-1	0	0
1-2	48	9.2
2-3	180	34.6
3-4	270	51.9
4-5	20	3.9
5-6	2	0.4
Total	520	100.0

TABLE III. Total Cost of Falope Ring Sterilization Excluding Surgical Fee

	Inpatient cost ($)	Outpatient cost ($)
Room	332	0
Anesthesia	315	50
Operating room charge	200	40
Recovery	150	30
Pharmacy	50	10
Laboratory	120	45
Central Service	70	15
Equipment depreciation	20	20
Total	$ 1257	$ 210

married for 10 years or longer. Each individual was committed not to raise a family and desired long-term, efficient, and safe contraception.

Presterilization counseling must be an integral part of all ambulatory sterilization programs. Detailed explanations should include alternative methods of birth control, including vasectomy; risks associated with reversibility of the procedure; potential failure; risks of potential complications; and freedom to withdraw the consent. There is a need for witnessed documentation and strict confidentiality. Each patient participated in a 30-minute woman-to-woman counseling session; well informed and motivated patients were found to be better candidates for outpatient surgery. Great caution must be exercised in the selection of patients for ambulatory surgery, especially when general anesthesia is administered. All patients should be class I (a healthy patient for an elective operation) as classified by the American Society of Anesthesiologists. To implement a successful outpatient surgical program, the facility should be in close proximity to a general hospital where the surgeon has full unrestricted privileges. There should be an emergency plan for unintended laparotomy, hemorrhaging or complications due to anesthesia. The laparoscopic tubal ring procedure requires training to develop technical skill and knowledge of indications and contraindications to the surgery. The physician and all personnel should be familiar with the equipment, including handling and cleaning. To avoid technical problems, two complete sets of laparoscopic instruments should be available at all times. Pelvic or abdominal pain after surgery has been reported more often when the Falope ring is utilized[9] as compared to electrosurgery. Subjective pain is a difficult symptom to evaluate; 24% of the patients in this study required sedation after surgery, with no effect on the time of final discharge from the recovery room. Transection of the fallopian tubes, with or without mesosalpingeal bleeding, may be serious. Drawing the tubes into the central cylinder of the laprocator is undoubtedly the most critical part of the operation, and only surgical skill and experience can reduce the risk. The incidence of tubal transection varies between 2% and 7% in the literature.[10,11] Failure of Silastic banding has been calculated to occur in about one per 100 women during the first two years. Although 25% of these patients did not have adequate follow-up, the pregnancy rate (0.8%) compares favorably with a collected series of 4,707 cases (1.8%) reported by McCann.[12]

The advantage of using the Falope ring is to eliminate electrical hazard, limit tubal damage, permit reversibility, and implant the smallest foreign body.[13] Table III shows the significant difference in cost between inpatient and ambulatory laparoscopic tubal ligation. Considering that 750,000 such procedures are performed annually in the United States, the outpatient approach can result in significant cost containment. A similar conclusion was reached by Greenspan et al[14] who collected data from 141 free-standing, ambulatory-care surgical facilities that performed 16,500 tubal sterilizations in 1980. Laparoscopic female sterilization with the Falope ring is a safe, simple, and effective method for permanent birth control. This outpatient approach provides equal safety, reduced psychological trauma, and substantial savings to the patient and the community.

REFERENCES

1. DeStefano F, Greenspan JR, Ory HW, Peterson HB, Mace JM, Smith JC: Demographic trends in tubal sterilization: United States, 1970-1978. *Am J Pub Health* 1982; 72:480-484.
2. Ryan G (ed): *Ambulatory Care in Obstetrics and Gynecology.* New York, Grune & Stratton, 1980.
3. Penfield AJ: Minilaparotomy for female sterilization. *Obstet Gynecol* 1979; 54:184-188.
4. Yoon IB, King TM: The laparoscopic Falope Ring technique. *Adv Plann Parent* 1975; 10:154-159.
5. Hulka JF, Mercer JP, Fishburne JI, et al: Spring clip sterilization: one year follow-up of 1,079 cases. *Am J Obstet Gynecol* 1976; 125:1039-1043.
6. Gunning JE, Tomasulo JP, Garite T: Laparoscopic tubal sterilization using thermal coagulation. *Obstet Gynecol* 1979; 54:505-509.
7. Reed TP, Erb RA: Tubal occlusion with silicone rubber: an update. *J Reprod Med* 1980; 25:25-28.
8. DeStefano F, Greenspan JR, Dicker CD, Peterson HB, Strauss LT, Rubin GL: Complications of interval laparoscopic tubal sterilization. *Obstet Gynecol* 1983; 61:153-158.
9. Leggat IT, Barr W: Post-operative analgesia following laparoscopic sterilization. *Scott Med J* 1979; 24:220.
10. Cunnanan RG, Courey NG, Lippes J, et al: Complications of laparoscopic tubal sterilization. *Obstet Gynecol* 1980; 55:501-505.
11. Levinson CJ, Daily HI, Marko MW, Richardson DC: Nonelectric laparoscopic sterilization: Experience with a silastic band. *Obstet Gynecol* 1976; 48: 494-496.
12. McCann MF, Cole LP: Laparoscopy and minilaparotomy: two major advances in female sterilization. *Stud Fam Plann* 1980; 11:119-127.
13. Yoon IB: The silicone ring, in Phillips JM (ed): *Laparoscopy.* Baltimore, Williams & Wilkins, 1977, pp 174-178.
14. Greenspan JR, Phillips JM, Ruben GL, Rhodenhiser EP, Ory HW: Tubal sterilizations performed in freestanding, ambulatory-care surgical facilities in the United States in 1980. *J Reprod Med* 1984; 29:237-241.

Chlorpromazine in experimental gastric ulcers induced by restraint and cold stress

CHARLES C. CANVER, MD

ABSTRACT. The influence of chlorpromazine, a phenothiazine compound, on the development of gastric ulcers in rats exposed to restraint and cold stress was investigated. The effect of this agent on gastric acidity was also examined. In three random groups of rats arbitrarily given CPZ, in low (2.5 mg/100 g), medium (5 mg/100 g), and high doses (10 mg/100 g) 30 minutes prior to stress, CPZ inhibited the development of gastric ulcers. CPZ induced characteristic cataleptic immobility in higher doses that resulted in lowered vulnerability to the restraint and cold stress. In low, medium, and high doses, CPZ increased titratable gastric acidity. This secretory influence during stress seems to be predominant in the mechanism of protective action of this drug. However, this study also points to the possibility that CPZ could exert its ulcer-protecting effects through other unexplained mechanisms.

(NY State J Med 1985; 85:101-102)

INTRODUCTION

Stress ulcers most often occur in individuals who are critically ill as a result of multiorgan system failures. Their occurrence in these patients demands aggressive treatment. Because the cause of such ulcers is not known, there is no specific therapy.

Numerous hypotheses have been postulated to explain the mechanism of development of gastric mucosal hemorrhagic ulcers in rats subjected to stress. This experimental stress model in rats shares several features with stress ulcers in human subjects. The stress of cold exposure and physical restraint leads to widespread erosion of the acid-secreting areas of the stomach. These erosions are linear or punctate, lie along the ridges of the gastric rugae, and are associated with bleeding, but do not appear to be associated with hypersecretion of acid.[1]

In this study, chlorpromazine (CPZ) was tested for its ability to protect against cold-restraint induced gastric erosions and its effect on whether severe stress induces the development of biochemical and morphologic changes in rats.

METHODS

Thirty-five male Swiss albino rats, weighing 150 g to 200 g, were used in the stress ulcer study. The rats were fed a standard diet of commercial rat chow and were housed in a room maintained at a constant temperature and with 12-hour light/dark cycles. They were conditioned to the room environment for at least 15 days prior to inclusion in the study. For the 48 hours prior to the experiments, all animals were deprived of food, but not of water, and were housed in individual cages with wide wire bottoms to prevent coprophagy. The rats were divided into five groups of seven rats each. Group 1 animals were the unstressed, untreated control group; group 2 rats received 2 mL saline administered intramuscularly; group 3 rats received low-dose CPZ, 2.5 mg/100 g body weight; group 4 rats received medium-dose CPZ, 5 mg/100 g; group 5 was given high-dose CPZ, 10 mg/100 g. The CPZ doses in the experimental rat groups were arbitrarily selected, and the drug was given intramuscularly 30 minutes prior to restraint in all subjects.

All rats were stressed using a modification of the method of Brodie and Hanson.[1] The animals were lightly anesthetized with ether. They were fixed on their backs to boards by threads tied tightly around the limbs. When the rats recovered from the anesthesia, the threads were tightened so that they could not move. The rats were further stressed using a modification of the procedure described by Brodie and Valitski[2] which combines the use of cold with starvation and restraint. Thus, the restrained rats were placed in a cold room (4°C) for four hours. Physical signs of stress among exposed rats were observed and recorded. Observations were made 60 min prior to the onset of restraint and were repeated every 30 min for the duration of the experiment. The possible inaccuracies inherent in these subjective assessments were minimized by ensuring that they were made by the same person, who was not aware of the protocol utilized.

At the end of the period of cold-restraint, the animals were removed from restraint and killed by rapid decapitation. The stomachs were removed after clamping the cardiac end. The fundi were then punctured, and the gastric contents were collected in a centrifuge tube. After centrifugation at 3,500 rpm for 15 min, gastric acidity was determined by automatic titration of gastric juice against 0.01N NaOH to pH 7.0; the titrated acid was expressed as mEq/L.

The stomachs were opened along the greater curvature, and the severity of the gastric erosions was evaluated using an illuminated magnifier (3×). Lesions greater than 1 mm in diameter were counted. The specimens were placed in formalin solution for fixation. The presence of gastric erosions was confirmed on histologic examination. In each group, the bleeding areas and ulcers were counted and measured by an independent investigator who was not aware of the protocol utilized. After the number and sizes of the ulcers were noted, an arbitrary ulcer index was calculated based on the sum of the length of each lesion per stomach. The severity of ulcer and hemorrhage was recorded[3] as +, ++, +++.

Statistical evaluation of the data was performed by means of Fisher's exact test or Student's t test. Bonferroni's inequality was employed to adjust for multiple comparisons. Differences between groups were considered to be statistically significant if $P < .05/3 = 0.0167$.

RESULTS

Table I gives the results of the effect of CPZ on the incidence of gastric stress ulcers. The ulcer index, severity of gastric ulcers, incidence of hemorrhage, and gastric acidity are also provided.

No rats died as a result of the cold-restraint stress. Acute gastric ulcers occurred in 100% of the animals in the saline control group, with a mean ulcer index of 21.1 ± 1.6. In the CPZ-pretreated groups both the incidence and the severity of the gastric erosions were significantly lower than those in the saline control group.

Rats in the saline control group showed physical signs of stress, manifested by irritability, struggling, hyperventilation, periodic chattering, and increased involuntary urination and defecation. These signs were considerably reduced in the low-dose CPZ group and were

---

Address correspondence to Dr Canver, Clinical Assistant Instructor, Department of Surgery, SUNY-Buffalo, The Buffalo General Hospital, 100 High St, Buffalo, NY 14203.

This research was sponsored by Kayseri University, Turkey. Financial support was provided by Tubitak, a government-owned research institute in Turkey.

TABLE I. Effect of Chlorpromazine (CPZ) on Gastric Ulcer Formation and Acidity.

Group	Incidence (No. of rats with ulcer)	%	Ulcer Severity +++	++	+	−	Hemorrhage	Titratable Gastric Acidity (mEq/L)	Ulcer Index (mm)
Unstressed, untreated (1)	0/7	0.0	0	0	0	0	−	32.7 ± 1.48†	0.0
Stressed rats pretreated with saline solution (2)	7/7	100	7	0	0	0	+++	8.8 ± 0.43	21.1 ± 1.6
Stressed rats pretreated with CPZ (2.5 mg/100 g) (3)	5/7	71.4	0	2	3	2	+	15.0 ± 0.95*	10.8 ± 2.2*
Stressed rats pretreated with CPZ (5 mg/100 g) (4)	3/7	42.8	0	0	3	4	−	19.0 ± 1.55*	4.3 ± 1.6*
Stressed rats pretreated with CPZ (10 mg/100 g) (5)	2/7*	28.5	0	0	2	5	−	27.2 ± 2.36*	2.1 ± 0.8*

* Significantly different from saline control group.
† Values are means ± SE.

absent in the medium-dose and high-dose CPZ groups. At low doses, operant behavior was reduced, and responses to cold-restraint stress were fewer, slower, and of less intensity, with diminished spontaneous motor activity. At high doses, CPZ induced characteristic cataleptic immobility that allowed the animal to tolerate the abnormal posture that persisted during the stress experiment. The animals appeared to be indifferent to stress, although they continued to withdraw from stimuli that were noxious or stressful. Even in the high-dose group, no CPZ-induced comas were observed.

A typical stomach from the control rats had stress hemorrhages and multiple ulcerations of the stomach mucosa. The lesions occurred only in the fundic glandular portion of the stomach. A typical stomach from a stressed rat pretreated with medium-dose or high-dose CPZ had pink mucosa with no evidence of ulceration. On microscopic evaluation, the mucosal defects in the untreated, stressed group were characterized by acute necrosis or absence of superficial mucosal cells, with extension of the necrotic process into the glandular epithelium. Many mucosal and submucosal capillaries and venules were dilated and congested. Submucosal edema was noted in many of the sections in which erosive injury was extensive. The most significant finding in the rats treated with medium-dose and high-dose CPZ was the marked reduction in the number of (or absence of) mucosal erosions. Pathologic findings such as edema and vasocongestion were minimized in the rats that had been treated with medium-dose and high-dose CPZ.

The data for the gastric acidity in cold-restrained rats are displayed in Table I. Acute stress, induced by restraint and 4°C for 4 hours, significantly reduced the titratable acidity in the saline treated rats when compared with the untreated, unstressed control group. In experimental rat groups treated with low, medium, and high doses of CPZ, the titratable acidity increased during stress. The differences were statistically significant in comparison to the saline group.

DISCUSSION

These results demonstrate that, within this experimental design, CPZ significantly diminished the incidence of acute gastric stress ulcers and hemorrhage. The findings are in agreement with those reported by Malecki et al[4] and Hano et al.[5] The mechanism of mucosal injury in this model is not known; however, the bleeding lesions occurred in the glandular portion of the stomach and were confined to the mucosa and submucosa.

The physical signs of stress were reduced considerably in rats pretreated with moderate CPZ doses. This can probably be attributed to the fact that the rats pretreated with CPZ may be less vulnerable to the stress conditions. Presumably, the peripheral features of the drug are not powerful enough to afford protection against cold-restraint induced erosions. The lack of protective activity seen with sedatives and tranquillizers[6] is a strong challenge to the concept that the central effects of CPZ may be important in the development of stress erosions.

Acute gastric stress ulceration is an acid peptic dissolution of the gastric mucosa in which neither an increase in luminal acidity nor an enhanced secretion of pepsin has been identified as a causative factor. Recent studies of pathogenesis have thus been directed toward identification of defects in the protective mechanisms of the gastric mucosa against the acid that diffuses back into it from the gastric lumen. Nonetheless, the acid present in the gastric lumen plays a key role in pathogenesis of these lesions.[7] In contrast to previous reports by Hano[5] and Takeuchi,[8] CPZ increased the gastric acidity during cold-restraint stress in this study. There is evidence to suggest that the protective actions of CPZ are related to their effects on gastric secretion.

It is concluded that the prevention of gastric ulcer development by CPZ may be the result of its augmentation of stress tolerance, probably due to its central neurodepressant effect and its gastric secretory influence during severe stress. However, the results obtained in this study point to the possibility that CPZ could exert its ulcer-protecting effects through other unexplained mechanisms.

This study demonstrates that CPZ was effective in preventing gastric mucosal ulcers and bleeding in rats treated with cold-restraint stress. It was more effective in higher doses without fatal complications. Further study is needed to evaluate the effects of CPZ in the prevention of gastric stress ulcers and to determine its mechanisms of action.

REFERENCES

1. Brodie DA, Hanson HM: A study of the factors involved in the production of gastric ulcers by the restraint technique. *Gastroenterology* 1960; 38:353-360.
2. Brodie DA, Valitski LS: Production of gastric hemorrhage in rats by multiple stresses. *Proc Soc Exp Biol Med* 1963; 113:998-1001.
3. Watanabe K: Some pharmacological factors involved in formation and prevention of stress ulcers in rats. *Chem Pharm Bull* 1966; 14:101-107.
4. Malecki I, Borkowska B, Węgrzyn I: Effect of psychotropic drugs on the development of experimental gastric ulcer produced by different stress-inducing factors in rats. *Acta Physiol Pol* 1979; 30:649-656.
5. Hano J, Bugajski J, Danek L, Wantuch C: The effect of neuroleptics on the development of gastric ulcers in rats exposed to restraint-cold stress. *Pol J Pharmacol Pharm* 1976; 28:37-47.
6. Beattie D: Effect of drugs on rats exposed to cold-restraint stress. *J Pharm Pharmacol* 1977; 29:748-751.
7. Silen W: The prevention and management of stress ulcers. *Hosp Pract* 1980; 15:93-100.
8. Takeuchi K, Okabe S, Tagaki K: A new model of stress ulcer in the rat with pylorus ligation and its pathogenesis. *Am J Dig Dis* 1976; 21:782-788.

REVIEW ARTICLE

The negligible role of ethylene dibromide in overall human cancer risk

JOHN H. WEISBURGER, PhD, MD

With increasing frequency, the mass media report the discovery of newly identified carcinogens. Are carcinogens unavoidable? Can actions be taken to curtail the staggering number of deaths from cancer—approximately 400,000 each year?

Many news reports would make it seem as though it is a simple matter to identify and remove cancer-causing agents from the environment. Indeed, successes along these lines have been achieved. When increased rates of bladder cancer were found in workmen engaged in the manufacture of dye-stuff intermediates, specifically 2-naphthylamine or 4-aminobiphenyl, the production of these chemicals was stopped in many countries. As a result, this occupationally induced disease is disappearing. Overall, relatively few people, perhaps no more than a few hundred, were affected.

Do chemical carcinogens play a role in human cancer in the general public? The answer most likely is yes. If so, what are these agents? Where can they be found and what can be done about them? What are the quantitative aspects, and how important is the amount of human exposure? What is known about their mode of action?

DETECTION METHODS

Progress has been made in the last 20 years in cancer research, especially in understanding chemical carcinogenesis and the epidemiology of cancer. Advances have also occurred in the field of analytical chemistry, with the development of techniques that can reliably detect and measure chemicals with a sensitivity of parts per billion and, in some instances, parts per trillion. This is the reason for the identification of "carcinogens" in food, water, and air. Thus the public is told that there are nitrosamines in beer or in bacon, or ethylene dibromide (EDB) in cereals or citrus fruits. Twenty years ago, there were no accurate means to measure these levels, especially in complex products such as foods. Therefore, the question becomes one of estimating the biological significance and the possibility of adverse effects of these trace amounts of carcinogens, even potent ones.

From studies of epidemiology and geographic pathology, researchers have learned that different cancers have distinct causative, enhancing, or protective elements.[1-3] Thus, cancer of the lung has one set of causative elements, separate from those applicable to cancer of the stomach, and distinct again from cancer of the colon. It is likely that in the same organ, the lung, bronchogenic carcinoma has a different cause—cigarette smoking—than oat cell carcinoma, which is often seen in nonsmoking individuals exposed to certain alkylating chemicals. Similarly, it is possible that cancer of the ascending colon, the descending colon, and the rectum have different etiologies.

CARCINOGENIC PROCESS

Few chemicals have a nonspecific carcinogenic effect. Most chemical carcinogens have specific targets, and studies on the mode of action of such chemicals attribute this organ specificity to the host-mediated biochemical metabolism of these chemicals, which depends on the chemical structure of the agents. A carcinogen that can induce cancer of the stomach usually does not also yield cancer of the colon or bladder, and vice versa. When different agents affect the same target organ, their action can be quantitatively additive; however, when they act on distinct organs, their effects are usually independent.

The carcinogenic process is complex. The conversion of normal cells to invasive metastatic cancer is a long, sequential process. Dissection of this process into specific components has yielded insight into the relevant mechanisms and has given hope that the inhibition of one or more of these steps might delay the occurrence of widespread disease and thus lower the risk of a fatal outcome.

TYPES OF CARCINOGENS

When metabolized, most carcinogens form reactive alkylating products that interact with cellular macromolecules, including DNA.[4] Such genotoxic carcinogens can be detected through their mutagenic effects.[5] Their activity under various experimental or environmental conditions depends on the effectiveness of the activation reaction, with the consequent modification of genetic material. The sensitivity of a person or a species of animals to a carcinogen is genetically controlled. Repair enzymes can restore the integrity of the normal genome following carcinogen-induced damage. The ultimate expression of a transformed genome is determined by subtle gene rearrangements caused by the genotoxic carcinogen.

The conditions that control the growth and development of cells with an abnormal genome are also important in the uncontrolled proliferation of neoplastic tissue. The growth process is enhanced by nongenotoxic epigenetic modifiers[6] that may take the form of synthetic chemicals and drugs as well as naturally-occurring compounds in one's diet. This aspect of the carcinogenic process is highly

---

Address correspondence to Dr Weisburger, Vice President for Research, American Health Foundation, Dana Rd, Valhalla, NY 10595-1599.

dose dependent and, up to a point, reversible, enabling investigators to control the effects of carcinogens by altering dosage or by removing agents with growth-promoting properties.

Little is known about the progression of a neoplasm from the differentiated to the undifferentiated state, the factors that might control the de novo appearance of an undifferentiated neoplasm, or the controlling elements in metastasis.

THE EPIDEMIOLOGY OF CANCER

During the past 30 years, researchers have acquired a better understanding of the main causes of human cancers. The American Cancer Society projects that in 1985 there will be 910,000 new cases of cancer in the United States and 462,000 deaths resulting from cancer. Twenty-two percent will involve cancer of the respiratory tract, pancreas, kidney, and bladder, attributable mainly to cigarette smoking. An additional 4% are cancers of the oral cavity and esophagus, associated with smoking, excessive drinking of alcohol (7 shots of whiskey or equivalent other alcoholic beverage per day), or chewing tobacco. This incidence of morbidity and mortality would be considered a major disaster if induced by an industrial chemical such as a dye or pesticide, rather than by tobacco smoking.

A consideration of the major causes of mortality—heart disease, and colon and breast cancers—and those diseases with a lower incidence—cancer of the ovary and endometrium—indicates that Western-style nutrition, particularly the level of dietary fat, is an important factor.[7,8] In Japan, an industrialized nation, there is much lower mortality from these diseases compared to America or other Western societies, perhaps due to the Japanese diet, which is low in fat. Interestingly, prostatic cancer exhibits a similar incidence in Japan as compared to the US, when considered as an in situ disease. However, in the US, prostatic cancer presents as an invasive, metastasizing, and hence fatal disease.

These nutritional differences have been reproduced under highly controlled conditions in the laboratory in animal models, for example, for colon or mammary cancer.[1,6,8] A considerably higher incidence of mammary gland or colon cancer is seen in animals fed diets containing 40% of calories as total fat—mimicking the American nutritional custom, than with diets of 10% fat content—representing the traditional Japanese intake. This is true whether the fat has an animal or plant origin, thus ruling out minor contaminants, such as pesticides, as representing the enhancing elements. In the case of colon cancer, the mechanism whereby fat translates to a higher disease risk has been partially elucidated. A higher fat level leads to increased endogenous cholesterol biosynthesis, a mechanism that may account for the risk of atherosclerosis and heart disease. Cholesterol is further metabolized to bile acids, which have been demonstrated to act as promoting elements in large bowel cancer.

In Finland, despite a high total fat dietary intake, the risk of colon cancer is low.[9] This low risk has been traced to a customary intake of higher amounts of cereal fiber that increase stool bulk, thereby diluting intestinal contents and the concentration of bile acids. This situation has also been reproduced in animal models. The data show that in the United States and other Western countries, the average bile acid concentration is about 12 mg/kg of stool. In Japan, the bile acid concentration is only 4 mg/kg, and in Finland, the concentration is also about 4 mg/kg. A high risk situation converts to a low risk by reducing the bile acid concentration by a factor of three. Thus, it is clear that in this instance, and in other cases in which promotion plays a role in causing cancer, a relatively small decrease in the amount and concentration of the promoters results in major effects. In the instance of cigarette smoking, the risk for lung cancer is high in individuals who smoke 40 cigarettes per day, but much lower with 10 cigarettes per day; the effect cannot be measured reliably with four cigarettes per day.

QUANTITATIVE ASPECTS

Quantitative carcinogenesis has been the subject of few sound research efforts. This is perhaps due to public perception. Legislation, such as the Delaney Clause, has called for the total removal of any agents with carcinogenic properties. Historically, this approach served to call attention to the need to consider carcinogenesis as a special case of pharmacology and insisted that human contact be minimized or eliminated as the best approach to cancer prevention. This is the method of choice whenever it can be readily implemented, as in occupational health or smoking. One cannot stop eating, however. Hence, for nutritional effects, other considerations must apply.

The time has come to examine whether the traditional method of cancer prevention does not require a new look for the 1980s. Genotoxic carcinogens should undergo different risk assessment than agents that act as promoters. There are laboratory procedures to classify and delineate agents that are genotoxic, in contrast to those that are not.[10] By such standards, it has been established that food additives such as saccharin and cyclamate, antioxidants such as butylated hydroxyanisole (BHA) or butylated hydroxytoluene (BHT), pesticides and insecticides such as DDT or chlordane, and drugs such as phenobarbital are all promoters. They are uniformly negative in tests for genotoxicity, but positive in assays currently being developed for promoters. Yet, in the traditional, classic sense, they have been called carcinogens because under conditions that usually involve very high level continuous dosing, in lifetime and sometimes in two generation studies, they have induced cancer. Since promoters do not affect the genetic material, but seem to act as growth-control elements, the action of promoters should be highly dose-dependent and reversible[6]; test results have supported this concept.

Human evidence also corroborates these concepts. For example, when conjugated estrogen therapy was discontinued in postmenopausal women, the associated higher incidence of endometrial carcinoma promptly declined—excellent evidence of reversibility.[11]

For genotoxic carcinogens, a paucity of experimental data exists for risk of cancer at the parts per billion (ppb) or even the low parts per million (ppm) dose ranges that chemists can now measure accurately. Thus, there has been a need to extrapolate from the high dose levels customarily used in carcinogen bioassays. In the absence of an accurate dose-response curve, this has given rise to controversial opinions, without the backing of facts.

Of four carcinogens, all affecting the liver in experimental animals, aflatoxin has induced cancer reliably in rats at the 1-50 ppb level in the diet; diethylnitrosamine has done so at the 1-40 ppm level; safrole at the 1,000-5,000 ppm level; and acetamide at the 10,000-50,000 ppm level. Thus, even without dose-response data for each chemical, it is evident that aflatoxin would be much more dangerous than acetamide, and the other two would assume risks of intermediary proportion. Yet, synthetic safrole, a food flavoring, has been banned even though certain plant extracts contain naturally-occurring safrole. On the other hand, for the mold toxin aflatoxin B_1—found on crops such as corn, grains, or peanuts—an official "action level" of 20 ppb, and for certain uses even 200 ppb, has been set. The defense of this regulatory action was that aflatoxin B_1 is an unavoidable, naturally-occurring contaminant. In certain African countries where agricultural practices are not as highly developed as in the United States, crops have been found to contain levels of aflatoxin B_1 100 to 500 fold higher than in the US. In the areas affected, primary liver cancer is a major disease (perhaps also because of the higher titers of hepatitis B antigen that potentiates the effect of the dietary aflatoxin).[3] In the US, where aflatoxin B_1 was isolated and its structure proven about 1960, primary liver cancer is a neoplasm with declining incidence. Thus, it would seem that the presence of this carcinogen in our diet was at a level such that in lifetime intakes the disease risk did not come to the fore. Although human exposure to known genotoxic carcinogens should be avoided, the aflatoxin data suggest that even lifetime exposure to trace amounts may not be a great risk to humans.

As noted, there are few reliable dose-response studies for powerful carcinogens in the ppb range. Unpublished data from a British study, for the liver carcinogens dimethylnitrosamine and diethylnitrosamine, have been interpreted to demonstrate a linear dose-response from the highest level tested (on the order of 16 ppm) to the lowest (32 ppb). However, this extrapolation of complex data requires further discussion, for within the limitations of the study and the number of animals used, an examination of the actual data suggests that levels from 32 ppb up to about 256 ppb yield an incidence of liver cancer within the range of the incidence seen in untreated controls.

For ethylene dibromide (EDB), no quantitative studies have been done. Stomach cancer, specifically squamous forestomach disease, was found with levels translated to several hundred ppm. Ethylene dibromide is a reactive, genotoxic carcinogen and causes cancer mainly at the point of entry: stomach upon oral intake, nasal cavity upon inhalation. It has been used in agriculture for about 30 years and presumably has led to contaminated grains during this entire period.

Virtually no action was taken to find replacements for this pesticide when the finding of its carcinogenicity and that of the related dibromochloropropane (DBCP) was published in 1973.[12] Use of DBCP was discontinued. Why was EDB not banned? Nonetheless, there is some reassurance that the levels prevailing in grains in 1984 will drop quickly as new grain crops without contaminant will dilute the older crops. Further solace can be found in the fact that the incidence of gastric cancer has been declining dramatically in the last 50 years.[1,9]

ROLE OF MEDIA IN CANCER PREVENTION

It seems puzzling, therefore, that the mass media have placed such emphasis on EDB contamination, thereby giving rise to public concern and alarm that is not justified. What is the public to think if the evening television news shows groceries being pulled from shelves because of claimed carcinogen-contaminated cereals or flour? Little mass media effort is made to discourage smoking, especially by young people. There is no media educational action to suggest that a lower intake of total fat would be beneficial to reduce the risk of premature fatal and disabling diseases such as atherosclerosis and coronary heart disease, or breast and colon cancer. There is little activity to suggest that white bread or baked goods in general with a low fiber content are not as beneficial as whole grain bread and whole grain bran-containing breakfast cereals in avoiding colon cancer and other intestinal diseases. There is a beginning effort by the Food and Drug Administration to label canned foods and products as to sodium content; however, the media do not often comment on salt use, particularly among some poorer people, in relation to the occurrence of hypertension. Other factors, such as stress, receive greater publicity in this regard, when, in fact, the scientific data base is stronger for the relationship of salt (especially sodium-potassium-calcium ratios) to high blood pressure, than for stress.

The literature contains thousands of papers and reviews substantiating the fact that lower fat and salt use, higher fiber intake, eating more fruits and vegetables, and control of smoking would have a major impact in lowering disease risk. The American Heart Association and the American Cancer Society have issued working documents in this important area of preventive medicine.

Do the media bring the facts to the public accurately? The "carcinogen of the week" syndrome—nitrite, dioxin, saccharin, DDT, and EDB—makes for exciting headlines. No wonder the public is resistant and confused and takes no action. Medical research has provided sound knowledge that is going stale, unless the media can be re-educated to better inform the public.

REFERENCES

1. Wynder EL, Leveille GA, Weisburger JH, Livingston GE: *Environmental Aspects of Cancer: The Role of Macro and Micro Components of Foods.* Westport, CT, Food and Nutrition Press, 1983.
2. Schottenfeld D, Fraumeni JF Jr (eds): *Cancer Epidemiology and Prevention: Current Concepts.* Philadelphia, WB Saunders, 1982.
3. Burchenal JH, Oettgen HF (eds): *Cancer: Achievements, Challenges and Prospects for the 1980's,* vols 1, 2. New York, Grune and Stratton, 1981.
4. Miller EC, Miller JA: Mechanisms of chemical carcinogenesis. *Cancer* 1981; 47:1055-1064.
5. Hollaender A, De Serres FJ (eds): *Chemical Mutagens: Principles and Methods for Their Detection,* vols 1-7. New York, Plenum Press, 1971-1982.
6. Pereira MA (ed): International Symposium on Tumor Promotion. *Environ Health Perspect* 1983; 50:3-330.
7. Farinara E, Stamler J, Mancini M: Proceedings of the International Symposium on Epidemiology and Prevention of Atherosclerotic Disease. *Prev Med* 1983; 12:1-234.
8. Newell GR, Ellison NM (eds): *Nutrition and Cancer: Etiology and Treatment. Progress in Cancer Research and Therapy,* vol 17. New York, Raven Press, 1981.
9. Reddy BS, Cohen LA, McCoy GD, Hill P, Weisburger JH, Wynder EL: Nutrition and its relationship to cancer. *Adv Cancer Res* 1980; 32:237-345.
10. Williams GM, Dunkel VC, Ray VA (eds): Cellular systems for toxicity testing. *Ann NY Acad Sci* 1983; 407:1-482.
11. Austin DF, Roe KM: The decreasing incidence of endometrial cancer: public health implications. *Am J Public Health* 1982; 72:65-68.
12. Olson WA, Habermann RT, Weisburger EK, et al: Induction of stomach cancer in rats and mice by halogenated aliphatic fumigants. *J Natl Cancer Inst* 1973; 51:1993-1995.

A pelvic mass with periosteal reaction

CASE HISTORY

A 13-year-old girl was admitted to the Albert Einstein College Hospital for evaluation of right hip pain and a pelvic mass. She was in good health until three weeks prior to admission when she was first seen by her physician because of pain in her right hip and a limp. There was no history of trauma or fever. Physical examination at that time was unremarkable except for tenderness in the region of the right pelvis, especially near the right sacroiliac joint. A complete blood count was within normal limits, but the erythrocyte sedimentation rate was elevated to 90 mm/hr. Five days later she was again seen by her physician when the hip pain persisted. A repeat complete blood count was normal, and fluorescent antinuclear antibody and latex fixation tests were negative. The patient was treated with aspirin but returned still complaining of pain without relief. At the time of hospital admission there was a firm irregular non-tender palpable mass, just above the edge of the right iliac crest in the right lower pelvis. Multiple radiographic examinations including, initially, plain films of the abdomen and pelvis were performed.

RADIOLOGIC DISCUSSION

Joshua Gross, MD (Resident, Department of Radiology): The initial plain films of the pelvis showed periosteal reaction along the superior aspect of the right pubic ramus (Fig 1). There was no visible soft tissue mass density. The underlying bone appeared intact without definable lytic or blastic changes. A radionuclide bone scan (Fig 2) showed asymmetric increased activity in the area of the right superior pubic ramus and periosteal reaction was noted on the radiographs.

Computed tomography was performed before and after intravenous contrast enhancement. There was a large soft tissue mass in the right pelvis, extending from the level of the mid portion of the right iliac bone to the acetabulum (Fig 3). It displaced the bladder and sigmoid colon to the left. The mass was primarily solid, but contained one small apparently cystic or necrotic area. It showed general mild contrast enhancement. There was no calcification in the mass or definite evidence of bowel involvement.

A barium enema was also performed, which confirmed displacement of the sigmoid to the left, without bowel wall involvement. Angiography showed the presence of a large avascular mass (Fig 4). There was mild splaying of several branches of the right internal iliac artery including the right inferior gluteal artery, but no neoplastic vascularity was demonstrated.

The differential diagnosis includes inflammatory and neoplastic masses. This patient complained of hip pain and periosteal bone reaction of the right superior pubic ramus. Periosteal reaction can be stimulated by intrinsic bone

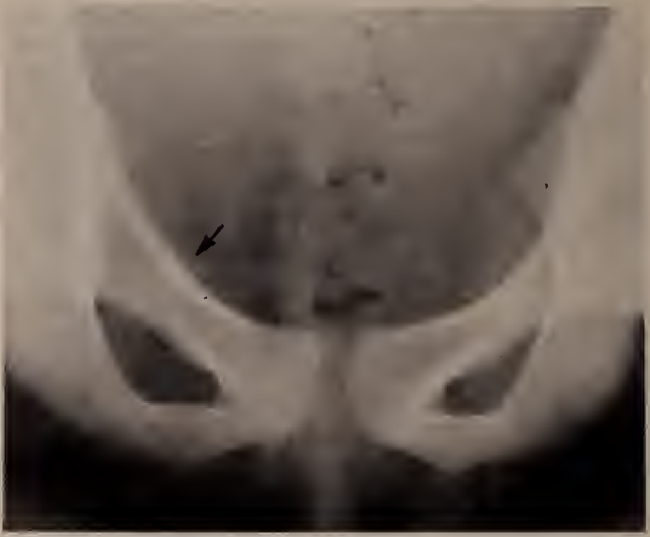


FIGURE 1. Periosteal reaction (arrow) at right superior pubic ramus.

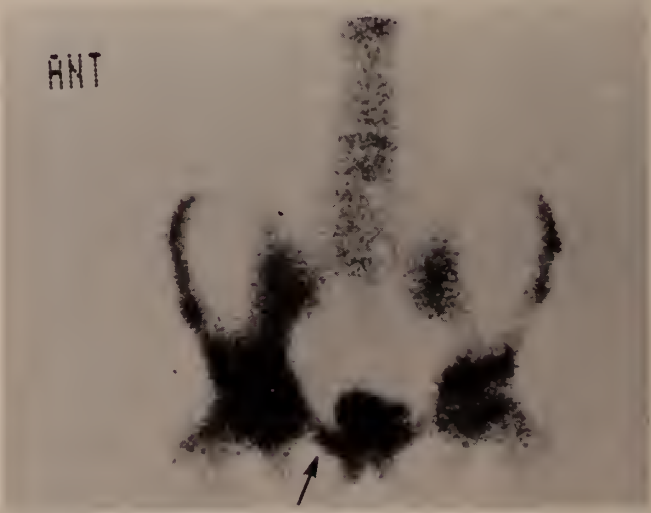


FIGURE 2. Radionucleotide scan showing increased activity of right hemipelvis and right superior pubic ramus (arrow).

Address correspondence to Dr Bennett, Director, Department of Anatomic Pathology, The Hospital of the Albert Einstein College of Medicine, 1825 Eastchester Rd, Bronx, NY 10461.

Inquiries regarding the submission of manuscripts to this section should be sent to John Flynn, MD, Consulting Editor, *New York State Journal of Medicine*, 420 Lakeville Rd, Lake Success, NY 11042.

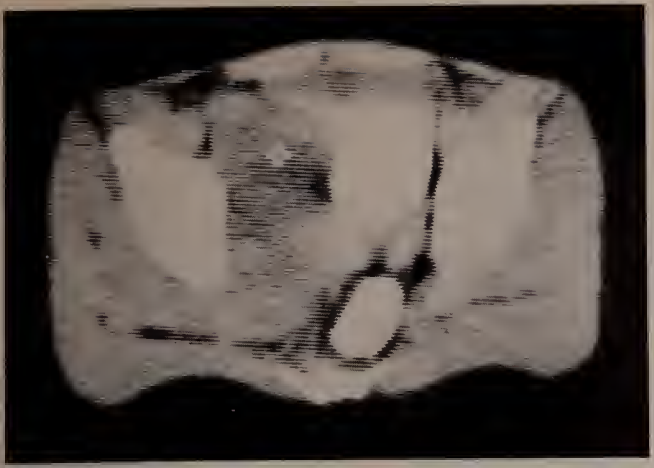


FIGURE 3. Computerized axial tomographic scan showing large right pelvic soft tissue mass (cursor) displacing opacified bladder and rectum toward the left.

disease—an infection or tumor—or by adjacent contiguous inflammatory neoplastic disease. Coupled with a positive isotopic bone scan, osteomyelitis or an associated pelvic abscess might be considered. However, lack of an elevated white blood cell count and fever suggests against this diagnosis. The pelvic mass also was not tender and did not have the typical characteristics of an abscess on computed tomography (CT). A neoplasm would therefore be the most likely consideration.

There was no history of trauma and no specific evidence of a fracture. If occult trauma was the cause of the patient's pain and periosteal reaction, the presence of the mass would have to be explained on the basis of hematoma formation. The results of a CT scan, ultrasonography, and laboratory tests were not consistent with the latter.

Rhabdomyosarcoma is the most common soft tissue sarcoma that occurs in infants and children. It is usually hypervascular, although exceptions do occur. Other types of soft tissue tumors such as lipomas, liposarcomas, fibrosarcomas, and parosteal fibrosarcomas are less common than rhabdomyosarcomas. Lipomas have the characteristic radiologic appearance of masses, with decreased attenuation on CT, and would not involve the adjacent bone. In cases of liposarcoma, fibrous tissue may be present that may give a mass with increased attenuation a radiologic appearance more similar to the soft tissue density. However, most liposarcomas occur in adults rather than children. They may be hypovascular or avascular on angiography.

Parosteal fibrosarcoma is an extra osseous sarcoma rising from an outer sheath of the periosteum or nearby fibrous tissues, and may contain calcifications. Some tumor vascularity is usually present.

CLINICAL DIAGNOSIS

Soft tissue sarcoma, most likely rhabdomyosarcoma.

PATHOLOGIC DISCUSSION

Boyce Bennett, MD (Professor, Department of Pathology): A laparotomy was performed, and the mass was found to be unresectable. Several biopsies of the mass were taken, and nearby lymph nodes were sampled. Much of the tumor was found to be necrotic, but areas of viable tissue showed the tumor to be highly cellular, containing uniform small cells and little supporting stroma (Fig 5). The tumor cells were found to be periodic acid-Schiff positive; the reaction was sensitive to diastase digestion, indicative of the presence of cytoplasmic glycogen. Material was also appropriately prepared for study by electron microscopy and immunohistochemical methods. The lymph nodes were not involved by tumor.

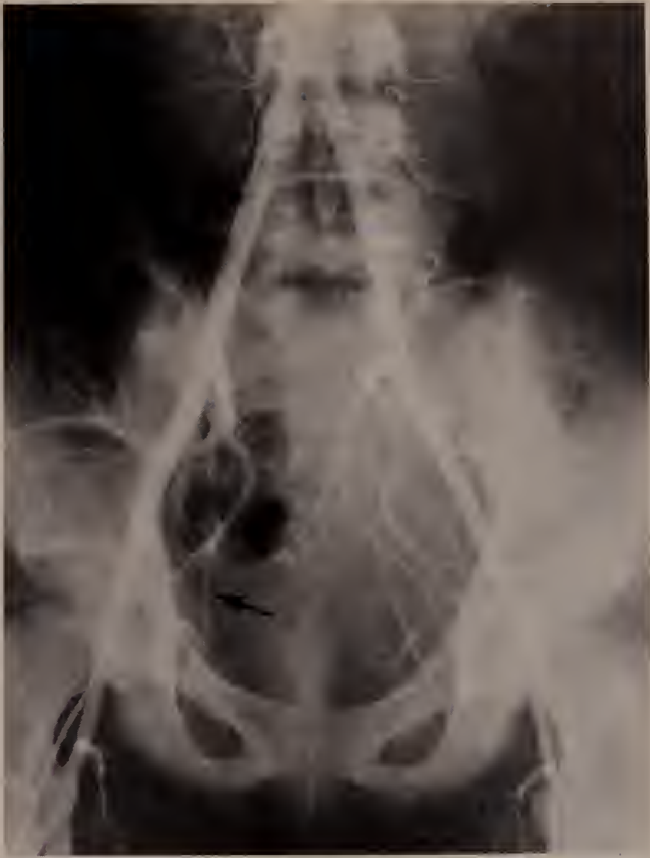


FIGURE 4. Angiography revealing deviation and stretching of right internal Iliac branches (including inferior gluted branch) by avascular pelvic mass (arrow).

Such small cell malignant tumors occurring in children present a difficult but common problem for the pathologist. The differential diagnosis rests among the following: lym-

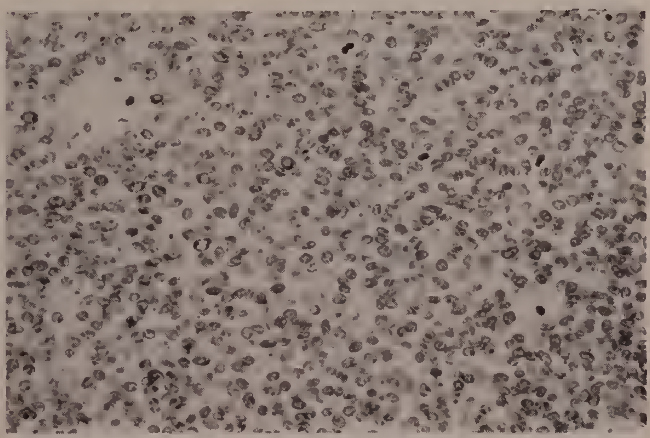


FIGURE 5. Photomicrograph of biopsy of tumor mass. The tumor consists of fairly uniform small cells with very little supporting stroma.

phoma or leukemia, Ewing's sarcoma, rhabdomyosarcoma, and neuroblastoma.[1] To distinguish among these possibilities usually requires electron microscopic and immunohistochemical study in addition to light microscopy, and the results of such studies are central in determining the treatment. The presence of glycogen indicated by the periodic acid-Schiff stain would indicate that the tumor was not likely to be lymphoma or leukemia.

The material processed for electron microscopic examination, however, proved to be necrotic tissue; additional material was therefore taken from the paraffin block and prepared for electron microscopy. Such treatment results in considerable artifact, but we were able to note that the neurosecretory granules that would be expected with a neuroblastoma were not present. Cytoplasmic fibrils were identified, but the banding characteristic of a rhabdomyosarcoma[2] was not seen. Immunohistochemical studies using an anti-myosin antibody on paraffin-imbedded, Bouin's fixed tissue were negative.

At the conclusion of these studies we still had no proof of the identity of the tumor. Based on its appearance under the light microscope, its location in the body, and the lack of neurosecretory granules on electron microscopy, we believed that the tumor was an embryonal rhabdomyosarcoma. The patient was therefore treated with radiotherapy to the pelvic mass, followed by chemotherapy. The pelvic mass disappeared, but the patient has evidence of recurrent disease one year after surgery.

FINAL DIAGNOSIS

Embryonal rhabdomyosarcoma.

DISCUSSION

Theodore R. Smith, MD (Associate Professor, Department of Radiology): Rhabdomyosarcoma is the most common soft tissue sarcoma of childhood and is said to constitute 4% to 8% of all malignancies in children under 15 years of age, and about 40% of childhood sarcomas.[3] The incidence in men and boys has been reported to be higher than in women and girls, about 58% to 42%, respectively. Rhabdomyosarcoma is highly malignant, with a tendency to involve local structures and eventually to metastasize. In the adult, the extremities and trunk are the most frequent sites of occurrence, while in children the pelvis is a common location. As seen in this case, approximately 20% of all childhood rhabdomyosarcomas arise from the pelvis, principally from the genito-urinary tract.[4] (The exact origin of this pelvic tumor could not be determined at surgery.) In the pelvis there is also a predilection for local lymph node metastasis.

Rhabdomyosarcomas may be classified under three histologic types: embryonal, alveolar, and pleomorphic.[5] The embryonal type is the most frequent in children, particularly those under age five, followed by the alveolar type. The pleomorphic form is the more common "adult" type. Alveolar rhabdomyosarcoma is more frequently seen in the extremities, mainly in the 10 to 15 year age group. Embryonal rhabdomyosarcoma occurs most commonly in the head and neck region, although a pelvic mass is common. Historically, patients with rhabdomyosarcoma have had an overall poor prognosis, with a five-year survival rate of 26% and a median survival of 10 months. More recently, with the advent of combined therapy including surgery, radiation, and multiple chemotherapeutic agents (vincristine, actinomycin D, and cyclophosphamide, for example) the prognosis has improved significantly. As a result of this combined approach, two-year survival rates with no relapse have been reported at 71% for patients with local or regional disease and 28% for those with distant metastasis on diagnosis.[4] Eighty percent of recurrences usually occur within the first two years following localized therapy.

Radiologic examination is used in detecting the existence and extent of a lesion and in determining the extent of follow-up required after therapy. In general, computed tomography (CT) is the best single method for defining the extent of a soft tissue tumor vis à vis adjacent anatomic structures such as organs, bone, blood vessels, and muscle planes.[6,7] Whereas CT can be used to delineate tumors, it is not usually specific for cell type or origin, with the exception of low attenuation lipomatous tumors. Intravenous contrast may enhance a rhabdomyosarcoma. Rarely, ossification has been reported in rhabdomyosarcoma[8] while cystic or necrotic areas of lower attenuation in a lesion may also be present.

Angiography is also useful for soft tissue tumors in helping to decide resectability, define the size of the tumor, and demonstrate unsuspected satellite lesions. Rhabdomyosarcomas generally show neovascularity, but in this case displacement of vessels was the chief manifestation. The tumor was relatively avascular, probably related to its partial cystic component.

This patient had arthritic symptoms and periosteal reaction. Arthritis may be present because a rhabdomyosarcoma frequently affects the growing skeleton, producing destructive bone lesions. However, as was apparently true in this case, the arthritic symptoms and periosteal reaction may be a response to a contiguous periarticular or periosseous tumor, rather than actual bone invasion.[9,10]

Demonstration of bone involvement with plain or tomographic roentgenography can often be as good as or more sensitive than CT in individual cases. Likewise, other conventional radiographic methods—barium enemas and intravenous urography—can also be helpful in detecting malignancy and illustrating the advantages of a multiformatted radiographic approach.

REFERENCES

1. Hajdu SI: *Pathology of Soft Tissue Tumors*. Philadelphia, Lea and Febiger, 1979, p 504.
2. Morales AR, Fine G, Horn Jr RC: Rhabdomyosarcoma: an ultrastructural appraisal. *Pathol Annu* 1972; 7:81-106.
3. Neifeld JP, Maurer HM, Godwin D, Berg JW, Salzberg AM: Prognostic variables in pediatric rhabdomyosarcoma before and after multi-modal therapy. *J Pediatr Surg* 1979; 14:699-703.
4. Fleischmann J, Perinetti EP, Catalona WJ: Embryonal rhabdomyosarcoma of the genitourinary organs. *J Urol* 1981; 126:389-391.
5. Danziger J, Handel SF, Jing BS, Wallace S: Computerized tomography in rhabdomyosarcoma of the head and neck. *Cancer* 1979; 44:463-467.
6. Egund N, Ekelund L, Sako M, Persson B: CT of soft-tissue tumors. *AJR* 1981; 137:725-729.
7. Levine E, Lee KR, Neff JR, Maklad NF, Robinson RG, Preston DF: Comparison of computed tomography and other imaging modalities in the evaluation of musculoskeletal tumors. *Radiology* 1979; 131:431-437.
8. Brasch RC, Kim OH, Kushner JH, Rosenau W: Ossification in a soft tissue embryonal rhabdomyosarcoma. *Pediatr Radiol* 1981; 11:99-101.
9. Weinblatt ME, Karp RI: Monoarticular arthritis: Early manifestation of a rhabdomyosarcoma. *J Rheumatol* 1981; 8:685-688.
10. Grosfeld JL, Smith JP, Clatworthy Jr HW: Pelvic rhabdomyosarcoma in infants and children. *J Urol* 1972; 107:673-675.

Hodgkin's disease in half-siblings

RICHARD J. HIRSCHMAN, MD, MYRON P. NOBLER, MD

Numerous reports of Hodgkin's disease occurring in family members have been published.[1-3] The most common family relationship is sibling pairs, and the second most common relationship is between parents and children. Several cases of Hodgkin's disease have been reported in married couples.[4] We report a case of Hodgkin's disease occurring in a half-brother and half-sister.

CASE REPORTS

Case 1. A 25-year-old woman, born in Russia, noted axillary and supraclavicular adenopathy at the age of 23 years. A biopsy was done, and the diagnosis of malignant lymphoma was made. She was treated in Russia, with a combination of chemotherapeutic drugs and radiation therapy using local treatment, small fields, and low doses. The adenopathy disappeared for approximately one and a half years, but shortly before immigrating to the United States, she noted the reappearance of a mass on the right upper anterior chest wall.

She was first seen by us in August 1977, shortly after she arrived in this country. At that time she was asymptomatic, but a nontender mass approximately 8 × 6 cm in size was found in the right upper anterior chest wall and infraclavicular region. The remainder of the physical examination was noncontributory. Nodular sclerosing Hodgkin's disease was diagnosed on the basis of a biopsy of the mass. The patient then underwent bone, gallium, and liver scans, lymphangiography, and a laparotomy and splenectomy. No other evidence of Hodgkin's disease was found. It was therefore believed that the patient had stage IIAE Hodgkin's disease with extranodal spread from the lymph nodes in the subclavicular area to the right upper anterior chest wall. She was treated with primary irradiation: a total tumor dose of 4,000 rad to mantle portals in a period of five weeks, followed by a booster dose of 600 rad to the residual subclavicular mass, which disappeared following completion of treatment. At a later time she received a course of radiation therapy to the para-aortic lymph nodes and splenic pedicle, to a total tumor dose of 4,000 rad. She has been followed since that time and has shown no evidence of recurrent Hodgkin's disease during the past six years.

Case 2. This 42-year-old man is the half-brother of the previous patient. They have the same mother but different fathers. He noted a right supraclavicular mass approximately nine years after the diagnosis was established in his sister. He had no systemic symptoms. An enlarged group of right supraclavicular lymph nodes were noted on physical examination. A chest film disclosed a mediastinal mass occupying approximately 75% of the chest, as well as bilateral hilar adenopathy. A gallium scan revealed increased uptake in the right neck, mediastinum, and both hilar regions. Computed tomographic scans of the chest, abdomen, and pelvis confirmed these findings. No other evidence of lymphadenopathy, hepatosplenomegaly, or other suspicious areas of active Hodgkin's disease were noted. The diagnosis was that of Hodgkin's disease, stage IIA. However, because of the huge mediastinal mass, chemotherapy was begun and the patient received two courses of treatment with mechlorethamine, vincristine, procarbazine, and prednisone, which resulted in a dramatic shrinkage of the mediastinal mass. He then received radiation therapy via opposing anterior and posterior mantle portals, to a total tumor dose of 4,400 rad, followed by a boost of 600 rad to the residual mediastinal adenopathy, in a period of approximately six weeks. He is now receiving an additional two cycles of chemotherapy, which will be followed by irradiation of the para-aortic lymph nodes and spleen, followed by two additional cycles of chemotherapy. His condition is stable and satisfactory at this time.

DISCUSSION

An estimated 7-fold increased risk for Hodgkin's disease has been reported in the siblings of young adults under the age of 45 years with this condition.[2] Moreover, siblings of the same sex as an affected person have double the risk of those siblings of the opposite sex. This suggests that if Hodgkin's disease is spread by an infectious agent, intense contact—as occurs between siblings of the same sex that grow up in the same household—may enhance the risk.

An additional finding in the literature, however, is that spouses of persons with Hodgkin's disease do not appear to be at increased risk for the disease. A possible explanation would be if transmission of the "infectious agent" occurs only at an early age. This would account for the increased risk in young adults. Another explanation would be that of a genetic "weakness," which certainly might be present in siblings but would not be likely to be present in spouses. It is of interest that these siblings are of opposite sexes, and that they share only one parent.

REFERENCES

1. Kaplan H: *Hodgkin's Disease*, 2nd ed. Cambridge, Harvard University Press, 1980, pp 34-37.

2. Gufferman S, Cole P, Smith PG, Lukes RJ: Hodgkin's disease in siblings. *New Engl J Med* 1977; 296:248-250.

3. Vianna NJ, Davies JNP, Polan AK, Wolfgang P: Familial Hodgkin's disease: an environmental and genetic disorder. *Lancet* 1974; 2:854-857.

4. Berliner A, Distenfeld A: Hodgkin's disease in a married couple. *JAMA* 1972; 221:703-704.

See also pp 91-92.

From the Departments of Medicine (Dr Hirschman), Cabrini Medical Center and Beth Israel Medical Center, and the Department of Radiation Therapy (Dr Nobler), Beth Israel Medical Center, New York.

Address correspondence to Dr Hirschman, Chief, Division of Medical Oncology, Cabrini Medical Center, 227 East 19th Street, New York, NY 10003.

Adult-onset Still's disease and inflammatory orbital pseudotumor

John J. Cush, md, Ian H. Leibowitz, md, Sandor A. Friedman, md

In 1971 Bywaters[1] reported on 14 patients with adult-onset Still's disease. Since that time, the disorder has been recognized with increasing frequency as a systemic illness characterized by fever, rash, and seronegative arthritis. We describe a young man with typical adult-onset Still's disease in whom transient ptosis and extraocular muscle weakness developed, attributed to an inflammatory orbital pseudotumor.

Case Report

In March 1983 a 21-year-old man sought treatment for fever, chills, sweats, sore throat, myalgia, and anorexia of two weeks duration. His physician had prescribed ampicillin, but because of dyspnea, nonproductive cough, and pleuritic chest pain, he came to Coney Island Hospital. He denied rash, joint pain, significant weight loss, or Raynaud's phenomenon.

Physical examination revealed an acutely ill and anxious young man in mild distress. He had tachypnea and tachycardia, and his temperature ranged from 102.4°F to 104°F. He had normal pupillary responses, intact extraocular movements, and no ptosis. There was pharyngeal and tonsillar inflammation, lymphadenopathy, and an evanescent maculopapular, non-pruritic rash over the back, arms, and knees. Splinting of respiration, decreased breath sounds, dullness, and egophony at both bases were noted on chest examination. The cardiovascular examination was normal. The spleen was not palpable, the liver span was 12 cm, and the neurologic examination was unremarkable.

Noteworthy laboratory data included white blood cell count, 62,000/mm^3 (49% polymorphonuclear leukocytes, 28% bands, 20% metamyelocytes, 2% myelocytes, and 1% lymphocytes); Westergren method for erythrocyte sedimentation rate, 48 mm/hr; creatine phosphokinase, 404 U/L (normal, 0 to 225 U/L); lactic dehydrogenase, 860 U/L (normal, 60 to 200 U/L); alkaline phosphatase, 267 U/L (normal, 30 to 115 U/L); SGOT, 64 U/L (normal, 0 to 41 U/L); SGPT, 75 U/L (normal, 0 to 45 U/L). Blood urea nitrogen, serum creatinine, and urinalysis studies were within normal limits. Tests for antinuclear antibodies and rheumatoid factor were negative. Arterial blood gas values were pH, 7.50; P_{CO_2}, 32 mm Hg; HCO_3, 25 mm Hg; P_{O_2}, 68 mm Hg; saturated O_2, 98%. An electrocardiogram showed sinus tachycardia at 140/m, intermediate axis, and slight ST wave elevation at V_{2-4}. Mild cardiomegaly with bilateral basilar interstitial and alveolar infiltrates and minimal bilateral pleural effusions were noted on a chest roentgenogram. A moderate pericardial effusion was demonstrated by echocardiogram. Lumbar puncture, thyroid function tests, and bone marrow aspiration were unremarkable.

Antistreptolysin O, rubella, cytomegalovirus, coxsackie, herpes, chlamydia, toxoplasma, brucella and legionella titers were within normal limits. Tests for mononucleosis and hepatitis B surface antigen, and cold agglutinin tests were negative. Thoracentesis was done on three occasions and revealed blood-tinged, exudative pleural fluid. Subsequent cultures for bacteria, mycobacteria, and fungi were negative. A transbronchial biopsy provided evidence of an acute pneumonitis without fibrosis. Throughout the patient's hospital stay, he exhibited a quotidian fever pattern and an evanescent rash that was temporally related to his febrile episodes. His course was not affected by extensive antibiotic therapy.

One week after admission the patient complained of difficulty in opening the right eye, with diplopia and orbital pain on right lateral gaze. He exhibited ptosis, periorbital edema, and slight inferior displacement of the right eye. Visual fields were normal, but abduction and elevation of the right globe were impaired. Neither proptosis nor anisocoria was noted. Funduscopy, performed by an ophthalmologic consultant, revealed blurring of the nasal disc margin and a lack of spontaneous venous pulsations on the right side. The remainder of the ophthalmologic and neurologic examination was normal. On the same day, a brain scan, using diethylenetriamine penta-acetic acid tagged with technetium-99m, showed increased uptake in the periphery of the right orbit. A computed tomographic (CT) scan, with limited orbital views, revealed normal paranasal sinuses and intracranial contents. Two days later ptosis was noted in the opposite eye, associated with a fever of 105°F and a confluent macular rash.

He continued to have high spiking fevers, evanescent rash, leukocytosis, thrombocytosis, elevated sedimentation rate, and nonhemolytic anemia. He developed hepatomegaly, arthralgia, and arthritis of the left wrist, elbows, shoulders, hips, and knees. He continued to have pneumonic infiltrates and pleuropericardial effusions. Two weeks after admission he was placed on high-dose salicylate therapy without substantial improvement. One week later indomethacin was added to the treatment regimen, with a prompt response manifested by a fall in temperature and resolution of the rash, synovitis, and polyserositis. Because of cardiac involvement (ejection fraction below 40%), the patient was discharged on prednisone (30 mg twice daily). The neuro-ophthalmologic and systemic symptoms did not recur with the institution of anti-inflammatory therapy.

Five months later the patient was asymptomatic and was taking 25 mg of prednisone per day; weaning off prednisone slowly continued. After 12 months of therapy and while on 5 mg/day of prednisone, he noted the return of daily fevers and polyarthritis of the hips, knees, ankle, and wrist. The dose of prednisone was increased to 20 mg/day, and a trial of intramuscular gold was attempted but discontinued because of an allergic febrile reaction. Alternatively, he was placed on hydroxychloroquine (400 mg daily) and indomethacin (200 mg daily) to control his fever and polyarthritis. The patient has been followed for a total of 19 months and is still troubled by persistent polyarthritis involving the hips, right ankle and shoulder, left elbow and wrist, and the metacarpophalangeal and proximal interphalangeal joints bilaterally. There has been no recurrence of neuro-ophthalmologic symptoms or myocarditis. He is currently receiving physiotherapy, prednisone (15 mg daily) and indomethacin (100 mg daily).

Discussion

This patient had a multisystemic illness characterized by high quotidian fevers, transient rash, sore throat, lymphadenopathy, hepatomegaly, polyserositis, seronegative arthritis, leukocytosis, and elevated hepat enzyme levels. Extensive investigations failed to disclose a specific infectious, neoplastic, or rheumatic cause. His course was

---

From the Department of Medicine, Coney Island Hospital, Brooklyn, NY.

Address correspondence to Dr Friedman, Chief, Department of Medicine, Coney Island Hospital, 2601 Ocean Parkway, Brooklyn, NY 11235.

consistent with adult-onset Still's disease, previously characterized by Bywaters,[1] Medsger and Christy,[2] and Esdaile et al.[3] This diagnosis was supported by his subsequent clinical course and response to anti-inflammatory agents.

Adult-onset Still's disease remains a clinical diagnosis of exclusion and is frequently confused with rheumatic fever, lupus erythematosus, lymphoma, tuberculosis, and varied viral syndromes (infectious mononucleosis, hepatitis B, and rubella). It is easily differentiated from the viral syndromes by virtue of its high fevers, characteristic rash, chronicity, and negative serologic values. A confident diagnosis can be made with adequate knowledge of these entities, appropriate laboratory investigations, and observation of the patient.

The occurrence of ptosis and an extra-ocular motility disorder has not been reported in patients with adult-onset Still's disease. A normal CT scan and lumbar puncture excluded a specific central nervous system or para-orbital process, and normal thyroid function made Grave's ophthalmopathy unlikely. These exclusions and the presence of peri-orbital edema, movement-induced eye pain, and brain scan demonstration of peripheral orbital uptake suggest that an orbital disorder underlies these clinical findings. We believe that these features were secondary to an acute inflammatory orbital pseudotumor.

Inflammatory orbital pseudotumor[4–6] is a heterogeneous clinical entity of unknown etiology and pathogenesis that can mimic orbital neoplasms and thyroid ophthalmopathy. It is a reactive process that may appear as an isolated, localized lesion (infections, foreign bodies, granulomas, dermoid cysts) or as a manifestation of systemic disease (autoimmune disease, midline granuloma, sarcoidosis, histiocytosis, lymphoma). Inflammatory orbital pseudotumor is rarely the initial or sole manifestation of a systemic disorder. Instead, it is likely to occur during the course of a systemic illness. A clinical or pathologic diagnosis of a pseudotumor, especially if bilateral, warrants consideration of an underlying systemic or autoimmune disorder. Among systemic immune-mediated disorders, inflammatory orbital pseudotumor has been reported in Wegener's granulomatosis, polyarteritis nodosa, dermatomyositis, Sjogren's syndrome, systemic lupus erythematosus,[7] Crohn's disease,[8] myasthenia gravis,[9] and autoimmune thyroid disease. Clinical findings are attributable to retro-ocular disease characterized by mononuclear inflammatory infiltration of adjacent connective tissues, nerves, and muscles. Some pseudotumors selectively involve the orbital muscles, suggesting that orbital myositis should be considered a subset or feature of inflammatory orbital pseudotumor.[5,10,11] Orbital abnormalities can be unilateral or bilateral and may last from days to months.[6,12,14] Patients with inflammatory orbital pseudotumor usually have orbital pain and diplopia, with varying degrees of proptosis, ptosis, periorbital edema, chemosis, papilledema, and ophthalmoplegia. In general, treatment is directed at the underlying disorder when known; otherwise, a prompt therapeutic response to corticosteroids is said to be characteristic of a pseudotumor.

During the peak of systemic adult-onset Still's disease activity, this patient manifested transient ptosis, ophthalmoplegia and painful extraocular movements, features consistent with an acute inflammatory orbital pseudotumor of the myositis type. During this same period, a systemic myositis was evident by myalgia, and increased creatine phosphokinase and lactate dehydrogenase values. While muscular involvement has been described in this disease,[13] orbital myositis has not been reported. The similarity of the mononuclear inflammatory infiltration found in biopsies of orbital pseudotumors[4] and peripheral muscles in patients with adult-onset Stills' disease[13] lends further support to an association between the two disorders. Other reports of limited orbital inflammation and Still's disease have appeared in the literature. Ganday et al[14] described a 29-year-old woman with adult-onset Still's disease in whom transient diplopia and a third cranial nerve palsy developed, and Kemp et al[15] reported on a 9-year-old patient with systemic juvenile rheumatoid arthritis and similar findings due to a superior oblique tenosynovitis that was responsive to steroid therapy. In this patient the lack of ongoing orbital inflammation is temporally related to effective therapy with anti-inflammatory agents.

The systemic inflammatory nature of adult-onset Still's disease and systemic juvenile rheumatoid arthritis has long been recognized. It is, therefore, not surprising that the clinical findings in this case and others introduce the orbit as a potential inflammatory target for these entities. Orbital inflammation appears to be an acute, limited process that is responsive to treatment with corticosteroids.

REFERENCES

1. Bywaters EGL: Still's disease in the adult. *Ann Rheum Dis* 1971; 30:121–133.
2. Medsger Jr TA, Christy WC: Carpal arthritis with ankylosis in late onset Still's disease. *Arthritis Rheum* 1976; 19:232–242.
3. Esdaile JM, Tannenbaum H, Hawkins D: Adult Still's disease. *Am J Med* 1980; 68:825–830.
4. Garner A: Pathology of "pseudotumors" of the orbit: a review. *J Clin Pathol* 1973; 26:639–648.
5. Blodi FC, Gass JDM: Inflammatory pseudotumor of the orbit. *Trans Amer Acad Ophthal Otolaryng* 1967; 71:303–323.
6. Walsh FB, Hoyt WF: *Clinical Neuro-ophthalmology*, 3rd ed. Baltimore, Williams and Wilkins, 1969, pp 2014–2018.
7. Grimson BS, Simons KB: Orbital inflammation, myositis, and systemic lupus erythematosus. *Arch Ophthalmol* 1983; 101:736–738.
8. Ludwig I, Tomsak RL: Acute recurrent orbital myositis. *J Clin Neuro Ophthalmol* 1983; 3:41–47.
9. Van de Mosselaer G, Deuren H, Dewolf-Peeters C, Missotten L: Pseudotumor orbitae or myasthenia gravis: a case report. *Arch Ophthalmol* 1980; 98:1621–1622.
10. Nugent RA, Rootman J, Robertson WS, et al: Acute orbital pseudotumors: classification and CT scan features. *AJR* 1980; 137:957–962.
11. Slavin ML, Glaser JS: Idiopathic orbital myositis: report of six cases. *Arch Ophthalmol* 1982; 100:1261–1265.
12. Keane JR: Alternating proptosis: a case report of acute orbital myositis defined by the computerized tomographic scan. *Arch Neurol* 1977; 34:642–643.
13. Schwarzberg CI, LeGoff P, LeMenn G: Adult-onset Still's disease with muscular involvement: a review of five cases. (in French) *Sem Hop Paris* 1982; 58:2408–2412.
14. Ganday G, Imbert Y, Coomans D, Leng B: Still's disease in the adult. (in French) *Sem Hop Paris* 1981; 57:1885–1889.
15. Kemp AS, Searle C, Horne S: Transient Brown's syndrome in juvenile chronic arthritis. *Ann Rheum Dis* 1984; 43:764–765.

See also pp 92–93.

Massive gastric hemorrhage from the rupture of an arterial malformation

PIN KIE TENG, MD, SAM CHO, MD

Massive bleeding in the gastrointestinal tract from arterial malformations is rare. The cause of these malformations is not known. This report of a man with massive gastric hemorrhage emphasizes the importance of angiography in localizing the source of bleeding after endoscopy and surgery proved unsuccessful.

CASE REPORT

A 66-year-old man was admitted to the hospital because of severe hematemesis. Prior to admission, the patient had a viral infection for 10 days with persistent non-productive cough, malaise, and weakness. He had taken tetracycline and two to three tablets of aspirin on the day of admission. On that day he had vomited a large amount of bright red blood. He was weak and pale. He had had hypertension for 30 years and had been treated with propranolol, hydralazine, and spironolactone; the dose of these medications was not known. A cerebral vascular accident in 1969 and another in 1976 resulted in minimal spasticity of his left arm and leg. He was a moderate drinker until five years previously, when he stopped drinking. The physical examination and laboratory data were unremarkable, except for a blood urea nitrogen of 63 mg and non-specific ST-T wave changes in the electrocardiogram. His blood pressure was 140/80 mm Hg. The evening after admission hematemesis recurred. Endoscopy revealed no ulcer, esophageal varices, inflammation, neoplasm, or bleeding point. Clotted blood was seen in the fundus of the stomach. An emergency laparotomy, gastrostomy, gastro-duodenostomy, vagotomy, and pyloroplasty were performed. No bleeding source was found during the surgery. Drainage of a small amount of blood from the nasogastric tube was noted. On the fifth day after surgery the patient had another massive hematemesis of 1,500 cc of red blood. A second endoscopy was noncontributory. A celiac angiogram (Fig 1) demonstrated extravasation of contrast material in the upper posterior wall of the stomach. A second gastrostomy was carried out, and the bleeding point was ligated. The patient did well after surgery, until he died suddenly on the fourth day after surgery. An autopsy was performed.

At autopsy, the stomach was normal in size and shape. The serosa was shiny. In the posterior wall of the body, below the fundus and near the lesser curvature of the stomach, there was a round mucosal fold that was plicated and sutured. The mucosa of the entire stomach showed no ulcer, petechiae, inflammation, or neoplasm. The sutured mucosal folding identified on celiac angiography as the bleeding point contained a small submucosal artery (Fig 2) that was unusually thick walled with three well-outlined layers of a muscular type, typical of a medium-sized artery. The thickened wall was due to over development of the muscular media. Serial sections showed abrupt interruption of the media without aneurysmal dilation. An elastic stain showed interruption in the internal elastic lamina without fragmentation of the rest of the lamina.[1] Fibrinoid necrosis and giant cell inflammation of the artery were absent. There were no esophageal varices. The brain was not examined because of restrictions of the autopsy consent. The patient died of a massive pulmonary embolus.

DISCUSSION

Gastrointestinal hemorrhage of unknown origin is a vexing clinical problem and a formidable diagnostic challenge.[2,3] Barium enema studies of the upper and lower gastrointestinal tracts, sigmoidoscopy, endoscopy, and gastroduodenostomy may all be negative, as in this case. Even though there is no coagulopathy present,[4] the patient's life is still severely threatened by massive blood loss.

Gastrointestinal hemorrhage of unknown origin should be divided into upper and lower gastrointestinal causes. In this report the clinical evidence suggested that the source of bleeding was in the upper gastrointestinal tract.

Angiodysplasia or vascular ectasia must be considered and has recently been recognized as an important cause of gastrointestinal bleeding. The endoscopic findings of angiodysplasia are reddened mucosal folds and linear erythema described as angioid streaks. On histologic examination there are numerous dilated and congested blood vessels of different diameters in the submucosa. Moore et al[5] have classified these findings as a type I arteriovenous malformation. It is believed to be an acquired vascular change, usually found in atherosclerotic valvular disease with low cardiac output; it most frequently in-

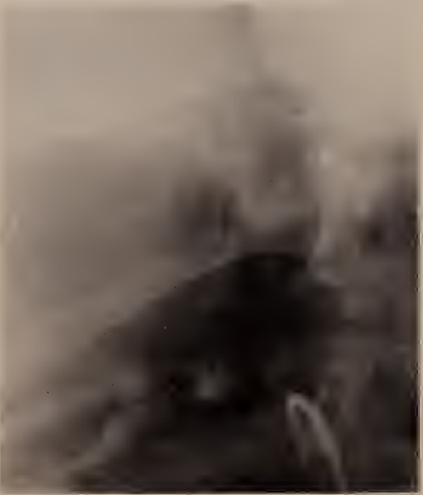


FIGURE 1. Celiac angiogram showing extravasation of contrast material in the upper posterior wall of stomach (arrow).

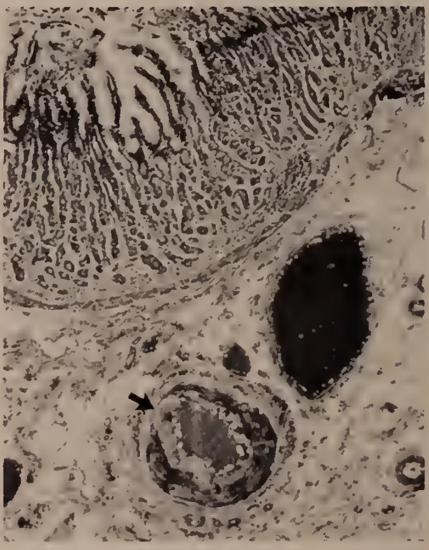


FIGURE 2. Abrupt interruption of an abnormally overdeveloped muscular media of a submucosal artery of the stomach (arrow). (Mason trichrome stain, original magnification ×100)

From the Departments of Pathology (Dr Teng) and Surgery (Dr Cho), Doctors Hospital, New York.

Address correspondence to Dr Teng, Director, Department of Laboratories, Doctors Hospital, 170 East End Ave at 87th St, New York, NY 10028.

volves the right and transverse colon.[6,7] Arterial malformations commonly occur in the upper part of the gastrointestinal tract.[8,9] They are thought to be congenital malformations, somewhat similar to berry aneurysms of the cerebral arteries.[10] The involved submucosal arteries are much larger than normal. They are tortuous and have the typical structure of larger muscular arteries, containing three distinct layers with thick muscular media. Arteriosclerosis of the intima is not necessarily present. If it is found, it is independent of the markedly thick media and typical of the congenital abnormality. It is important to differentiate those changes that may be arteriosclerotic in nature from those of a true congenital malformation.

The cases reviewed by Mandelbaum et al,[11] Goldman,[12] and Bailey[13] were similar to this case, although the latter had no minute mucosal erosion detectable to the naked eye. This case is more like the one reported by Richter,[14] with rupture of a single large submucosal artery that displayed no arteriosclerosis, aneurysm, or elastic abnormality. In reported cases of hemorrhage from gastric arterial malformation, the patients were usually less than 50 years of age, and one involved a 16-year-old.[8] The patient in this case was 66 years old, apparently the oldest case on record. Whether the hypertension and arteriosclerosis played a role in his illness is debatable. It seems unlikely that there was an association, as the pathognomonic lesion was in the media and not in the intima, and the patient's arterial blood pressure was well controlled at 140/80 mm Hg.

In the absence of a peptic ulcer, mucosal inflammation, drug administration,[15,16] and inflammation of the vessels,[17] the sudden interruption of the media of an abnormal gastric artery is likely to be a congenital malformation. The histopathology of the lesion proved this. From the clinical standpoint, it is important to emphasize the need for angiography in patients with massive bleeding in whom conventional techniques have failed to localize the source of bleeding.[18,19]

REFERENCES

1. McKusick VA: Pseudoxanthoma elasticum, in *Heritable Disorders of Connective Tissue*, 4th ed. St. Louis, CV Mosby Co, 1972, pp 475–520.
2. Palmer ED: The vigorous diagnostic approach to upper gastrointestinal hemorrhage: a 23-year prospective study of 1,400 patients. *JAMA* 1969; 207: 1477–1480.
3. Berquist TH, Nolan NG, Adson MA, Schutt AJ: Diagnosis of Meckel's diverticulum by radioisotope scanning. *Mayo Clin Proc* 1973; 48:98–102.
4. Cream JJ, Gumpel JM, Peachey RDG: Schönlein-Henoch purpura in the adult. A study of 77 patients with anaphylactoid or Schönlein-Henoch purpura. *QJ Med* 1970; 39:461–484.
5. Moore JD, Thompson NW, Appleman HD, et al: Arteriovenous malformations of the gastrointestinal tract. *Arch Surg* 1976; 111:381–389.
6. Welch CE, Athanasoulis CA, Galdabini JJ: Hemorrhage from the large bowel with special reference to angiodysplasia and diverticular disease. *World J Surg* 1978; 2:73–83.
7. Teague RH, Thornton JR, Manning AP, Salmon PR, Read AE: Colonoscopy for investigation of unexplained rectal bleeding. *Lancet* 1978; 1: 1350–1352.
8. Gough M: Submucosal arterial malformation of the stomach as the probable cause of recurrent severe hematemesis in a 16 year old girl. *Br J Surg* 1977; 64:522–524.
9. Sherman L, Shenoy S, Satchidanand SK, Neumann PR, Barrios GG, Peer RM: Arteriovenous malformation of the stomach. *Am J Gastroenterol* 1979; 72:160–164.
10. Glynn LE: Medial defects in the circle of Willis and their relation to aneurysm formation. *J Pathol Bacteriol* 1940; 51:213–222.
11. Mandelbaum I, Kaiser GC, Lempke RE: Gastric intramural aneurysm as a cause for massive gastro-intestinal hemorrhage. *Ann Surg* 1962; 155: 199–203.
12. Goldman, RL: Submucosal arterial malformation ("aneurysm") of the stomach with fatal hemorrhage. *Gastroenterology* 1964; 46:589–594.
13. Bailey ME: Rupture of a gastric artery aneurysm. *Can Med Assoc J* 1956; 75:131–133.
14. Richter, RM: Massive gastric hemorrhage from submucosal arterial malformation. *Am J Gastroenterol* 1975; 64:324–326.
15. Leonards JR, Levy G, Niemczura R: Gastrointestinal blood loss during prolonged aspirin administration. *N Engl J Med* 1973; 289:1020–1022.
16. Simon LS, Mills JA: Nonsteroidal antiinflammatory drugs. *N Engl J Med* 1980; 302:1179–1185, 1237–1243.
17. Cabal E, Holtz S: Polyarteritis as a cause of intestinal hemorrhage. *Gastroenterology* 1971; 61: 99–105.
18. Pillow RP, Hill LD, Ragen PA, Siemen JS, Walker LA: Newer methods for localization of obscure small-bowel bleeding. *JAMA* 1962; 179:23–26.
19. Retzlaff JA, Hagedorn AB, Bartholomew LG: Abdominal exploration for gastrointestinal bleeding of obscure origin. *JAMA* 1961; 177:104–107.

See also pp 93–94.

Amylase in serous ovarian neoplasms

GITA RAMASWAMY, MD, VENKITA JAGADHA, MD, VICTOR TCHERTKOFF, MD

There are many sources of elevated tissue amylase aside from the pancreas[1-8]—salivary gland, bile duct epithelium, fallopian tube epithelium. Other pertinent sources of increased amylase levels in the blood are neoplasms of the lung, ovary, breast, parotid region, and prostate, and Hodgkin's disease.[2,4,5,7,8] Recent publications point to amylase as a marker for serous ovarian neoplasms.[1,9,10] This paper reaffirms such an association.

CASE REPORT

A 51-year-old woman with adult-onset diabetes mellitus and manic-depressive psychosis was admitted with a complaint of increasing abdominal girth. The patient had had diabetes mellitus for six years, with one episode of diabetic ketoacidosis. She had received neutral protamine Hagedorn insulin for the diabetes and lithium for the manic-depressive illness. She claimed to have gained over 50 lb during the six months prior to admission—the weight gain having been refractory to diet control. The patient also complained of mild dyspnea on lying supine. She denied nausea, vomiting, jaundice, or abdominal pain. The only surgical procedure she had undergone was a hemorrhoidectomy 11 years prior to admission.

Physical examination revealed a markedly obese female in no acute distress, with blood pressure, 120/80 mm Hg; pulse rate, 100 beats/min; respiratory rate, 24/min; and temperature, 98.4°F. There was no scleral icterus, and the neck was supple without adenopathy. The lungs were clear, with no evidence of pleural effusion. The heart sounds were within normal limits.

The abdomen was markedly distended, tense, and not painful. Fluid wave or shifting

---

From the Department of Pathology, New York Medical College—Metropolitan Hospital Center, New York.

Address correspondence to Dr Tchertkoff, Chief, Department of Pathology, Metropolitan Hospital Center, 1901 First Ave, New York, NY 10029.

dullness could not be elicited. The liver and spleen were not palpable. Bowel sounds were unremarkable. There was 2+ pitting edema of both lower extremities. There was no neurologic deficit.

Routine laboratory tests disclosed no abnormalities. The serum amylase level was 170 Caraway units (normal range, 60 to 160 Caraway units). An electrocardiogram and chest roentgenogram were within normal limits. A flat plate roentgenogram of the abdomen had a diffuse ground-glass appearance.

The clinical impression was of ascites, and paracentesis was performed. Clear, colorless fluid was removed, which had a lactate dehydrogenase level of 8 U/L, and an amylase value of 970 Caraway units—predominantly of the salivary type as determined by polyacrylamide gel electrophoresis.

An upper gastrointestinal series disclosed loops of small bowel displaced to the right by large cystic masses. The patient underwent an exploratory laparotomy with hysterectomy and bilateral cystectomy.

Gross pathologic examination of the resected surgical specimen showed bilateral ovarian cysts; the right cyst measured 15 × 15 × 10 cm and weighed 1,550 g. The external surface was smooth and glistening. Cut sections contained a multilocular cyst filled with clear, colorless, watery fluid. The cyst measured 0.2 cm in thickness, and the internal surface was smooth and glistening. The left ovarian cyst measured 21 × 20 × 8 cm and weighed 2,860 g. The external surface and smooth and white, while the inner surface had numerous friable papillary projections that measured 2 × 2 × 1 cm in greatest dimensions. The rest of the inner surface appeared granular. The contents of the cyst were watery and clear.

On microscopic examination the right ovarian cyst was lined with a single layer of cuboidal or ciliated columnar epithelial cells, with no cellular atypia, and was interpreted as a benign serous cystadenoma (Fig 1). The left ovarian cyst was also lined with ciliated tall columnar epithelial cells, but it exhibited cellular stratification and complicated papillary and glandular patterns (Fig 2). There was moderate cellular atypia but no stromal invasion. The epithelium stained strongly positive with the periodic acid-Schiff stain with diastase digestion, and failed to stain positive for mucin, indicating that this was a papillary serous cystadenocarcinoma of low-grade malignant potential.

The patient had an uneventful recovery and is well more than four years after surgery.

DISCUSSION

While it is imperative to consider and exclude a pancreatic cause of high amylase in the body fluids and serum, other sources enter into the differential diagnosis.[1-8] Salivary gland disease, cerebral trauma, appendicitis, perforated peptic ulcer, acute gall bladder disease, choledocholithiasis, biliary dyskinesia, pregnancy, shock, diabetic ketoacidosis, burns, infectious mononucleosis, macroamylasemia, fallopian tube afflictions including ectopic pregnancy, gonococcal salpingitis and hydrosalpinx, and ruptured graafian follicles are known to produce hyperamylasemia.[10-16] Tumor amylasemia may be an important indicator of tumors of the lung, ovary, and parotid region, metastatic breast carcinoma, prostatic cancer, and Hodgkin's disease.[2,4,5,7,8]

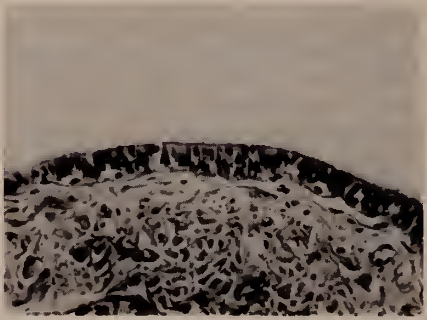


FIGURE 1. Ciliated columnar epithelial cells lining the serous cystadenoma of the right ovary. (hematoxylin and eosin, original magnification ×450)

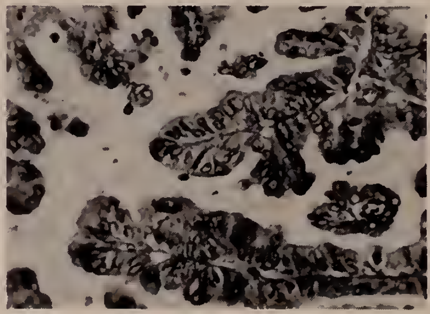


FIGURE 2. Papillary serous cystadenocarcinoma of the left ovary, of borderline type, showing cellular stratification and pleomorphism. (hematoxylin and eosin, original magnification × 450)

High amylase levels in the pleural fluid have been observed in congestive heart failure associated with tuberculosis, pulmonary embolism, pneumothorax, and esophageal perforation.[4] An elevated amylase level may be produced by obstruction of the thoracic duct by a non-amylase-producing tumor.

Abdominal conditions with high ascitic fluid amylase levels include pancreatic adenocarcinoma, metastatic breast carcinoma, ovarian cystadenocarcinoma,[4] renal cell carcinoma,[3] and esophageal leiomyosarcoma.[3]

In ovarian neoplasms, high amylase levels are seen in tumors with an endosalpingeal type of epithelial lining—serous tumors. Vankey et al[1] reported amylase activities averaging 2,253 U/L in serous tumors and 77 U/L in nonserous tumors. The amylase activities in the serous tumors consistently exceeded the limit of the serum reference ranges.

The concept of amylase as a marker for serous tumors is supported by the resemblance of the epithelium of the tumors to the lining of the normal fallopian tube, and the mounting evidence that amylase is a product of normal endosalpingeal epithelial cells.[6] Endosalpingeal epithelium—unlike the pancreatic, salivary, or lung epithelium—is of Muellerian origin, with a mesodermal derivation, and it releases secretions directly into a large caliber lumen, apparently by "pinching off" the apical cytoplasm. Endosalpingeal amylase is thought to act on endosalpingeal and endometrial glycogen to provide glucose as a nutrient for early embryonic development after fertilization. Fluid from cases of hydrosalpingeal and parasalpingeal cysts are consistently rich in amylase, offering further proof of amylase production. Ultrastructural examination has verified the presence of varying numbers of apical secretory granules of variable electron density within these cells.

Because serum amylase has components of varied origin, research has focused on defining isoenzyme patterns in the differential diagnosis of hyperamylasemia. When separated by electrophoretic methods and column chromatography, the two major isoenzyme types—pancreatic and salivary—have been shown to be composed of multiple subtypes. Serous type ovarian tumors contain an acidic isoenzyme of amylase closely akin to the salivary isoenzyme. The acidic nature does not appear to be due to sialic acid residues, because the chromatographic pattern is unaffected by treatment with neuraminidase.[9] Zakowski et al,[10] using an affinity chromatographic procedure, were able to purify and characterize serous ovarian tumor amylase. It differs from the pancreatic isoenzyme in its specific activity, doublet formation on sodium dodecyl sulfate-polyacrylamide gel electrophoresis, and sensitivity to specific alpha-amylase inhibitors. Although the two isoenzymes are very similar, the salivary isoenzyme differs from serous tumor amylase in its net electrical charge.

Among gynecologic neoplasms, ovarian cancer is the leading killer in the United States. Of these, the serous neoplasms are the most frequent. Being a silent growth, the tumor may already be advanced or disseminated at initial presentation. The association of high amylase levels with serous ovarian tumors therefore has far-reaching implications for the use of amylase values as a diagnostic tool. Many other markers have been described for serous ovarian tumors, including ribonuclease, lactate dehydrogenase, human chorionic gonadotropin, carcinoplacental isoenzymes of alkaline phosphatase, urinary nonesterified cholesterol,[11-14] and antigenic markers like carcinoembryonic antigen and alpha-fetoprotein.[15] Carcinoembryonic antigen and alpha-fetoprotein are nonspecific and are detectable in gynecologic and non-gynecologic lesions, whereas the others primarily distinguish benign from malignant lesions.[1,13,15] Amylase is unique in its specificity for serous ovarian tumors, in that it also represents a specific differentiated function. Several investigators have reported that increased levels may fluctuate with altered tumor burden, surgery, and chemotherapy.[4]

This patient represents an example of serous cystadenocarcinoma and serous cystadenoma of the ovary with an increased tumor amylase level, stressing the importance of screening for nonpancreatic causes of increased amylase values in body fluids.

REFERENCES

1. Van Kley H, Cramer S, Bruns DE: Serous ovarian neoplastic amylase (SONA): a potentially useful marker for serous ovarian tumors. *Cancer* 1981; 48:1444-1449.
2. Ammann RW, Berk JE, Fridhandler L, Ueda M, Wegmann W: Hyperamylasemia with carcinoma of the lung. *Ann Intern Med* 1973; 78:521-525.
3. Ende N: Studies of amylase activity in pleural effusions and ascites. *Cancer* 1960; 13:283-287.
4. Lehrner LM, Ward JC, Karn RC, Ehrlich CE, Merrit AD: An evaluation of the usefulness of amylase isoenzyme differentiation in patients with hyperamylasemia. *Am J Clin Pathol* 1976; 66:576-587.
5. Salt 2nd WB, Schenker S: Amylase—its clinical significance: a review of the literature. *Medicine (Baltimore)* 1976; 55:269-289.
6. Bruns DE, Mills SE, Savory J: Amylase in fallopian tube and serous ovarian neoplasms: immunohistochemical localization. *Arch Pathol Lab Med* 1982; 106:17-20.
7. Light RW, Ball WC: Glucose and amylase in pleural effusions. *JAMA* 1973; 225:257-260.
8. Hammarsten JF, Honska WL, Limes BJ: Pleural fluid amylase in pancreatitis and other diseases. *Am Rev Tuberc* 1979; 79:606-611.
9. Zakowski JJ, Bruns DE: Improved DEAE-Sephadex column chromatography in measuring amylase in serous ovarian neoplasms and results for 13 cases. *Clin Chem* 1982; 28:2095-2098.
10. Zakowski JJ, Gregory MR, Bruns DE: Amylase from human serous ovarian tumors: purification and characterization. *Clin Chem* 1984; 30:62-68.
11. Acevedo HF, Campbell EA, Frich Jr Jc, Hayeslip DW, Gilmore J: Urinary cholesterol. VIII. Its excretion in women with ovarian neoplasms. *Cancer* 1976; 37:2847-2857.
12. Awais GM: Serum lactic dehydrogenase levels in the diagnosis and treatment of carcinoma of the ovary. *Am J Obstet Gynecol* 1973; 116:1053-1057.
13. Fishman WH, Raam S, Stolbach LL: Markers for ovarian cancer: Regan isoenzyme and other glycoproteins. *Semin Oncol* 1975; 2:211-216.
14. Sheid B, Lu T, Pedrinan L, Nelson JH, Jr.: Plasma ribonuclease: a marker for the detection of ovarian cancer. *Cancer* 1977; 39:2204-2208.
15. Barlow JJ, Bhattacharya M: Tumor markers in ovarian cancer: Tumor associated antigens. *Semin Oncol* 1975; 2:203-209.

Gruntzig balloon dilation for fibromuscular dysplasia of the internal carotid arteries

ETHAN L. WELCH, MD, JOYCE A. LEMKIN, MD, PHD, JOSEPH E. GEARY, MD

Although fibromuscular dysplasia is most often seen in the renal arteries, the second most common site is in the internal carotid artery.[1,2] Graduated dilation using Bake's dilators has provided long-term relief of symptoms, and rates of recurrence are low.[3,4] Recent advances in the use of Gruntzig balloon angioplasty catheters prompted an improved modification of the technique of dilating the fibromuscular lesion in the internal carotid artery.[5-7] We report on two patients in which the balloon angioplasty technique was applied through an open arteriotomy for the correction of symptomatic bilateral internal carotid artery fibromuscular dysplasia.

CASE REPORTS

Case 1. A 53-year-old right-handed woman was referred to us by her family physician because of complaints of dizzy spells and headaches for more than a year. The onset of these symptoms was gradual. The disequilibrium was unrelated to time or orthostatic cause and lasted from a few seconds to several minutes. The headaches occurred occasionally without the accompanying disequilibrium. She denied visual symptoms but did experience episodes of numbness in the right hand. Her symptoms were unrelieved by aspirin therapy during a six-month period prior to consultation. There were no complaints of motor dysfunction. She had been otherwise in good health.

Low grade bilateral carotid bruits were noted on physical examination. Her blood pressure was 130/70 mm Hg in both arms. There were no abnormal neurologic findings. Ophthalmoplethysmographic readings were 112 mm Hg on the right and 110 mm Hg on the left. Arch arteriography with subtraction views demonstrated bilateral fibromuscular dysplasia in the internal carotid arteries extending to the base of the skull (Fig 1). There was no evidence of intracranial aneurysm formation. A computed tomographic (CT) scan, including posterior fossa, was unremarkable. No fibromuscular dysplasia of the renal arteries was demonstrated on an abdominal aortogram. Although her symptoms were primarily global, there were some lateral effects originating in the left hemisphere. This finding, together with the extent of the disease seen on angiography, prompted a recommendation for elective surgery.

Under general anesthesia, the treatment consisted of arteriotomy at the left carotid bifurcation and dilation of the internal carotid artery at sequential points using a 7 French, 4-mm Gruntzig balloon catheter, starting at the base of the skull and finishing at the bifurcation. Each dilation lasted for 15 seconds with 3 atmospheres of pressure (Fig 2). At the

---

From the Section of Vascular Surgery (Drs Welch and Geary) and the Department of Radiology (Dr Lemkin), Rochester General Hospital and The University of Rochester School of Medicine and Dentistry, NY.

Address correspondence to Dr Welch, Attending Surgeon, Section of Vascular Surgery, Rochester General Hospital, 1425 Portland Ave, Rochester, NY 14621.

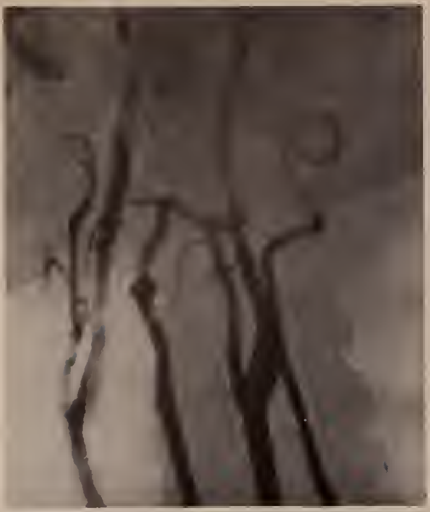


FIGURE 1. Subtraction view of four-vessel arch study showing fibromuscular dysplasia of both intimal carotid arteries extending to the base of the skull.

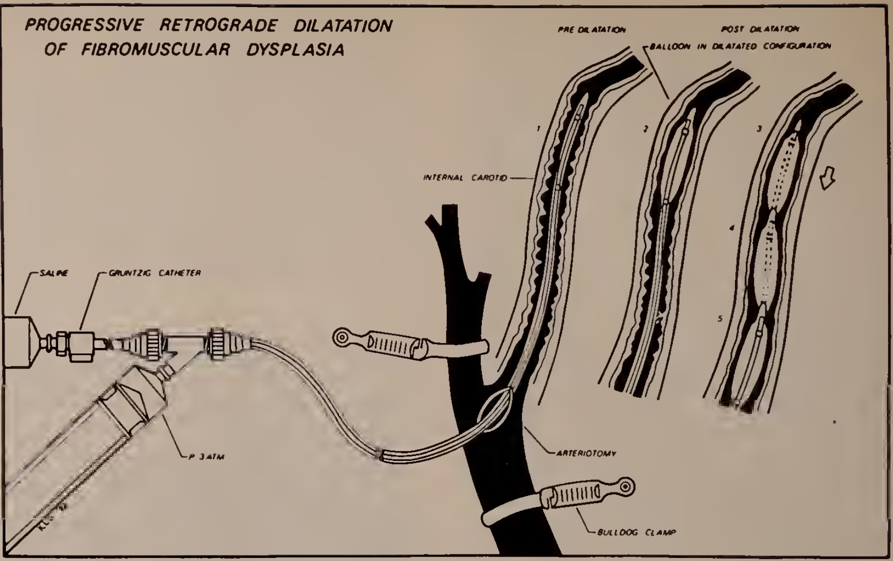


FIGURE 2. Dilation technique starting from the most cephalad point and proceeding toward the bifurcation.

completion of each dilation, the catheter was deflated completely before being moved to a new position. Following removal of the Gruntzig catheter, retrograde flow was flushed to remove debris and to avoid embolism formation. No systemic heparinization was employed. After closing the arteriotomy, angiography was performed at the operating table to document the result (Fig 3). Her postoperative course was uneventful.

In view of the extensive disease in the right internal carotid system, the procedure was also suggested for that side. The same procedure was performed on the right carotid system six weeks following the first surgery. The patient was treated with dipyridamole, 25 mg administered orally three times per day for three months to provide protection against platelet emboli during completion of intimal healing. She has remained asymptomatic two and a half years after the procedures.

Case 2. A 56-year-old woman sought treatment for intermittent light-headedness and dizziness of two years' duration. A particularly bothersome episode brought her to her family physician. She was noted to be moderately obese. Her blood pressure was 120/60 mm Hg in both arms, and a bruit was heard over the left carotid artery. Non-invasive vascular laboratory studies provided an ophthalmoplethysmographic value of 102 mm Hg bilaterally, and ultrasound images showed minimal plaque in the left carotid bifurcation. Arch angiography demonstrated bilateral internal carotid artery fibromuscular dysplasia (Fig 4). Minimal plaque was also noted in the left bifurcation. A cerebral CT scan did not show evidence of an intracranial disorder. There was no evidence of subclavian disease.

Results of a neurologic examination confirmed the conclusion that the symptoms were primarily global, and that the bilateral fibromuscular dysplasia was responsible, noting the absence of other causes of disequilibrium syndromes and light-headedness. There was no evidence of cardiac disease, acoustic neuroma, or subclavian steal syndrome.

Because of the extent of the disease seen on arteriography, it was thought that a surgical

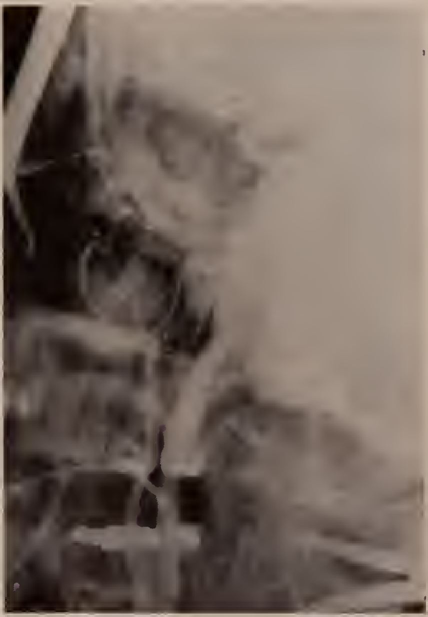


FIGURE 3. Post-dilation angiogram done in the operating room.

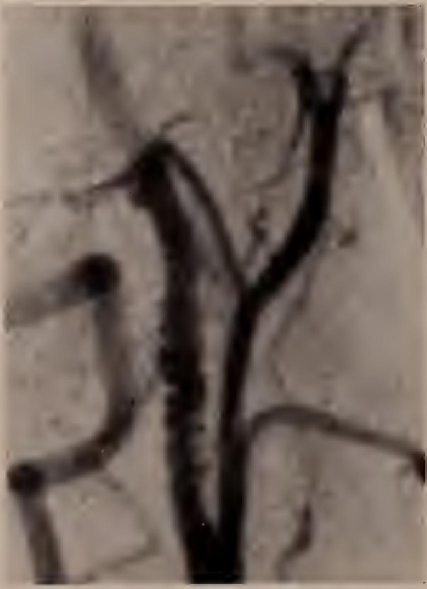


FIGURE 4. Subtraction view from arch study of case 2 showing typical fibromuscular dysplasia changes in right intimal carotid artery.

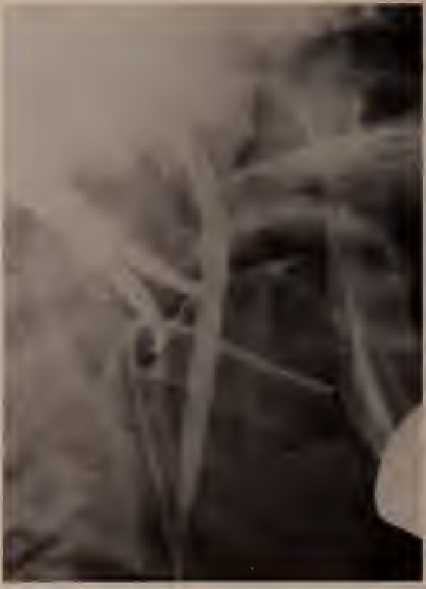


FIGURE 5. Post-dilation angiogram at completion of surgery on right intimal carotid artery in case 2.

approach would provide the only permanent result; a trial of therapy with platelet inhibitory drugs was not selected.

She was admitted to the hospital and underwent an open right internal carotid Gruntzig dilation procedure as described in case 1. Six weeks later, the same procedure was performed on the left side. Figure 5 shows the right post-dilation view at surgery. The procedures produced no complications. She was also treated with dipyridamole, 25 mg orally three times per day for three months. She remains asymptomatic without medication one year after the last procedure.

DISCUSSION

The pathogenesis of carotid fibromuscular dysplasia is not entirely known, as Wells et al[8] show in their series. The exact cause of its symptoms is also not clear. Collins et al,[9] in an excellent summary of the causes of symptoms in fibromuscular dysplasia point out the following: (1) Decreased blood flow may account for some symptoms, as Morris et al[10] demonstrated a gradient across lesions in some cases. (2) Cerebral/retinal embolization is an attractive hypothesis but has been documented only in single instances by Collins,[9] Gee,[11] and Perry.[12]

Lateralizing symptoms seem to be most easily explained by a platelet embolization theory. Less easily understood is the presence of nonlateralizing or global symptoms, as noted in these two patients. In patients with bilateral extensive disease of the internal carotid, we can only suppose that there exists some embolic or flow problem, or perhaps a combination of these. The symptoms did not recur. This leads to the conclusion that the procedure should not be restricted to patients with transient ischemic attacks.

The graduated metal dilation procedure has not been without complications.[4,13] Late angiographic studies were not done on these patients because they remained asymptomatic, but it is recognized that such information may prove critical to the long range understanding of this problem. Dissection, or a "snowplow effect," is always a possibility. Since balloon dilation has been used successfully elsewhere in the vascular system, it seemed a logical alternative to graduated dilation. Percutaneous transluminal angioplasty has been used to dilate successfully critical stenoses of the internal carotid artery caused by fibromuscular dysplasia,[6] and, recently, Garrido and Montoya[7] reported dilation via the lumen of the common carotid artery, but not via arteriotomy and dilation followed by adequate retrograde flushing. Although embolic phenomena have not been specifically reported in the few cases done by a closed technique, embolic problems have been reported following dilation at other points in the vascular system to be as high as 11.4%.[14] The use of a Gruntzig balloon with an open technique at surgery seems to avoid both the potential embolic complications of angioplasty and the possible dissection problem of a graduated metal dilation technique.

REFERENCES

1. Ehrenfeld WK, Wylie EJ: Fibromuscular dysplasia of the internal carotid artery. *Arch Surg* 1974; 109:676-681.
2. Houser OW, Baker Jr HL, Sandok BA, Holley KE: Cephalic arterial fibromuscular dysplasia. *Radiology* 1971; 101:605-611.
3. Effeney DJ, Ehrenfeld WK, Stoney RJ, Wylie EJ: Fibromuscular dysplasia of the internal carotid artery. *World Surg* 1979; 3:179-186.
4. Starr DS, Lawrie GM, Morris Jr GC: Fibromuscular disease of carotid arteries: long term results of graduated internal dilatation. *Stroke* 1981; 12:196-199.
5. Grüntzig A, Kumpe DA: Technique of percutaneous transluminal angioplasty with the Grüntzig balloon catheter. *AJR* 1979; 132:547-552.
6. Hasso AN, Bird CR, Zinke DE, Thompson JR: Fibromuscular dysplasia of the internal carotid artery: percutaneous transluminal angioplasty. *AJR* 1981; 136:955-960.
7. Garrido E, Montoya J: Transluminal dilatation of internal carotid artery in fibromuscular dysplasia: a preliminary report. *Surg Neurol* 1981; 16:469-471.
8. Wells RP, Smith RR: Fibromuscular dysplasia of the internal carotid artery: a long term follow-up. *Neurosurgery* 1982; 10:39-43.
9. Collins GJ, Rich NM, Clagett GP, Spebar MJ, Salander JM: Fibromuscular dysplasia of the internal carotid arteries. *Ann Surg* 1981; 194:89-96.
10. Morris GC Jr, Lechter A, DeBakey ME: Surgical treatment of fibromuscular disease of the carotid arteries. *Arch Surg* 1968; 96:636-643.
11. Gee W, Burton R, Stoney RJ: Atypical fibromuscular hyperplasia involving the carotid artery. *Ann Surg* 1974; 180:136-138.
12. Perry MO: Fibromuscular disease of the carotid artery. *Surg Gynecol Obstet* 1972; 134:57-60.
13. Balaji MR, DeWeese JA: Fibromuscular dysplasia of the internal carotid artery: its occurrence with acute stroke and its surgical reversal. *Arch Surg* 1980; 115:984-986.
14. Greenfield AJ: Femoral, popliteal, and tibial arteries: percutaneous transluminal angioplasty. *AJR* 1980; 135:927-935.

SUMMER RESEARCH ASSISTANT POSITION AT THE *JOURNAL*

Applications are invited from medical students (including those who will be entering medical school in the fall) for the position of research assistant at the *New York State Journal of Medicine*. This 10-week paid position is an opportunity for a student to participate in the editorial process of a peer reviewed scientific journal and to develop an original project for possible publication. Inquiries should be directed to the Editor. Applications should consist of a curriculum vitae and a covering letter. Deadline: May 1.

DIFFERENTIAL THERAPEUTICS IN PSYCHIATRY: THE ART AND SCIENCE OF TREATMENT SELECTION

By Allen Frances, MD, John Clarkin, PhD, Samuel Perry, MD. 420 pp. New York, Brunner/Mazel Publishers, 1984. $30.00 (hardcover)

The impressive advances in the field of psychiatry following World War II resulted in vast changes in this medical specialty. Now there is a book that undertakes not only the assessment of this knowledge but an explanation of how to utilize it most effectively for individual patients. It is a valuable book and fulfills a need that the psychiatric community will fully appreciate.

The authors, two physicians, Drs Allen Frances and Samuel Perry, and a psychologist, Dr John Clarkin have a rich and diversified background in mental health care. All are senior members of the faculty at the Cornell University Medical College. Their style of writing is crisp and concise with a clarity not frequently encountered in current literature. Each chapter is carefully organized and so skillfully presented that, at times, the reader can almost experience the feeling of being present at a stimulating staff conference. All of the 11 chapters are meaningful and rewarding, but two deserve special mention: "Combination of Treatments" and "Research and Treatment Planning."

This book also deserves praise for its presentation of a broad spectrum of treatment choices. This permits a choice of treatment based solely on what will be most beneficial for the patient. Such choices are even more meaningful today, when cost containment is emphasized in every branch of medicine. Despite this, the authors are firm in their convictions and are able to write that "the least expensive acceptable treatment is by no means necessarily the treatment of choice." The humanist physician Sir William Osler would have been pleased to read this.

I can recommend this book most enthusiastically and without any qualifications. Although it is primarily addressed to professionals working in the field of mental health, it will prove interesting and valuable to all physicians. The frequent interlacing of clinical and theoretical material and the extensive references add greatly to its uniqueness. I hope that in the near future, Drs Frances, Clarkin, and Perry will provide us with another gem in their field of differential therapeutics in psychiatry.

STEPHEN NORDLICHT, MD
Doctors Hospital
New York City 10128

INVASIVE URORADIOLOGY. A MANUAL OF DIAGNOSTIC AND THERAPEUTIC TECHNIQUES

By Morteza K. Elyaderani, MD, Stanley J. Kandzari, MD, Wilfrido R. Castaneda, MD, Paul H. Lange, MD. 250 pp, illustrated. Lexington, MA, Collamore Press, 1984. $40.00 (hardcover)

In recent years many hospital departments of radiology and urology have become active in the new field of invasive uroradiology. Although it is also known as endourology, I agree with the authors' use of the term invasive uroradiology—it more accurately reflects the cooperative effort required between radiologists and urologists. This field has had a significant impact on the diagnosis and treatment of various urologic disorders and has resulted in a significant reduction in patient morbidity and length of hospital stay. This text, a collaborative effort between radiologists and urologists from the schools of medicine at West Virginia University and the University of Minnesota, is "designed to meet the need for a practical, current, and comprehensive manual" of invasive uroradiology.

The authors appropriately begin with a discussion of the imaging methods that can be used. They note that these procedures can be performed with either ultrasound, computed tomography (CT), fluoroscopy, angiography, or combinations of these. Ultrasound is clearly their imaging technique of choice, and in the introductory chapter they give a brief overview of renal ultrasound, emphasizing its application in the diagnosis of hydronephrosis and renal calculi. The major endourologic procedures are presented, including antegrade pyelography and the ureteral perfusion test, percutaneous nephrostomy, ureteral stent insertion and brush biopsy, nephrolithotomy, retrograde extraction, chemolysis, intra-operative ultrasonographic localization of urinary calculi, percutaneous aspiration and biopsy procedures, percutaneous transluminal renal angioplasty, renal embolization, and pediatric and intrauterine percutaneous techniques.

This text meets its primary objective of providing explicit instructional information. Each procedure is clearly and concisely described. While the emphasis is on technique, clinical indications, contraindications, and complications are also discussed.

As one involved in imaging, I was impressed with the excellent reproductions of radiographic, CT, and ultrasound images. There are helpful step-by-step graphic illustrations of each of the major procedures described, which allow for a clear understanding of the techniques. Supplementary photographs of equipment are provided as necessary. The chapter bibliographies are comprehensive and up-to-date.

I would strongly recommend this text for any professional interested in or involved in the field of invasive uroradiology.

PHILIP C. MOUDY, MD
Millard Fillmore Hospital
Buffalo, NY 14209

CANCER THERAPY IN CHILDREN

Edited by Carl Pochedly, MD. 233 pp, illustrated. Thorofare, NJ, Slack, Inc, 1983. $27.50 (hardcover)

The theme of this textbook is the multifaceted management of pediatric patients with cancer. The editor claims that the book contains "detailed descriptions of recommended therapy regimens for the management of various childhood malignancies" and that it is "useful as a guide for all primary physicians who care for children with cancer." Dr Pochedly has gathered an impressive list of pediatric oncologists to write the individual chapters.

In the past few years there have been many publications dealing with the specialty of pediatric oncology. This book is not as detailed as *Clinical Pediatric Oncology* by Sutow, Fernback, and Vietti (Mosby, 1984) and is not a very good guide for pediatric oncologists. Most of the chapters are summaries of the classic diagnostic work-ups for the individual diseases and current results with various treatment schemes. Besides lacking detail, there is no discussion about the management of the patient with recurrent or metastatic disease. It is the latter patient that makes up approximately 50% of the pediatric oncologist's practice and who puts the most demand on the pediatric oncologist for seeking sources of new treatment and expertise in management.

Other general deficiencies in the book that detract from its ability to be a definitive source for the pediatric oncologist include the lack of discussion of new advances and new forms of experimental treatment. In particular, there is no mention of the role of magnetic resonance imaging in the diagnosis and evaluation of pediatric solid tumors. Clearly, in the field of pediatrics, magnetic resonance imaging will be superior in many areas over the computed tomographic (CT) scan. At the present time, CT scans, lymphangiograms, and intravenous pyelograms, as described in the text, can hardly be considered new advances in the diagnosis of pediatric malignancies.

Despite these deficiencies, this textbook is useful: it is an excellent source book for medical students, house staff, and the general pediatric practitioner. It describes the classic approach to the work-up of children with primary malignancies and the diagnosis of their disorders, and it alerts the pediatrician to the good results obtained with current treatments for malignant disorders that may rarely be seen in practice.

Most of the chapters are written with simplicity and clarity. Particularly well written are the chapters on acute lymphocytic leukemias (Bleyer), rhabdomyosarcomas (Ghavimi), pediatric brain tumors (Walker and Allen), retinoblastoma (Wolff), and bone marrow transplantation (Lenarsky and Feig). It is this latter chapter that brings fresh material to the literature on pediatric oncology by discussing this promising new method for the treatment of pediatric leukemias, with a hint given at the end of the chapter to its possible usefulness in the treatment of solid tumors. Even this chapter, however, is not written in sufficient depth to be useful as a guide to the pediatric oncologist. However, it does conform to the overall effect of the book as being an excellent source for the general pediatrician, and an introduction to pediatric oncology for the medical student and house officer.

GERALD ROSEN, MD
UCLA Medical Center
Los Angeles, CA 90024

ANTINUCLEAR ANTIBODIES. CONTEMPORARY TECHNIQUES AND CLINICAL APPLICATION TO CONNECTIVE TISSUE DISEASES.

By Gale A. McCarty, Donald W. Valencia, Marvin J. Fritzler. 95 pp, illustrated. New York, Oxford University Press, 1984. $29.95 (hardcover)

This book purports to give a brief background of rheumatic diseases associated with positive antinuclear antibody (ANA) tests and to explain the current methodology and limitations of the ANA. The book is meant for use in a laboratory setting and is aimed at internists, rheumatologists, and laboratory technicians. The authors are two immuno-rheumatologists who have worked on the standardization of ANA reagents, and the president of a firm that specializes in immunologic diagnostic tests.

While the concept of this book is good, the presentation is not ideal. The book begins with a synopsis of rheumatic diseases which is both sketchy and, in places, annoyingly picayune. In a book on ANA, is it necessary, or even accurate, to discuss the rare resemblance of a rash seen in patients with lupus erythematosus to lichen planus, transverse pitting of the nails that occurs in 6% of patients with lupus, aspiration pneumonitis in dermatomyositis, the technique used in a rose bengal dye test for Sjogren's syndrome, or the resemblance of scleroderma to Werner's syndrome and progeria?

In the discussion of ANA, one chapter is devoted to fluorescence microscopy, with suggestions for good technique and maintenance of equipment but no warnings, for instance, about the fading of fluorescence intensity with continued observation. The authors discuss rare antigens and many substrates and techniques.

Much emphasis is placed on fluorescence ANA, but discussion of counterimmunoelectrophoresis tests for Ro, La, Sm, and ribonucleoprotein precipitins are squeezed into one chapter. There is no discussion of automated techniques for reading ANA tests. Methods are reviewed in discursive rather than tabular form, which makes for easy reading, but also makes it more difficult to use the text as a reference source or to look up specific points.

The book was not well proofread. For instance, when the Ro and ANA antigens are introduced, the reader is twice referred to page "00" for fuller discussion. There are 169 references, almost all clinical, some to textbooks and many relevant to the clinical diagnosis of rheumatic disease rather than to ANA.

This book will be useful primarily to the laboratory technician who asks "Why am I doing this test and what does it mean?" It is not sufficiently well structured to enable one to set up an assay laboratory. Physicians concerned with laboratory error, variability, and interpretation of results would better profit by reading the original articles upon which this book is based.

MICHAEL DAN LOCKSHIN, MD
Hospital for Special Surgery
New York City 10021

PROSPECTIVE PAYMENT. WHAT IT IS/HOW TO COPE. YOUR QUESTIONS ANSWERED

By International Health Services, Ltd. 134 pp, illustrated. Concord, MA, International Health Services, 1983. $10.00 (softcover)

This book reports on a survey of more than 500 hospital executives conducted just prior to the activation of the federal prospective payment system (PPS) on October 1, 1983. Despite the fact that the federal regulations were modified as of January 3, 1984, the contents of this volume are still valuable to health care providers. Currently, the federal government's PPS only applies to hospital inpatients covered by Medicare. However, the legislation does imply that physician fees as well as costs for ambulatory care will be included in the future.

Packed into 134 pages, in an easy to read style, are an executive summary, a PPS summary, reactions of insurers, reactions of providers, and a future orientation section. In addition, there are seven appendices, 17 figures, and 13 tables. Readers will appreciate ready access to the information outlined in a five-page table of contents.

The topics covered include Medicare, diagnosis related groups (DRGs), casemix issues, outliers, pass-throughs, capital expense problems, return on equity, medical education expenses, Peer Review Organizations (PROs), Preferred Provider Organizations (PPOs), Health Maintenance Organizations (HMOs) as revenue producers, and the non-profit response to DRGs. The book enables readers to become familiar with the latest additions added to the jargon of the United States health care system.

Hospital executives reported that they anticipated having to alter their data processing activities significantly to comply with the DRG system. That prediction has been borne out in the actual experience since implementation on October 1, 1983. An article in the August 1984 issue of *Fortune*, discussing software for DRGs, noted that "the companies that provide computer software programs to hospitals are busier than an emergency room on New Year's Eve."

Whereas 28% of the executives surveyed felt that the impact of the DRG system was unknown, 38% expected a negative financial reaction, and 24% anticipated a favorable financial impact. Only 10% expected no change. Experience, according to the federal government, revealed large savings from the DRG cost-containment program. Judging from the critical articles in the professional literature, more hospitals are being negatively affected than was anticipated in the survey.

Strategies suggested to accommodate the lower income generated include staff layoffs, inventory reductions, greater use of generic drugs, smaller pharmaceutical formularies, group negotiated purchasing, prime vendor contracting, more standardization in supplies, and tighter inventory controls.

Since the sponsors of this investigation included about 20 companies engaged in business with hospitals and health care providers, there is an emphasis on the impact of the DRG system on specifics such as purchasing medical supplies and buying drugs and equipment.

This survey was supervised by a management consulting firm, and the research team included representatives from the American Hospital Association, Hospital Corportion of America, a private law firm, and consulting firms. In-depth interviews and phone calls were used to collect the data.

This book is highly recommended for people who want to digest complex material about prospective payment without indigestion.

ALLEN SPIEGEL, PHD
SUNY-Downstate Medical Center
New York City 11203

HEALTH AND HEALING. UNDERSTANDING CONVENTIONAL AND ALTERNATIVE MEDICINE

By Andrew Weil, MD. 296 pp. Boston, Houghton Mifflin Co, 1983. $13.95 (hardcover)

Andrew Weil provides medical practitioners and the general public with valuable insights into the nature of illness as perceived by the person as "mindbody." This book is an attempt to place modern scientific medicine in a context that allows practitioners to maximize the healing powers available to them beyond the technical aspects of treatment. The author provides the reader with a useful description of the current understanding of placebo responses:

> I recognize three dimensions of belief of placebo responses to active treatments: The patient's belief in the method, the doctor's belief in the method, and the patient's and the doctor's belief in each other. If all these factors work optimally, even procedures based on ridiculous theories can produce real cures. If they do not interact productively, even the most scientific and rational treatments may fail to cure.

Dr Weil, a graduate of Harvard Medical School and a research associate there in ethnopharmacology as well as a lecturer in the Division of Social Perspectives in Medicine at the University of Arizona College of Medicine, has a long standing interest in psychoactive drugs, herbal medicines, and mind-body relationships. In this book he outlines his own philosophy of health and wholeness and describes a variety of healing systems such as allopathic medicine, homeopathy, osteopathy, chiropractic, naturopathy, Chinese medicine, shamanism, psychic healing, holistic medicine, and quackery. He also provides an interesting discussion of the mind's power over the healing process and concludes with some interesting thoughts regarding future directions in healing.

The book is well-organized, clearly written, and a pleasure to read. It represents a valuable contribution to our understanding of the social-psychological dimensions of the healing art, an area underemphasized in the training of physicians.

This book is akin to Jerome Frank's *Persuasion and Healing* in its emphasis on the importance of placebo responses, to Ivan Illich's *Medical Nemesis* in its criticism of modern scientific medicine for its dangerous aspects and the dependency it fosters in patients, to Robert Mendelsohn's *Confessions of a Medical Heretic* in its outlining of the limitations of modern scientific medicine, and to Norman Cousins' *The Healing Heart* in its description of the power the human mind can have over organic disease. It should be required reading for all practitioners of the healing art.

HENRY B. PERRY, III, MD
Mountain Medical Center
Clyde, NC 28721

Milton Rosenberg, MD, 1923–1985

An Indefatigable Spirit

Milton Rosenberg, MD, Executive Vice-President of the Medical Society of the State of New York, died suddenly on March 1 while at work. Dr Rosenberg served as president of MSSNY in 1981–1982. Prior to assuming the position of executive vice-president in January, 1984, he had practiced obstetrics and gynecology in Patchogue, Long Island for 25 years, during part of which he had served as director of the Department of Obstetrics and Gynecology at Brookhaven Memorial Hospital. He was an assistant clinical professor of obstetrics and gynecology at the State University of New York at Stony Brook School of Medicine, and consultant at Mather Memorial Hospital, Port Jefferson, and Central Suffolk Hospital, Riverhead.

Dr Rosenberg was born in Brooklyn. Originally intending a career in agriculture, he switched to a pre-medical course and received his medical degree from the University of Wisconsin in 1948. He served an internship at Morrisiania City Hospital, New York, followed by a residency in obstetrics and gynecology at that institution and at French Hospital, New York. Board certified in obstetrics and gynecology, he was a Fellow of the American College of Obstetrics and Gynecology and a Fellow of the American College of Surgeons. He served as president of the Suffolk County Obstetrics and Gynecology Society, the Suffolk Academy of Medicine, and, for an unprecedented two terms, the Suffolk County Medical Society.

In 1983 he was named Doctor of the Year by the Suffolk County Medical Society. Among his positions at MSSNY, in addition to that of president, were those of vice-president, member of the Council, chairman of the Commission on Insurance and Membership Benefits, delegate to the American Medical Association, and board member of the Medical Liability Mutual Insurance Company.

The extent of Milton Rosenberg's contribution to the Medical Society of the State of New York and to the entire medical profession is measured far beyond his brief tenure as executive vice-president. In just 15 months at the helm, he led the Society toward greater achievements, including the development of a model peer review organization, progress toward the establishment of a rehabilitation program for impaired physicians, and stepped-up political activism in Albany. His daily dialogue with legislative and governmental leaders brought enhanced respect and admiration for the Society.

Dr Rosenberg's love of the practice of medicine and zest for debate inspired enthusiasm among colleagues and staff alike. During the past several years he played a catalytic role in seeking a solution to the runaway problem of medical liability litigation. With positive, forceful, and convincing arguments and an endearing sense of humor, he flourished in committee and Council discussions.

Dr Rosenberg showed the same compassion and concern for the staff of MSSNY as for his patients and family. He is survived by his beloved and devoted wife Estelle and daughters Dori, Beth, and Harriet.

Contributions in memory of Dr Rosenberg may be sent to Temple Beth El, 45 Oak Street, Patchogue, New York 11772; Patchogue Chapter of Hadassah, 200 North Prospect, Patchogue, New York 11772; or Brookhaven Memorial Hospital, 101 Hospital Road, Patchogue, New York 11772.

AB

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

William Brewster Allan, MD, New York. Died July 31, 1984; age 86. Dr Allan was a 1925 graduate of the New York University College of Medicine. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Benjamin J. Alperin, MD, Rockville Centre. Died November 7, 1984; age 76. He was a 1934 graduate of the Queens University Faculty of Medicine. Dr Alperin was a Diplomate of the American Board of Ophthalmology, and a member of the Academy of Medicine, the American Academy of Ophthalmology and Otolaryngology, the Nassau County Medical Society and the Medical Society of the State of New York.

William Benjamin Birnkrant, MD, New York. Died April 20, 1984; age 76. He was a 1930 alumnus of the State University of New York Downstate Medical Center. Dr Birnkrant was a Diplomate of the American Board of Internal Medicine, and a member of the American Public Health Association, the New York County Medical Society and the Medical Society of the State of New York.

Remo Romolo Cerulli, MD, New York. Died November 13, 1984; age 77. Dr Cerulli was a 1940 alumnus of Università degli Studi di Roma, Facoltà di Medicina e Chirurgia. He was a Fellow of the American Psychiatric Association, and a member of the New York County Medical Society and the Medical Society of the State of New York.

Tzu Pei Chou, MD, Stamford, Connecticut. Died December 6, 1984; age 89. Dr Chou was a 1922 graduate of Hsiang Ya Medical College, Changsha. He was a Qualified Fellow of the International College of Surgeons, and a member of the New York County Medical Society and the Medical Society of the State of New York.

R. William Curth, MD, Bronx. Died November 3, 1984; age 85. He was a 1924 graduate of Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin, Prussia. Dr Curth was a Diplomate of the American Board of Dermatology, Inc. His memberships included the Academy of Medicine, the New York State Dermatological Society, the American Academy of Dermatology, the New York County Medical Society and the Medical Society of the State of New York.

Harry Ertel, MD, Flushing. Died November 20, 1984; age 73. He was a 1937 graduate of Hahnemann Medical College and Hospital of Philadelphia. Dr Ertel was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Emil Albert Falk, MD, New York. Died December 18, 1984; age 86. Dr Falk was a 1925 alumnus of Harvard Medical School. He was a member of the Academy of Medicine, the Endocrine Society, the New York County Medical Society and the Medical Society of the State of New York.

Harold R. Feinstein, MD, Elmhurst. Died November 23, 1984; age 72. Dr Feinstein was a 1938 alumnus of the New York University College of Medicine. He was a member of the American Rheumatism Association, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Benjamin H. Finklestein, MD, Newburgh. Died November 7, 1984; age 81. Dr Finklestein was a 1928 alumnus of New York Medical College, Flower and Fifth Avenue Hospitals. He was a member of the Medical Society of the County of Orange and the Medical Society of the State of New York.

Morris Fishman, MD, Flushing. Died November 14, 1984; age 75. Dr Fishman was a 1935 alumnus of Universität Zurich Medizinische Fakultät, Zurich. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Richard P. Giliberty, MD, Great Neck. Died December 15, 1984; age 72. Dr Giliberty was a 1939 graduate of Hahnemann Medical College and Hospital of Philadelphia. He was a Qualified Fellow of the International College of Surgeons, and a member of the Academy of Medicine, the Nassau County Medical Society and the Medical Society of the State of New York.

K. Eileen Hite, MD, New York. Died November 30, 1984; age 70. She was an alumna of the University of Chicago School of Medicine. Dr Hite was a member of the American Psychiatric Association, the Academy of Psychoanalysis, the New York County Medical Society and the Medical Society of the State of New York.

Rene Houri, MD, Staten Island. Died November 1, 1984; age 59. He was a 1956 alumnus of Université de Paris Faculté de Médecine. Dr Houri was a Diplomate of the American Board of Radiology and the American Board of Nuclear Medicine. He was a member of the Richmond County Medical Society and the Medical Society of the State of New York.

Donald S. James, MD, New York. Died October 4, 1984; age 72. He was a 1936 graduate of New York Medical College, Flower and Fifth Avenue Hospitals. Dr

James was a Fellow of the American College of Gastroenterology, a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the Academy of Medicine, the New York Gastroenterological Society, the Aerospace Medical Association, the American Occupational Medical Association, the New York County Medical Society and the Medical Society of the State of New York.

Leo Kleban, MD, Rego Park. Died December 4, 1984; age 73. He was a 1935 graduate of the New York University College of Medicine. Dr Kleban was a member of the American Academy of Family Physicians, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Malvina Weinstock Kremer, MD, New York. Died November 23, 1984; age 77. Dr Kremer was a 1941 alumna of the New York University College of Medicine. She was a Diplomate of the American Board of Psychiatry and Neurology, and a member of the American Psychiatric Association, the Academy of Psychoanalysis, the New York County Medical Society and the Medical Society of the State of New York.

Stuart E. Krohn, MD, Utica. Died November 2, 1984; age 82. He was a 1930 alumnus of the McGill University Faculty of Medicine, Montreal. Dr Krohn was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the Medical Society of the County of Oneida and the Medical Society of the State of New York.

William Kropf, MD, Halandale, Florida. Died December 12, 1984; age 78. Dr Kropf was a 1933 graduate of New York Medical College, Flower and Fifth Avenue Hospitals. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

John Lo Verme, MD, New York. Died October 22, 1984; age 85. He was a 1925 graduate of the Boston University School of Medicine. Dr Lo Verme was a Qualified Fellow of the International College of Surgeons, and a member of the Pan American Medical Association, the New York County Medical Society and the Medical Society of the State of New York.

Bruce Davenport Lott, MD, Binghamton. Died November 13, 1984; age 70. Dr Lott was a 1941 graduate of the McGill University Faculty of Medicine, Montreal. He was a Diplomate of the American Board of Colon and Rectal Surgery. His memberships included the Academy of Medicine, the American Society of Colon and Rectal Surgeons, the Pan American Medical Association, the Broome County Medical Society and the Medical Society of the State of New York.

George Emanuel Lynch, MD, Hilton. Died November 25, 1984; age 76. He was a 1931 graduate of Queens University Faculty of Medicine. Dr Lynch was a member of the American Academy of Family Physicians, the Medical Society of the County of Livingston and the Medical Society of the State of New York.

Martin Gary Marmon, MD, Tarrytown. Died December 2, 1984; age 66. He was a 1943 graduate of the State University of New York Downstate Medical Center. Dr Marmon was a Diplomate of the American Board of Dermatology, Inc. His memberships included the New York State Dermatological Society, the American Academy of Dermatology, the Society for Investigative Dermatology, the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Ethel C. Morris-Russell, MD, Philadelphia, Pennsylvania. Died November 26, 1984; age 99. Dr Morris-Russell was a 1919 graduate of Womans Medical College of Pennslvania. She was a member of the Medical Society of the County of Ulster and the Medical Society of the State of New York.

Joseph J. Panzarella, MD, Setauket. Died December 9, 1984; age 65. He was a 1945 alumnus of the State University of New York Downstate Medical Center. Dr Panzarella was a Diplomate of the American Board of Physical Medicine and Rehabilitation. His memberships included the American Academy of Compensation Medicine, Inc, the American Academy of Physical Medicine and Rehabilitation, the American Geriatrics Society, the Pan American Medical Association, the Suffolk County Medical Society and the Medical Society of the State of New York.

Oscar Pelzman, MD, Patchogue. Died November 29, 1984; age 73. Dr Pelzman was a 1936 graduate of Medizinische Fakultät der Universität, Wien. He was a Diplomate of the American Board of Psychiatry and Neurology, and a member of the American Psychiatric Association, the Suffolk County Medical Society and the Medical Society of the State of New York.

Joseph Aaron Pincus, MD, New York. Died December 15, 1984; age 73. He was a 1937 graduate of Universität Basel Medizinische Fakultät, Basel. Dr Pincus was a Fellow of the American Academy of Family Physicians and a Diplomate of the American Board of Family Practice. His memberships included the Academy of Medicine, the American Rheumatism Association, the American Occupational Medical Association, the American Geriatrics Society, the American Society of Internal Medicine, the Aerospace Medical Association, the New York County Medical Society and the Medical Society of the State of New York.

George Francis Price, MD, Brightwaters. Died December 2, 1984; age 86. Dr Price was a 1924 graduate of the University of Michigan Medical School. He was a Diplomate of the American Board of Dermatology, Inc, and a member of the New York State Dermatological Society, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Clifford H. J. Reason, MD, Newark. Died November 29, 1984; age 58. He was a 1951 alumnus of the University of Western Ontario Faculty of Medicine, London. Dr Reason was a Fellow of the American College of Surgeons, a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Pathology. He was a member of the Medical Society of the County of Wayne and the Medical Society of the State of New York.

George Aloysius Reich, MD, Schenectady. Died December 8, 1984; age 76. Dr Reich was a 1934 graduate of the State University of New York Downstate Medical Center. He was a member of the Medical Society of the County of Schenectady and the Medical Society of the State of New York.

Alfred Abraham Richman, MD, New York. Died December 8, 1984; age 92. He was a 1914 alumnus of New York Medical College, Flower and Fifth Avenue Hospitals. Dr Richman was a Fellow of the American College of Chest

Physicians and a Qualified Fellow of the International College of Surgeons. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Alan Burns Rothballer, MD, Valhalla. Died December 11, 1984; age 58. He was a 1948 alumnus of the University of Pennsylvania School of Medicine. Dr Rothballer was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Neurological Surgery. His memberships included the Academy of Medicine, the New York Society of Neurosurgery, the American Association of Neurological Surgeons, the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Herman B. Rubler, MD, Palm Beach, Florida. Died December 18, 1984; age 77. He was a 1932 alumnus of the New York University College of Medicine. Dr Rubler was a Fellow of the American College of Angiology and the American College of Cardiology. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Joseph Rutenberg, MD, New York. Died September 24, 1984; age 72. Dr Rutenberg was a 1948 alumnus of Université de Genève Faculté de Médecine, Genève. He was a member of the Clinical Society of the New York Diabetes Association, the American Academy of Family Physicians, the New York County Medical Society and the Medical Society of the State of New York.

Mardoqueo I. Salomon, MD, New York. Died November 18, 1984; age 78. He was a 1933 graduate of Université de Genève Faculté de Médecine. Dr Salomon was a Fellow of the American College of Cardiology, the American College of Chest Physicians, the American College of Gastroenterology and the American College of Angiology. He was a member of the Academy of Medicine, the New York Academy of Gastroenterology, the New York State Society of Internal Medicine, the International Society of Internal Medicine, the Bronx County Medical Society and the Medical Society of the State of New York.

Marilyn T. Schittone, MD, Stony Brook. Died December 7, 1984; age 69. She was a 1943 alumna of the New York University College of Medicine. Dr Schittone was a Diplomate of the American Board of Internal Medicine, and a member of the International Society of Hematology, the American Medical Women's Association, the Bronx County Medical Society and the Medical Society of the State of New York.

A. Albert Schwartz, MD, Brooklyn. Died June 11, 1984; age 70. He was a 1941 graduate of Université de Lausanne Faculté de Médecine, Lausanne. Dr Schwartz was a Fellow of the American College of Surgeons and the American College of Obstetricians and Gynecologists, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Harold T. Schweitzer, MD, Buffalo. Died November 21, 1984; age 76. He was a 1931 graduate of the University of Buffalo School of Medicine, a Fellow of the American College of Physicians, and a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Calixto C. Singson, MD, Elmhurst. Died August 8, 1983; age 46. Dr Singson was a 1961 alumnus of the University of Santo Tomas Faculty of Medicine and Surgery, Manila. He was a Diplomate of the American Board of Pediatrics and a member of the New York County Medical Society and the Medical Society of the State of New York.

Edward Stebbins, MD, Niagara Falls. Died November 18, 1984; age 95. He was a 1918 alumnus of the University of Michigan Homeopathic Medical School. Dr Stebbins was a member of the Medical Society of the County of Niagara and the Medical Society of the State of New York.

Grete Stohr, MD, New York. Died November 16, 1984; age 91. She was a 1919 alumna of Medizinische Fakultät der Universität, Wien. Dr Stohr was a Diplomate of the American Board of Pathology, and a member of the Academy of Medicine, the New York Pathological Society, the New York County Medical Society and the Medical Society of the State of New York.

Joseph Samuel Stovin, MD, New York. Died March 23, 1984; age 89. He was a 1922 graduate of the University of Maryland School of Medicine. He was a Diplomate of the American Board of Otolaryngology, and a member of the American Academy of Otolaryngology, the American Academy of Facial Plastic and Reconstructive Surgery, the New York County Medical Society and the Medical Society of the State of New York.

Bronislawa Szuldberg, MD, Piedmont, California. Died September 30, 1984; age 93. Dr Szuldberg was a graduate of Uniwersytet Warszawski Akademie Medyczna, Warsawa, and a member of the New York County Medical Society and the Medical Society of the State of New York.

Herminio Cruze Tiangco, MD, New York. Died November 4, 1984; age 59. He was a 1953 alumnus of the Manila Central University College of Medicine, Manila. Dr Tiangco was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the New York State Surgical Society, the American Society of Abdominal Surgeons, the New York County Medical Society and the Medical Society of the State of New York.

Solomon J. Turel, MD, Miami Beach, Florida. Died October 24, 1984; age 90. Dr Turel was a 1917 alumnus of Dalhousie University Faculty of Medicine, Halifax and a member of the New York County Medical Society and the Medical Society of the State of New York.

Philip S. Van Orden, MD, Albany. Died November 26, 1984; age 86. Dr Van Orden was a 1926 graduate of Albany Medical College of Union University. He was a member of the Medical Society of the County of Albany and the Medical Society of the State of New York.

Orin A. Wahl, MD, Newburgh. Died October 12, 1984; age 61. He was a 1952 graduate of the New York University College of Medicine. Dr Wahl was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the Medical Society of the County of Orange and the Medical Society of the State of New York.

Edward J. Werdein, MD, Rochester. Died November 30, 1984; age 58. Dr Werdein was a 1952 alumnus of the Georgetown University School of Medicine. He was a member of the Academy of Medicine, the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Medical Society of the State of New York

Annual Meeting of the
HOUSE OF DELEGATES
June 6-9, 1985

ANNUAL CME ASSEMBLY
HEALTH CARE TODAY
June 7-9, 1985

The New York Hilton
New York City

HIGHLIGHTS. . . . .
30 Sessions • Panel Discussions
- Meet the Professors
- Technical Exhibits
- President's Dinner Dance

CLIP & MAIL

Director of Front Office Operations
The New York Hilton
1335 Avenue of the Americas
New York, N.Y. 10019

Phone: 212-586-7000

for hotel use only

NAME ______________________ TITLE ____________

FIRM ______________________ PHONE ____________

STREET ______________________

CITY/STATE/ZIP ______________________

REMARKS ______________________

(please print)

NOTE: Reservations must be received no later than May 16, 1985 and will be held only until 6 p.m. on day of arrival unless later hour is specified.

please print

ARRIVAL DATE ______________________

☐ 2-4 p.m. ☐ 4-6 p.m. ☐ 6-8 p.m. ☐ after 8 p.m.

DEPARTURE DATE ______________________

(checkout time is 1 p.m.)

HAVE A CREDIT CARD? You can register at our QUICK CHECK desk. If you wish to take advantage of this credit card PAYMENT GUARANTEED reservation, please complete the information below.

☐ AMERICAN EXPRESS
☐ DINNERS CLUB
☐ CARTE BLANCHE

Expiration Date ____________

____
Credit Card No.

Signature ______________________

MEDICAL SOCIETY OF THE STATE OF NEW YORK
Annual Meeting of the House of Delegates
June 6-9, 1985
Annual CME Assembly
June 7-9, 1985

Circle preferred rate:

Singles:	$120	$135	$145
Double/Twin:	$145	$160	$170
Executive Tower:	$190, Single/Double		
Suites: (P&1):	$315	$350	
Suites: (P&2):	$425	$460	

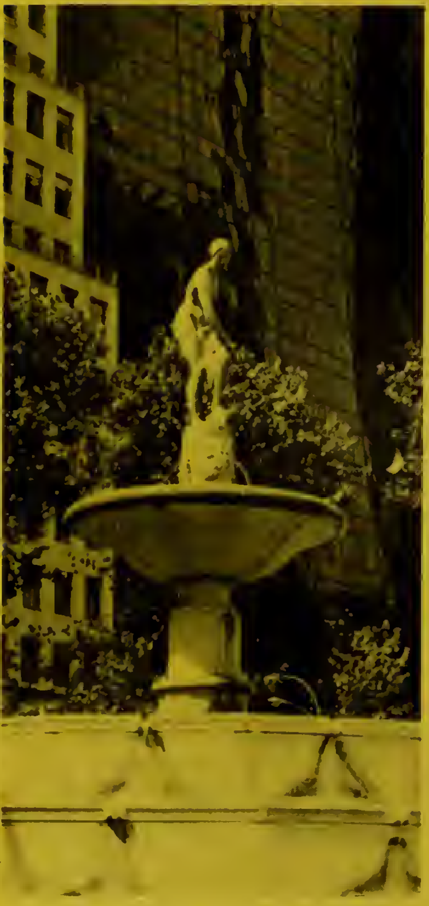
Rates subject to 8¼% N.Y. State sales tax and $2.00 per room per night N.Y.C. occupancy tax. (If room at the rate requested is unavailable, one at the nearest available rate will be reserved.)

General Information

The Medical Society of the State of New York's 1985 Annual CME Assembly sessions and exhibits are open to all physicians, their guests, and the allied professions. The two and one-half days of meetings will provide the individual physician with a possible 18 credits out of a total of 93 Category 1, CME credit hours being offered. Over 135 outstanding speakers, including a number from out-of-state, will present papers at 30 sessions.

On Friday, June 7th and Sunday, June 9th, five sessions of Meet the Professor will be held simultaneously. These two-hour sessions will include a continental breakfast.

The theme of the CME sessions will be *"Health Care Today"*. Some highlights of this year's sessions are, the Developments in the Relationship between Diet and Cancer; Update on Pacemakers; Computer Applications in Clinical Medicine; the Physician as a Witness; and today's concepts of Chest Diseases diagnosis and management. Our program will offer the clinician new approaches in diagnosis and treatment of diseases in a wide range of medical specialties. Don't miss this opportunity to gain Category 1 credits over a weekend when you are free from office hours. This year there is no registration fee for member physicians which makes it an economical way to brush up on your skills. Professional representatives from more than 70 companies will be in the Rhinelander Gallery Exhibit Hall to discuss their products and services. Technical Exhibits will include drug products, clinical laboratories, office equipment, computer services, educational and financial services.



COURTESY N.Y CONVENTION & VISITORS BUREAU

PULITZER FOUNTAIN AT GRAND ARMY PLAZA

For the first time since 1979, the CME Assembly will be combined with the Annual Meeting of the House of Delegates, and the Auxiliary to the Medical Society.

SPECIAL EVENTS

Thursday, June 6: The Annual Dinner Dance will be held in the Trianon Ballroom of the New York Hilton, in honor of Allison B. Landolt, M.D., President, preceded by a reception in the Mercury Ballroom at 7:00 p.m. All attendees are welcome. Tickets should be ordered in advance from the Medical Society.

Friday, June 7 and Saturday June 8: A reception for physicians, the allied professions, and exhibitors will be held on both days in the Exhibit Hall, between 5:00 p.m. and 6:00 p.m., compliments of the Medical Society of the State of New York.

HOTEL RESERVATIONS

Physicians must make their own reservations with the New York Hilton no later than May 16, 1985. If making reservations by telephone, please mention that you are attending the Medical Society Convention for billing at the special rate.

REGISTRATION

CME Registration will take place in the Rhinelander Gallery Exhibit Hall, located on the second floor of the hotel and accessible by escalators from the lobby. Registration will take place at the following hours:

Friday, June 7th–
7:30 a.m.-5:00 p.m.
Saturday, June 8th–
8:00 a.m.-5:00 p.m.
Sunday, June 9th–
7:30 a.m.-12:30 p.m.

House of Delegates Registration will take place on the third floor, West Promenade, at the following hours: Wednesday, June 5 from 12 noon-7:30 p.m.; Thursday, June 6 from 8:30 a.m.-5:30 p.m.; Friday, June 7 from 8:30 a.m.-5:30 p.m.; Saturday, June 8 from 8:30 a.m.-5:30 p.m.; and Sunday, June 9 from 8:30 a.m.-12 noon.

CME MEETING HOURS

Friday, June 7th–
Meet the Professor–
8:00 a.m.-10:00 a.m.
All other sessions–
10:00 a.m.-1:00 p.m.
2:00 p.m.-5:00 p.m.

Saturday, June 8th–
All sessions–
10:00 a.m.-1:00 p.m.
2:00 p.m.-5:00 p.m.

Sunday, June 9th–
Meet the Professor–
8:00 a.m.-10:00 a.m.
All other sessions–
10:00 a.m.-1:00 p.m.

1985
PRELIMINARY PROGRAM
Annual CME Assembly
June 7-9, 1985

Annual Meeting of the
House of Delegates
June 6-9, 1985

The New York Hilton
New York City

INDEX

Hotel Reservation Form 19A
General Information 20A
Scientific Program Committee 23A
Scientific Program 24A
Technical Exhibits 39A
Advance Registration 41A
Meet the Professor 42A

REGISTRATION & MEETING HOURS

CME Assembly

Registration is open to MSSNY members, spouses, invited guests, non-member physicians, and members of the allied professions. There is no registration fee for MSSNY members, residents/interns, speakers, medical students, medical assistants, and spouses. There is a registration fee for non-member physicians—$50; and allied professions—$10.

An advance registration form will be found in this section. For those who register in advance, your identification badge will be held at the CME Registration Desk located in the Rhinelander Gallery Exhibit Hall on the second floor of the hotel. For those who have not registered in advance, registration will take place on Friday, June 7, from 7:30 a.m. to 5:00 p.m.; Saturday, June 8, from 8:00 a.m. to 5:00 p.m.; and on Sunday, June 9 from 7:30 a.m. to 12:30 p.m.

Identification badges are required for admittance to all CME meetings and exhibits. All registrants are required to show identification at the registration desk before a badge is issued.

CME meeting hours will be on Friday, June 7—Meet the Professor breakfast meetings from 8:00 a.m. to 10:00 a.m.; all other sessions from 10:00 a.m. to 1:00 p.m., and from 2:00 p.m. to 5:00 p.m. Saturday, June 8 from 10:00 a.m. to 1:00 p.m., and from 2:00 p.m. to 5:00 p.m. Sunday, June 9—Meet the Professor breakfast meetings from 8:00 a.m. to 10:00 a.m.; all other sessions from 10:00 a.m. to 1:00 p.m.

Delegates

Registration for delegates will take place on the third floor, West Promenade, as follows: Wednesday, June 5 from 12 Noon to 7:30 p.m.; Thursday, June 6 from 8:30 a.m. to 5:30 p.m.; Friday, June 7 from 8:30 a.m. to 5:30 p.m.; Saturday, June 8 from 8:30 a.m. to 5:30 p.m.; and Sunday, June 9 from 8:30 a.m. to 12 Noon.

The 179th Annual Meeting of the House of Delegates will be called to order at 1:00 p.m. on Thursday, June 6, 1985 in the East Ballroom of the New York Hilton. The schedule follows: Thursday, June 6 from 1:00 p.m. to 5:00 p.m.; Saturday, June 8 from 9:00 a.m. to 5:30 p.m. (and 8:00 p.m. to 10:00 p.m. if necessary); Sunday, June 9 from 9:00 a.m. to 1:00 p.m.

AUXILIARY TO MSSNY

The Auxiliary House of Delegates meeting will take place on Friday, June 7 from 8:30 a.m. to 6:00 p.m., and on Saturday, June 8 from 8:00 a.m. to 12 Noon in the Trianon Ballroom of the New York Hilton.

CME CREDITS

The Medical Society of the State of New York designates the continuing medical education activities included in this CME Assembly as meeting the criteria for the designated number of credit hours in Category 1 of the Physician Recognition Award of the AMA, and/or MSSNY's Method #6.

Each program is acceptable for prescribed or elective credits by the American Academy of Family Physicians (AAFP) as designated. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. The Medical Society's CME credit forms will be issued at the entrance to each meeting room at the opening of each session.

EXHIBITS

Technical exhibits will be located in the Rhinelander Gallery on the second floor of the New York Hilton. Exhibits will be on view Friday, June 7 from 9:00 a.m. to 10:30 a.m., and from 1:00 p.m. to 6:00 p.m.; Saturday, June 8 from 9:00 a.m. to 10:30 a.m., and from 1:00 p.m. to 6:00 p.m.; and Sunday, June 9 from 9:00 a.m. to 1:00 p.m.

PHYSICIAN MESSAGE CENTER

Messages for the physicians attending the CME sessions may be left at the MSSNY Information Desk located in the Exhibit Hall on the second floor. Messages for Delegates may be left at the Delegates Information Desk on the third floor, West Promenade.

COFFEE BREAKS

Coffee breaks sponsored by the Medical Society of the State of New York, will be held in the Exhibit Hall on the second floor daily from 9:00 a.m. to 10:00 a.m. All registered attendees are welcome.

DINNER DANCE

On Thursday, June 6, the Annual Dinner Dance will be held in the Trianon Ballroom, preceded by a reception in the Mercury Ballroom at 7:00 p.m.. Tickets should be ordered in advance from the Medical Society of the State of New York. Subscription is $70.00 per person. See the order form in this issue.

RECEPTIONS

A reception, sponsored by the MSSNY, will be held on Friday, June 7 and Saturday, June 8 from 5:00 p.m. to 6:00 p.m. in the Rhinelander Gallery Exhibit Hall located on the second floor of the hotel. All physicians, their spouses, the allied professions and exhibitors are welcome.

1985 ANNUAL CME ASSEMBLY

HEALTH CARE TODAY

Medical Society of the State of New York
New York Hilton, New York City

FRIDAY, JUNE 7

8:00 a.m. - 10:00 a.m.	Meet the Professor Breakfast Meetings (5)
10:00 a.m. - 1:00 p.m.	Cardiovascular Diseases, *Beekman Parlor* Pediatrics/Dermatology, *Sutton Parlor North* Obstetrics/Gynecology, *Sutton Parlor South* Physical Medicine & Rehabilitation, *Sutton Parlor Center* Neurosurgery, *Regent Parlor*
2:00 p.m. - 5:00 p.m.	Neurology, *Sutton Parlor Center* Medical Legal & Workers´ Compensation/Occupational Medicine, *Beekman Parlor* Cancer Update, *Sutton Parlor North* Chest Diseases, *Sutton Parlor South* Computers in Medicine, *Regent Parlor*

SATURDAY, JUNE 8

10:00 a.m. - 1:00 p.m.	Ophthalmology (All day), *Sutton Parlor North* Urology/Pathology, *Sutton Parlor Center* Internal Medicine (All day), *Beekman Parlor* General Surgery/Orthopedic Surgery/Hand Surgery/Gastroenterology Anesthesiology/Radiology, *Sutton Parlor South* Psychiatry/Alcoholism (All day), *Regent Parlor*
2:00 p.m. - 5:00 p.m.	Ophthalmology, *Sutton Parlor North* Otolaryngology—Head and Neck Surgery, *Sutton Parlor Center* Emergency Medicine, *Sutton Parlor South* Internal Medicine, *Beekman Parlor* Psychiatry/Alcoholism, *Regent Parlor*

SUNDAY, JUNE 9

8:00 a.m. - 10:00 a.m.	Meet the Professor Breakfast Meetings (5)
10:00 a.m. - 1:00 p.m.	Allergy & Immunology, *Beekman Parlor* Women´s Medical Society of New York State/Family Practice, *Sutton Parlor Center* The Physician as a Witness, *Sutton Parlor North*

◆

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis.

◆

Technical Exhibits

1985 ANNUAL CME ASSEMBLY

SCIENTIFIC PROGRAM COMMITTEE

Stephen Nordlicht, M.D., New York City, Chairman
Martin Lipkin, M.D., New York City, Vice Chairman
George J. Lawrence, Jr., M.D., Lake Success
Director, Division of Scientific Activities

ASSOCIATE CHAIRMEN

Thomas S. Bumbalo, M.D., Alden
Samuel Cytryn, M.D., Levittown
John Davis, M.D., Cooperstown
David Hirsh, M.D., Scarsdale
Joseph J. Kaufman, M.D., Newark
Marilyn M. S. Kritchman, M.D., Great Neck
A. W. Martin Marino, Jr., M.D., Brooklyn
Maxwell Spring, M.D., Bronx

MEMBERS REPRESENTING SPECIALTY SOCIETIES

Alcoholism

Anne Geller, M.D., Brooklyn
American Medical Society on Alcoholism—New York Chapter

Allergy & Immunology

Renata Frenkel, M.D., New York City

Anesthesiology

Kenneth Freese, M.D., East Meadow
New York State Society of Anesthesiologists

Cardiovascular Diseases

Mohammad Zahir, M.D., Brooklyn
New York Cardiological Society

Chest Diseases

John Vance, M.D., Buffalo
American College of Chest Physicians

Dermatology

Franklin Glickman, M.D., Brooklyn
New York State Society of Dermatology

Emergency Medicine

Lorraine M. Giordano, M.D., Jamaica
New York Chapter of American College of Emergency Physicians

Family and General Practice

Bruce E. Baker, M.D., Leroy
New York State Academy of Family Physicians

Hand Surgery

Thomas Palmieri, M.D., Brookville
New York Society for Surgery of the Hand

Internal Medicine

Sidney Weinstein, M.D., Rochester
New York State Society of Internal Medicine

Medical Legal & Workers' Compensation Matters

Gottfried F. H. Lehmann, M.D., Jamaica

Neurology

Richard Satran, M.D., Rochester

Neurosurgery

James L. Budny, M.D., Buffalo
New York State Neurosurgical Society

Obstetrics and Gynecology

John C. Boyce, M.D., Sands Point
American College of Obstetricians and Gynecologists, District II

Ophthalmology

Alfred Cossari, M.D., Port Jefferson Station
New York State Ophthalmological Society

Orthopedic Surgery

David Hirsh, M.D., Scarsdale
New York State Society of Orthopedic Surgeons

Otolaryngology

Steven Parnes, M.D., Albany
New York State Society of Otolaryngology—Head and Neck Surgery

Pathology

Edward C. Zaino, M.D., Garden City
New York State Society of Pathologists

Pediatrics

Marvin Blumberg, M.D., Jamaica
American Academy of Pediatrics

Physical Medicine and Rehabilitation

Samuel Sverdlik, M.D., New York City
New York Society of Physical Medicine and Rehabilitation

Psychiatry

Richard Drooz, M.D., New York City
New York State Psychiatric Association

Radiology

Rita Girolamo, M.D., Valhalla
New York State Chapter of the American College of Radiology

Surgery

Murry Friedman, M.D., New Hyde Park
New York State Society of Surgeons

Urology

Pellegrino Tozzo, M.D., New Rochelle
New York State Urological Society

Women's Medical Society of New York State

Jeanne Smith, M.D., New York City

FRIDAY, JUNE 7, 1985

MEET THE PROFESSOR

Planned and Organized by Maxwell Spring, M.D., Associate Chairman, Scientific Program Committee
Clinical Associate Professor of Medicine, New Jersey Medical School

8:00 A.M.–8:30 A.M. Continental Breakfast
8:30 A.M.–10:00 A.M. Meeting

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 1½ Hours, Category 1,
AAFP, 1½ Hours, Prescribed

An opportunity to meet informally and discuss clinical problems on a one-to-one basis with experts in your field.

Registration Fee: $35.00 Per Session
(*includes continental breakfast*)

8:30 A.M. **Update on Thyroid and Pituitary Diseases** (Room 529)

Charles Hollander, M.D., New York City

Professor of Medicine and Chief of Endocrinology,
New York University School of Medicine

Objective: To provide a very brief review of the highlights of the revolutionary developments in our understanding of the physiology and pathophysiology of thyroid, pituitary and hypothalamic disorders and to show how these developments have dramatically improved the manner in which we test for these disorders.

8:30 A.M. **Hepatitis Update** (Room 534)

Carroll Leevy, M.D., Newark, New Jersey

Professor and Chairman, Department of Medicine,
New Jersey Medical School, College of Medicine
and Dentistry of New Jersey

Objective: Immunologic abnormalities are key factors in pathogenesis of Hepatitis due to infections, alcoholism, and drug induced toxicity. Prevention, diagnosis and treatment have been greatly facilitated by recent advances in genetics, availability of monoclonal antibodies and the use of recombinant D.N.A. (Deoxyribonucleic Acid) technology.

8:30 A.M. **Newer Approaches to the Management of Diabetes Mellitus** (Room 537)

Harold E. Lebovitz, M.D., Brooklyn

Chief, Endocrinology and Metabolism,
State University of New York Downstate Medical
Center

Objective: To provide an understanding of the pathophysiology of the two major types of diabetes; to use this information to design appropriate management strategies; to develop a rational set of goals for the treatment of patients with diabetes.

8:30 A.M. **Hypertension: Alternate Approaches to the Initial Treatment of Hypertension** (Room 540)

Harold D. Itskovitz, M.D., Valhalla

Professor of Medicine and Pharmacology,
New York Medical College

Objective: To familiarize the physician with new drugs used to treat hypertension; to stress changing concepts concerned with the etiology, work-up and management of hypertension.

8:30 A.M. **The Approach to the Severely Ill Patient With Pneumonia** (Room 543)

Donald Louria, M.D., Newark, New Jersey

Professor and Chairman,
Department of Preventive Medicine and Community
Health, New Jersey Medical School, College of
Medicine and Dentistry of New Jersey

Objective: To develop a programatic approach to the seriously ill patient with a putative infection. This will be done by a focus on differential diagnosis, risk factors, and a nine-point approach to therapy.

(continued)

Cardiovascular Diseases
Jointly Sponsored by the New York Cardiological Society

10:00 A.M.–1:00 P.M.
BEEKMAN PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

UPDATE ON PACEMAKERS

DORIS J. W. ESCHER, M.D., Bronx, *Moderator*

Professor of Medicine, Albert Einstein College of Medicine of Yeshiva University; Director, Emeritus and Senior Consultant, Cardiac Catheterization Lab, Montefiore Hospital and Medical Center

Objective: To provide up-to-date information about pacemakers.

10:00 A.M. **Choice of Pacemaker: Physiologic or Economic**

BERNARD H. BOAL, M.D., Flushing

Physician-in-Charge, Division of Cardiology, Booth Memorial Medical Center

10:30 A.M. **Follow-up: Techniques and Schedules**

LAWRENCE A. GOULD, M.D., Brooklyn

Chief, Division of Cardiology, The Methodist Hospital

11:00 A.M. **Indications for Pacemaker Implantations: Clinical and Regulatory Problems**

JOSEPH LAUTERSTEIN, M.D., Monticello

Chief, Cardiology; Director, Coronary Care Unit, Community General Hospital of Sullivan County; Assistant Attending Physician, St. Vincent's Hospital and Medical Center of New York

11:30 A.M. **Malfunction and Pseudo-Malfunction Misreading of ECG's (Electrocardiogram's) in Duel Chamber Pacing**

PETER T. KLEMENTOWICZ, M.D., Bronx

Instructor of Medicine, Albert Einstein College of Medicine of Yeshiva University; Attending Physician, Pace Maker Center, Moses Division, Montefiore Hospital and Medical Center

12:00 NOON–1:00 P.M.
Panel Discussion
The Above Speakers

Dermatology/Pediatrics
Joint Meeting With
New York State Society of Dermatology
and
New York State District II of the American Academy of Pediatrics

10:00 A.M.–1:00 P.M.
SUTTON PARLOR NORTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1: AAFP, 3 Hours, Prescribed

SKIN DISEASES IN CHILDREN AND ADOLESCENTS

FRANKLIN S. GLICKMAN, M.D., Brooklyn, *Moderator*

Clinical Professor of Dermatology, State University of New York Downstate Medical Center

and

MARVIN L. BLUMBERG, M.D., Jamaica, *Moderator*

Chairman, Department of Pediatrics, Jamaica Hospital; Associate Professor of Clinical Pediatrics, State University of New York at Stony Brook

Objective: To make the physician aware of the significance of skin pathology as a manifestation of disease in children, and to increase the effectiveness of the physician in diagnosing and treating these conditions. Particular attention will be paid to three areas: Skin disease in the neonate; life-threatening disorders; and sexually transmitted disorders in children and adolescents.

(continued)

10:00 A.M. **Critical Care: Pediatric Dermatology**

MARK E. GROSSMAN, M.D., New York City

Assistant Clinical Professor of Dermatology, Columbia-Presbyterian Medical Center

10:40 A.M. **Discussion**

10:45 A.M. **Neonatal Dermatology**

SAMUEL WEINBERG, M.D., New York City

Clinical Professor of Dermatology, New York University Medical Center; Chief, Pediatric Dermatology, Long Island Jewish Hillside Medical Center, New Hyde Park

11:25 A.M. **Discussion**

11:30 A.M. **Blisters and Pustules in the Newborn**

ALFRED T. LANE, M.D., Rochester

Assistant Professor of Medicine (Dermatology); Assistant Professor of Pediatrics, University of Rochester School of Medicine and Dentistry

12:10 P.M. **Discussion**

12:15 P.M. **Sexually Transmitted Disease in Children and Adolescents**

YEHUDI FELMAN, M.D., Brooklyn

Director, Bureau of Venereal Diseases, New York City Department of Health; Clinical Professor of Dermatology, State University of New York Downstate Medical Center

12:25 P.M.-1:00 P.M.—Discussion

Neurosurgery
Joint Meeting With
New York State Neurosurgical Society, Inc.

10:00 A.M.-1:00 P.M.
REAGENT PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Elective

CERVICAL SPINE: NEW DIAGNOSTIC AND THERAPEUTIC MODALITIES

JAMES L. BUDNY, M.D., Buffalo, *Moderator*

Assistant Clinical Professor of Neurosurgery, State University of New York at Buffalo

Objective: The three guest speakers will present topics of interest in reference to current concepts of cervical spine disease. It is hoped that through this series of lectures that the medical community as a whole will benefit from an increased understanding of the newer approaches in diagnosis and treatment of cervical spine disease.

10:00 A.M. **Neurodiagnostic Modalities Including CT (Computerized Tomography) Scanning, Magnetic Resonance Imaging, and Ultrasonography of the Cervical Spine**

FRANCIS GAMACHE, JR., M.D., New York City

Assistant Professor of Surgery (Neurosurgery), Cornell University Medical College

11:00 A.M. **Technical Advances in Treatment of Cervical Spinal Cord Tumors**

ROBERT DECKER, M.D., New Hyde Park

Clinical Associate Professor, Neurosurgery, State University of New York at Stony Brook; Instructor in Surgery, North Shore University Hospital, Manhasset

12:00 NOON-1:00 P.M. **Current Concepts and Recent Advances in the Treatment of Acute Cervical Spinal Cord Injuries**

FRED GEISLER, M.D., Baltimore Maryland

Assistant Professor, Neurosurgery; Medical Director, Neurotrauma, University of Maryland Medical Systems

(*continued*)

Obstetrics and Gynecology
Joint Meeting With
The American College of Obstetricians and Gynecologists, District II

10:00 A.M.-1:00 P.M.
SUTTON PARLOR SOUTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

THE PREMENSTRUAL TENSION SYNDROME

JOHN G. BOYCE, M.D., Sands Point, *Moderator*

Professor and Director, GYN Oncology Division, State University of New York Downstate Medical Center

Objective: To familiarize all physicians involved in the care of the female patient with the physiological and psychological changes occurring in some females with the hormonal changes at various stages of the menstrual cycle. Physicians will also be familiarized with recent advances in the medical management of this syndrome.

10:00 A.M. **Endocrinology of Premenstrual Syndrome**

VICTOR REYNIAK, M.D., New York City

Clinical Professor and Director, Reproductive Endocrinology, Department of Obstetrics-Gynecology and Reproductive Science, Mount Sinai Hospital

11:00 A.M. **Psychiatric Aspects of Premenstrual Syndrome**

EDGAR HENRIQUES, M.D., Albany

Professor and Vice Chairman, Department of Obstetrics and Gynecology; Head, Reproductive Endocrinology Department, Albany Medical College of Union University

10:30 A.M. **Premenstrual Syndrome from the Standpoint of the Female Physician**

VICKI SELTZER, M.D., Jamaica

Director, Obstetrics and Gynecology; Queens General Hospital; Associate Professor, Department of Obstetrics and Gynecology, Long Island Jewish Hillside Medical Center

11:30 A.M. **Premenstrual Syndrome—Treatment**

NATHAN KASE, M.D., New York City

Dean, Mount Sinai School of Medicine of the City University of New York

12:00 NOON-1:00 P.M. **Questions and Answer**

Physical Medicine & Rehabilitation
Joint Meeting With
New York State Society of Physical Medicine and Rehabilitation

10:00 A.M.—1:00 P.M.
SUTTON PARLOR CENTER

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

CHRONIC PAIN: MEDIATION; MODULATION; MEDICATION; REHABILITATION

Samuel S. Sverdlik, M.D., New York City, *Moderator*

Director, Department of Rehabilitation Medicine, St. Vincent's Hospital & Medical Center of New York; Clinical Professor, New York Medical College, Valhalla

Objective: Physicians attending this program wil be updated on the present status of pain mediation through prostaglandin modulation. Medical and physical medicine therapy will be discussed.

10:00 A.M. **Chronic Pain: Mediation**

JOHN C. MCGIFF, M.D., Valhalla

Professor and Chairman, Department of Pharmacology, New York Medical College

10:40 A.M. **Chronic Pain: Modulation**

ERIC SIMON, Ph.D., New York City

Professor of Psychiatry & Pharmacology, New York University School of Medicine

11:20 A.M. **Questions and Answers**

11:30 A.M. **Chronic Pain: Medication**

THOMAS KANTOR, M.D., New York City

Professor of Medicine, New York University School of Medicine

12:10 P.M. **Chronic Pain: Rehabilitation**

JOHN DOWNEY, M.D., New York City

Professor and Chairman, Department of Rehabilitation Medicine, Columbia University College of Physicians and Surgeons

12:50 P.M.-1:00 P.M. **Questions and Answers**

(continued)

GENERAL SESSION
Cancer Update 1985

2:00 P.M.–5:00 P.M.
SUTTON PARLOR NORTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Elective

DIETARY RECOMMENDATIONS IN CANCER PREVENTION

MARTIN LIPKIN, M.D., New York City, *Moderator*

Professor of Medicine, Cornell University Medical College, Memorial Sloan-Kettering Cancer Center; Vice Chairman, Scientific Program Committee, Medical Society of the State of New York.

Objective: The attendee at this program will be familiarized with recent developments in the relationship between diet and cancer. Newer uses of MRI (Magnetic Resonance Imaging) in cancer diagnosis and evaluation will also be covered.

2:00 P.M. **Diet in Cancer Prevention**

JOHN H. WEISBURGER, M.D., Ph.D., Valhalla

Vice President for Research; Director, Naylor Dana Institute for Disease Prevention, American Health Foundation

2:20 P.M. **Discussion**

2:30 P.M. **Newer Studies on Calcium in Cancer Prevention**

MR. HAROLD NEWMARK, New York City

Visiting Investigator, Memorial Sloan-Kettering Cancer Center

2:50 P.M. **Discussion**

3:00 P.M. **Diet and Nutritional Factors in Esophageal Cancer**

C. S. YANG, Ph.D., Newark, New Jersey

Professor of Biochemistry, College of Medicine and Dentistry of New Jersey

3:20 P.M. **Discussion**

3:30 P.M. **Cancer and the Fear of Death**

STEPHEN NORDLICHT, M.D., New York City

Consulting Psychiatrist, Memorial Sloan-Kettering Cancer Center

3:50 P.M. **Discussion**

NEWER ADVANCES IN MAGNETIC RESONANCE IMAGING (M.R.I.)

YAHYA M. BERKMEN, M.D., New York City, *Moderator*

Professor of Clinical Radiology, The New York Hospital-Cornell Medical Center

4:00 P.M. **MRI of Pelvic Malignancies**

ELIAS KAZAM, M.D., New York City

Associate Professor of Radiology,
The New York Hospital-Cornell Medical Center

4:20 P.M. **Discussion**

4:30 P.M. **Evaluation of Brain Metastases by MRI**

BENJAMIN LEE, M.D., New York City

Associate Professor of Radiology,
The New York Hospital-Cornell Medical Center

4:50 P.M.–5:00 P.M.—Discussion

Chest Diseases
Joint Meeting With
The New York State Chapter of the American College of Chest Physicians

2:00 P.M.–5:00 P.M.
SUTTON PARLOR SOUTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

WHAT IS NEW IN CHEST DISEASES IN 1985

JOHN VANCE, M.D., Buffalo, *Moderator*

Associate Clinical Professor of Medicine, State University of New York at Buffalo

Objective: To update the practitioner in today's concepts of important chest disease diagnosis and management.

(*continued*)

2:00 P.M. **Respiratory Muscle Dysfunction**

A. Ross Hill, M.D., Brooklyn

Assistant Professor of Medicine, State University of New York Downstate Medical Center

2:30 P.M. **Sleep Disorders: A Review**

Andras J. Vari, M.D., Buffalo

Clinical Assistant Professor of Medicine, State University of New York at Buffalo

3:00 P.M. ***The 34th Howard Lilienthal Memorial Lecture:* Pulmonary Emphysema—A Recent Update**

Ronald Crystal, M.D., Bethesda, Maryland

Director, National Heart, Lung and Blood Institute, National Institutes of Health, Bethesda, Maryland

4:00 P.M. **Management of Lung Cancer with Non-Mediastinal Lympth Node Involvement**

Nael Martini, M.D., New York City

Chief, Thoracic Surgery, Memorial Sloan Kettering Cancer Center; Professor of Surgery, Cornell University Medical College

4:30 P.M.-5:00 P.M. **Surgical and Adjuvant Therapy of Lung Cancer**

Martin F. McKneally, M.D., Albany

Associate Professor of Thoracic Surgery; Head, Division of Cardiothoracic Surgery, Albany Medical College of Union University

---

Computers in Medicine
Presented by the Committee on Information Technology in Medicine, Medical Society of the State of New York

2:00 P.M.-5:00 P.M.
REAGENT PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

COMPUTER APPLICATIONS IN CLINICAL MEDICINE

William A. Bauman, M.D., South Salem, *Moderator*

Chairman, Committee on Information Technology in Medicine, Medical Society of the State of New York

Objective: The session will inform physicians on the state-of-the-art of computer applications in clinical decision making. The attendee will learn of the objectives, methods and results of automated decision support systems through presentation of specific clinical applications dependent on information systems technology.

2:00 P.M. **Microcomputers in Medical Education**

Michael Anbar, M.D., Buffalo

Professor and Chairman, Department of Biophysical Sciences, State University of New York at Buffalo

2:20 P.M. **Diagnostic Prompting Program: The Example of Reconsider**

Stuart Nelson, M.D., Stony Brook

Assistant Professor of Clinical Medicine, Department of Medicine, Health Sciences Center, State University of New York at Stony Brook

2:40 P.M. **Microcomputer Based Patient Simulations**

William J. Hall , M.D., Rochester

Attending Physician, Rochester General Hospital

3:00 P.M. **MED CAPS: Medical Computer Assisted Problem Solving**

Olle Jane Sahler, M.D., Rochester

Associate Professor, Pediatrics and Psychiatry; Coordinator of Medical Student Education, University of Rochester School of Medicine and Dentistry

3:20 P.M. **Computer-Assisted Monitoring of Process and Outcome: Estimating Cost-Effectiveness of Diabetic Patient Education**

Paul J. Davis, M.D., Buffalo

Professor and Vice Chairman, Department of Medicine, Veterans Administration Medical Center

3:40 P.M. **Computers in Anesthesiology**

John Gage, M.D., Stony Brook

Assistant Professor of Anesthesiology, State University of New York at Stony Brook

4:00 P.M. **A Computerized Aid to Selection and Interpretation of Diagnostic Tests**

Robert J. Panzer, M.D., Rochester

Assistant Professor of Medicine, Department of Medicine, University of Rochester School of Medicine and Dentistry

4:20 P.M.-5:00 P.M.—**Discussion**

(*continued*)

Medical-Legal and Workers' Compensation Matters
Occupational Medicine

2:00 P.M.–5:00 P.M.
BEEKMAN PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Elective

CURRENT ISSUES IN OCCUPATIONAL HEALTH AND WORKERS' COMPENSATION

GOTTFRIED F. H. LEHMANN, M.D., Jamaica, *Moderator*

Consultant in Occupational Medicine and Workers' Compensation, Private Practice; Former Medical Director, Long Island Railroad

Objective: The program will inform physicians about current medical issues in the workplace and what is being done to recognize, assess, contain, treat and prevent potential and actual health problems. Adverse health effects due to worksite exposure and applicable compensation laws are explored. Referral sources for occupational medicine and information material will be provided. Physicians are encouraged to submit topic-appropriate case studies and questions up to two weeks prior to the meeting.

2:10 P.M. **Medical Surveillance in Occupational Medicine**

JACQUELINE MESSITE, M.D., New York City

Regional consultant, National Institute for Occupational Safety and Health (NIOSH), Region II; Clinical Professor, Environmental Medicine, New York University School of Medicine

2:35 P.M. **Physical Examinations in Industry**

GOTTFRIED F. H. LEHMANN, M.D., Jamaica

3:00 P.M. **Indoor Air Pollution**

ROBERT J. MCCUNNEY, M.D., Boston, Massachusetts

Corporate Medical Director, Cabot Corporation; Director, Occupational Medicine, Goddard Memorial Hospital

3:25 P.M. **New Ideas of Gasoline Toxicity**

JOSEPH M. CANNELLA, M.D., New York City

Corporate Medical Director, Mobil Corporation

3:50 P.M. **Workplace Injuries and the Law**

LAWRENCE A. KLEIN, Esq., New York City

Attorney-Specialist in Workers' Compensation Law and Occupational Diseases, Klein, Wagner and Morris

4:15 P.M.–5:00 P.M.
Panel Discussion: Case Studies
Questions and Answers

Neurology

2:00 P.M.—5:00 P.M.
SUTTON PARLOR CENTER

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Elective

RECENT ADVANCES IN NEUROLOGY

RICHARD SATRAN, M.D., Rochester, *Moderator*

Professor and Acting Chairman, Department of Neurology, University of Rochester School of Medicine and Dentistry

Objective: To review recent advances in selected areas of neurology.

2:00 P.M. **Coma: Evaluation, Treatment and Prognosis**

JOHN CARONNA, M.D., New York City

Professor and Vice Chairman, Department of Neurology, Cornell University Medical College

2:45 P.M. **Recent Advances in Sleep Medicine**

MICHAEL J. THORPY, M.D., Bronx

Assistant Professor of Neurology; Director of Sleep Disorders Center, Albert Einstein College of Medicine of Yeshiva University

3:30 P.M. **An Overview of Newer Anticonvulsant Drugs, Their Uses and Side Effects**

RONALD KRALL, M.D., Skokie, Illinois

Director of Clinical Research, Lorex Pharmaceuticals

4:15 P.M.–5:00 P.M.
Parkinson's Disease: Advances in Therapy

ROGER KURLAN, M.D., Rochester

Assistant Professor of Neurology, University of Rochester School of Medicine and Dentistry

(*continued*)

SATURDAY, JUNE 8, 1985

GENERAL SESSION
General Surgery/Orthopedic Surgery/Hand Surgery
Gastroenterology/Anesthesiology/Radiology
Joint Meeting With
NYS Society of Surgeons, NYS Society of Orthopedic Surgeons, NYS Society of Anesthesiologists, NYS Chapter of the American College of Radiology, The New York Society for Surgery of the Hand

10:00 A.M.-1:00 P.M.
SUTTON PARLOR SOUTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

HEALTH CARE 1985: SURGICAL HORIZONS

MURRY FRIEDMAN, M.D., New Hyde Park, *Moderator*

Governor, American College of Surgeons; Clinical Professor, Surgery, School of Medicine, State University of New York at Stony Brook

Objective: To acquaint the medical profession at large with significant recent advances in surgical care.

10:00 A.M. **Computer Applications in Surgery**

STUART J. NELSON, M.D., Stony Brook

Assistant Professor, Clinical Medicine, Health Sciences Center, State University of New York at Stony Brook

10:25 A.M. **Psychiatric Problems in Surgery**

ROBERT DICKES, M.D., New York City

Professor, Emeritus, State University of New York Downstate Medical Center; Clinical Professor, Psychiatry, New York University School of Medicine

10:50 A.M. **Application of Laser Therapy to G.I. (Gastrointestinal) Tract**

RICHARD MCCRAY, M.D., New York City

Chief, Gastrointestinal Endoscopy, St. Luke's-Roosevelt Hospital Center; Associate Clinical Professor, Medicine, Columbia University College of Physicians and Surgeons

11:15 A.M. **Perioperative Anesthesia Assessment**

KENNETH J. FREESE, M.D., Stony Brook

Assistant Professor of Clinical Anesthesia, School of Medicine, Health Sciences Center, State University of New York at Stony Brook

11:40 A.M. **Are Interventional Radiologists Really Surgeons in Lead Scrub Suits?**

HOWARD BERMAN, M.D., Valhalla

Associate Professor of Radiology; Associate Professor of Surgery, New York Medical College

12:05 P.M. **Joint Replacement**

DAVID HIRSH, M.D., Bronx

Associate Clinical Professor, Orthopedic Surgery, Albert Einstein College of Medicine of Yeshiva University

12:30 P.M.-1:00 P.M. **Advances in Hand Surgery**

THOMAS JOSEPH PALMIERI, M.D., New Hyde Park

Assistant Professor of Clinical Surgery, State University of New York at Stony Brook; Physician-In-Charge, Hand Surgery, Long Island Jewish-Hillside Medical Center

(*continued*)

Urology/Pathology
Joint Meeting With
New York State Society of Pathologists
and
New York State Urological Society, Inc.

10:00 A.M.-1:00 P.M.
SUTTON PARLOR CENTER

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Elective

UPDATE ON UROLOGY AND PATHOLOGY

PELLEGRINO TOZZO, M.D., New Rochelle, *Moderator*
Chief, Urology, New Rochelle Hospital Medical Center; Associate Professor of Urology, New York Medical College

and

EDWARD ZAINO, M.D., Garden City, *Moderator*
Director of Pathology, Mercy Hospital, Rockville Centre; Clinical Associate Professor of Pathology, State University of New York at Stony Brook

Objective: To provide an update on the present status of urology and pathology today.

10:00 A.M. **Current Status and Experience with Extra Corporeal Lithotripsy**
E. DARRACOTT VAUGHN, M.D., New York City
Professor of Urology; Chairman, Division of Urology, Cornell University Medical College

10:25 A.M. **Percutaneous Stone Extraction Today**
ARTHUR D. SMITH, M.D., New Hyde Park
Chief, Division Urology, Long Island Jewish-Hillside Medical Center; Associate Professor, State University of New York at Stony Brook

10:50 A.M. **Carcinoma of Prostate**
GERALD P. MURPHY, M.D., Buffalo
Director, Roswell Park Memorial Institute

11:15 A.M. **Urinary Incontinence in Females**
PASQUALE GRECO, M.D., Buffalo
Clinical Associate Professor of Urology, State University of New York at Buffalo

11:40 A.M. **The Use of the Cavitron in Genitourinary Surgery**
JOSEPH ADDONIZIO, M.D., Valhalla
Chairman, Department of Urology, New York Medical College

12:05 P.M. **Cytology of the Genitourinary Tract**
ELLEN GREENEBAUM, M.D., Bronx
Professor of Pathology, Albert Einstein College of Medicine of Yeshiva University; Director of Pathology, Montefiore Hospital and Medical Center

12:30 P.M.-1:00 P.M. **Aspiration Cytology of the Prostate**
KLAUS SCHREIBER, M.D., Bronx
Associate Professor of Pathology, Albert Einstein College of Medicine of Yeshiva University; Director of Cytology, Montefiore Hospital and Medical Center

Internal Medicine
Joint Meeting With
New York State Society of Internal Medicine

10:00 A.M.-5:00 P.M.
BEEKMAN PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 6 Hours, Category 1; AAFP, 6 Hours, Prescribed

THE SOCIOECONOMICS OF ALPHABET SOUP

SIDNEY S. WEINSTEIN, M.D., Rochester, *Moderator*
Clinical Associate Professor of Medicine, University of Rochester School of Medicine and Dentistry

(continued)

Objective: To acquaint physicians with social and economic changes affecting their ability to deliver medical care

10:00 A.M. **PROs (Professional Review Organizations) Implemented**

GEORGE POLLACK, M.D., Lake Success

Vice President and Medical Director, Empire State Medical Scientific and Educational Foundation, Inc.

10:40 A.M. **HMOs (Health Maintenance Organizations) and PPOs (Preferred Provider Organizations)**

RUTH ECKELMANN, Rochester

President, Preferred Care

11:20 A.M. **The Current Status of Hospital Reimbursement Under DRGs (Diagnostic Related Groups)**

HENRY DESMARAIS, M.D., Baltimore, Maryland

Director, Bureau of Eligibility Reimbursement and Coverage, Health Care Financing Administration

12:00 NOON **The Prospect of DRG (Diagnostic Related Group) Payments for Physician Services**

ALLEN DOBSON, Ph.D., Baltimore, Maryland

Director, Office of Research, Health Care Financing Administration

12:40 P.M.–1:00 P.M. **Panel Discussion**

2:00 P.M.–5:00 P.M.

2:00 P.M. **Cognitive Services: Catalyst to High Quality Medical Care at Less Expense**

C. BURNS ROEHRIG, M.D., Boston, Massachusetts

President, American Society of Internal Medicine

2:50 P.M. **The Changing Nature of Hospital-Physical Relationships**

JAMES BLOCK, M.D., Rochester

President, Rochester Area Hospital Corporation

3:40 P.M. **Medical Practice in a Professional Liability Crisis**

NORMAN BLACKMAN, M.D., Brooklyn

Assistant Professor Clinical Medicine, State University of New York Downstate Medical Center; Past President, New York State Society of Internal Medicine

4:30 P.M.–5:00 P.M. **Panel Discussion**

Ophthalmology
Joint Meeting With
New York State Ophthalmological Society

10:00 A.M.–5:00 P.M.
SUTTON PARLOR NORTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 6 Hours, Category 1; AAFP 6 Hours, Prescribed

ADVANCES IN PEDIATRIC OPHTHALMOLOGY AND STRABISMUS

ALFRED J. COSSARI, M.D., Port Jefferson Station, *Moderator*

Assistant Professor of Clinical Surgery, State University of New York at Stony Brook

Objective: To provide the ophthalmic clinician with a review of some recent advances which affect the field of pediatric ophthalmology and strabismus.

10:00 A.M. **The Significance of New Genetic Technology in Ophthalmology**

FREDERICK M. WANG, M.D., New York City

Assistant Clinical Professor of Ophthalmology and Pediatrics, Albert Einstein College of Medicine of Yeshiva University

10:30 A.M. **Vision Research: Animal Models**

S. MURRAY SHERMAN, Ph.D., Stony Brook

Professor of Neurobiology,
State University of New York at Stony Brook

11:00 A.M. **The Child with Reduced Vision: Electrophysiologic and Psychophysical Aspects**

MARTIN E. LEDERMAN, M.D., White Plains

Attending Physician, Ophthalmology, White Plains Hospital

11:30 A.M. **Vision Assessment in Preverbal and Nonverbal Children**

JOHN W. SIMON, M.D., Albany

Assistant Professor of Ophthalmology and Pediatrics, Albany Medical Center Hospital

12:00 NOON **Contrast Sensitivity Testing**

ALFRED J. COSSAIR, M.D., Port Jefferson Station

12:30 P.M.–1:00 P.M. **Panel Discussion**

2:00 P.M.–5:00 P.M.

2:00 P.M. **Optic Nerve Problems in Children**

MYLES M. BEHRENS, M.D., New York City

Associate Professor of Clinical Ophthalmology, Columbia University College of Physicians and Surgeons

(*continued*)

2:30 P.M. **Advances in the Treatment of Retinopathy of Prematurity and Retrolental Fibroplasia**

BEN ZANE COHEN, M.D., New York City

Clinical Instructor, Retinal Service, Mount Sinai School of Medicine of the City University of New York

3:00 P.M. **Childhood Glaucoma**

EDWARD L. RAAB, M.D., New York City

Professor of Ophthalmology; Associate Professor of Pediatrics; and Director of Pediatric Ophthalmology and Strabismus, Mount Sinai School of Medicine of the City University of New York

3:30 P.M. **Botulinum Therapy for Strasbismus**

HENRY S. METZ, M.D., Rochester

Professor and Chairman, Department of Ophthalmology, University of Rochester School of Medicine and Dentistry

4:00 P.M. **Nasolacrimal Problems in Children**

ORKAN G. STASIOR, M.D., Albany

Clinical Professor of Ophthalmology, Albany Medical College; Director, Oculoplastic Service; and Chief, Department of Ophthalmology, The Childs Hospital

4:30 P.M.–5:00 P.M. **Panel Discussion**

Psychiatry/Alcoholism
Joint Meeting With
New York State Psychiatric Association
and
American Medical Society on Alcoholism & Other Drug Dependencies—New York State Chapter

10:00 A.M.–5:00 P.M.
REGENT PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 6 Hours, Category 1; AAFP, 6 Hours, Elective

THE SPECTRUM OF PSYCHIATRY IN HEALTH CARE TODAY

RICHARD B. DROOZ, M.D., Brooklyn, *Moderator*

Associate Professor of Psychiatry (Ret.), State University of New York Downstate Medical Center; Attending Psychiatrist (Ret.), Kings County Hospital Center

Objective: To provide a modern view of the broad range of psychiatry; to provide updated information on key areas of psychiatry; and to illuminate the importance and application of psychiatry and its allied fields in modern health care.

10:00 A.M. **Introduction and Welcome**

STEPHEN NORDLICHT, M.D., New York City

Chairman, Scientific Program Committee, Medical Society of the State of New York; Consulting Psychiatrist, Memorial Hospital for Cancer and Allied Disease

10:10 A.M. **The Treatment of Atypical Depressions with Monoamine Oxidase Inhibitors**

FREDERIC M. QUITKIN, M.D., New York City

Associate Director, Clinical Psychiatry; Director, Depression and Evaluation Service, New York State Psychiatric Institute

10:50 A.M. **Recent Advances in Alcoholism Research: A Clinician's Perspective**

ANNE GELLER, M.D., New York City

Chief, Smithers Alcoholism Center of St. Luke's-Roosevelt Hospital Center; Assistant Professor of Clinical Social Medicine, Columbia University College of Physicians and Surgeons

11:30 A.M. **The Continuing Education and Development of Psychiatrists: The Educational Alliance and the Role of Small Study Groups**

JEROME S. SILVERMAN, M.D., New York City

Associate Professor of Clinical Psychiatry, Faculty, Psychoanalytic Institute, New York University School of Medicine

Formal Discussant:

MARK KANZER, M.D., Harrison

Past Director, Division of Psychoanalytic Education;

Clinical Professor of Psychiatry (Ret.), State University of New York Downstate Medical Center

12:20 P.M. **Malpractice in Psychiatry: The Lessons to be Learned: A Critical Review of 625 Cases**

HAROLD E. BERSON, M.D., Brooklyn

Attending Psychiatrist, Maimonides Medical Center; Attending Neuropsychiatrist, Interfaith Medical Center

2:00 P.M.–5:00 P.M.

2:00 P.M. **An Approach to the Treatment of Self-Deprecatory Delusions**

GEORGE M. HECHT, M.D., New York City

Assistant Professor of Psychiatry, Mt. Sinai School of Medicine of the City University of New York; Senior Candidate, Psychoanalytic Institute, New York University School of Medicine

(*continued*)

2:35 P.M. **Emergency Care In Mental Health: Pioneering in Nassau County**

Isidore Shapiro, A.C.S.W., Mineola

Commissioner of Mental Health, Mental Retardation, and Developmental Disabilities, Nassau County, State of New York; Associate Professor of Health Sciences, Health Sciences Center, State University of New York at Stony Brook

Formal Discussant:

Henry I. Schneer, M.D., Lido Beach

Professor of Clinical Psychiatry, Health Sciences Center, State University of New York at Stony Brook; President, New York State Hospitals Medical Alumni Association

3:10 P.M. **A Study of Attitudes to Patients and to Psychopathology: The Experience of Clerkship In Psychiatry**

Gerald Roskin, M.D., New Hyde Park

Director, Education and Training, Department of Psychiatry, Long Island Jewish-Hillside Medical Center

Charles J. Rabiner, M.D., New Hyde Park

Chairman, Department of Psychiatry, Long Island Jewish-Hillside Medical Center

Marjorie L. Carson, M.D., New Hyde Park

Coordinator, Medical Student, Psychiatric Education, Long Island Jewish-Hillside Medical Center

Susan Marell, Ph.D., New Hyde Park

Psychologist, Senior Psychometrician, Long Island Jewish-Hillside Medical Center

3:45 P.M. **Suicidology: The Relatives Who Have Survived**

Simeon L. Feigin, M.D., New York City

Psychiatric Consultant, Mental Health Association of Westchester County; Assistant Attending Psychiatrist, New York University Medical Center

Margaret M. Wessler, A.C.S.W., White Plains

Psychiatric Case Worker—Suicide Prevention/ Crisis Intervention Service, Mental Health Association of Westchester County

4:20 P.M. **Psychiatric Care in the Nursing Home: Procedures, Problems, and the Need for Rationing**

Lewis S. Glickman, M.D., Brooklyn

Director of Psychiatric Consultation Liaison Service, Kings County Hospital Center; Clinical Associate Professor of Psychiatry, State University of New York Downstate Medical Center

Emergency Medicine
Joint Meeting With
New York Chapter of the American College of Emergency Physicians

2:00 P.M.–5:00 P.M.
SUTTON PARLOR SOUTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

ADVANCES IN EMERGENCY MEDICINE

Lorraine M. Giordano, M.D., Jamaica Estates, *Moderator*

Medical Director, Emergency Department, Jamaica Hospital

Objective: To familiarize participants with the scope of emergency medicine; to present an overview of the components and growth of emergency medical services; to review current legislation for a New York State comprehensive EMS (Emergency Medical Service) system; to discuss updated management of selected problems in toxicology; to discuss recent updates in CPR (Cardiopulmonary Resuscitation) and ACLS (Advanced Cardiac Life Support); to examine the role of the emergency physician in disaster and environmental medicine.

2:00 P.M. **Emergency Medical Services: Twenty Years of Growth and Development**

Anthony Mustalish, M.D., New York City

Chief, Emergency Department, Lenox Hill Hospital

2:30 P.M. **An Emergency Medical System for New York City: Current Emergency Department Standards and Categorization**

Diane Sixsmith, M.D., New York City

Chairperson, Regional Emergency Medical Services Council of New York City; Medical Director, Emergency Services, Emergency Care Institute, New York Infirmary-Beekman Downtown Hospital

3:00 P.M. **An Emergency Medical System for New York State**

Mark Henry, M.D., Flushing

Vice Chairman, New York State EMS (Emergency Medical Services) Council; Director, Department of Emergency Medicine, Booth Memorial Medical Center

3:30 P.M. **Advances in Toxicology**

Louis Goldfrank, M.D., New York City

Director, Emergency Services, Bellevue Hospital Center and University Hospital; Associate Professor of Clinical Medicine, New York University School of Medicine

4:00 P.M. **Updates in CPR (Cardiopulmonary Resuscitation) and ACLS (Advanced Cardiac Life Support)**

Thomas Kwiatkowski, M.D., New York City

Physician-in-Charge, Emergency Services, Queens General Hospital

4:30 P.M.–5:00 P.M. **The Emergency Physician's Role In Environmental and Disaster Medicine**

Wayne Longmore, M.D., New York City

Clinical Instructor, Department of Surgery; Attending Physician, Emergency Department, Bellevue Hospital Center and New York University Medical Center

(continued)

Otolaryngology—Head & Neck Surgery
Joint Meeting With
New York State Society of Otolaryngology—Head & Neck Surgery

2:00 P.M.–5:00 P.M.
SUTTON PARLOR CENTER

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Elective

RECENT ADVANCES IN OTOLARYNGOLOGY

STEVEN M. PARNES, M.D., Albany, *Moderator*

Head, Division of Otolaryngology, Albany Medical College of Union University; Member and Representative, N.Y.S. Society of Otolaryngology—Head & Neck Surgery

Objective: To acquaint the practicing Otolaryngologist with specific recent developments in our specialty.

2:00 P.M. **Introduction**

STEVEN M. PARNES, M.D.

2:05 P.M. **Application of the YAG (Yttrium Aluminum Garnet) Laser in Otolaryngology—Head & Neck Surgery**

STANLEY SHAPSHY, M.D., Burlington, Massachusetts

Chairman, Department of Otolaryngology—Head & Neck Surgery; Director, Laser Research Laboratory, Lahey Clinical Hospital

2:40 P.M. **Cochlear Implants**

SUSAN WALTZMAN, Ph.D., New York City

Associate Professor, Department of Otolaryngology—Head & Neck Surgery, New York University Medical Center

3:40 P.M. **Surgical Approaches to Sleep Disorders**

AARON E. SHER, M.D., New York City

Assistant Professor of Otolaryngology, Albert Einstein College of Medicine of Yeshiva University

4:45 P.M.–5:00 P.M. **Questions and Discussion**

SUNDAY, JUNE 9, 1985

MEET THE PROFESSOR

Planned and organized by Maxwell Spring, M.D., Associate Chairman, Scientific Program Committee
Clinical Associate Professor of Medicine, New Jersey Medical School

8:00 A.M.–8:30 A.M. Continental Breakfast
8:30 A.M.–10:00 A.M. Meeting

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 1½ Hours, Category 1;
AAFP, 1½ Hours, Prescribed

An opportunity to meet informally and discuss clinical problems on a one-to-one basis with experts in your field.

Registration Fee: $35.00 Per Session (includes continental breakfast)

8:30 A.M. **Dose Of Oxygen** (Room 529)

ANTHONY REBUCK, M.D., Toronto, Canada

Professor of Medicine,
University of Toronto Faculty of Medicine

Objective: Physicians attending this session will be made familiar with the proper use of O_2 in the every day care of cardiac and pulmonary patients.

8:30 A.M. **Bronchial Asthma** (Room 534)

MICHAEL H. GRIECO, M.D., New York City

Director, R. A. Cooke Institute of Allergy,
St. Luke's—Roosevelt Hospital Center

Objective: The presentation will review current concepts concerning the immunologic and nonimmunologic mechanisms involved in pathogenesis of bronchial asthma. In addition, the management of ambulatory asthmatics will be discussed.

(continued)

8:30 A.M. **The Drug Therapy of Arthritis with Special Attention to the Nonsteroidal Drugs** (Room 537)

THOMAS KANTOR, M.D., New York City

Professor of Medicine; Chief, Division of Rheumatology,
New York University School of Medicine

Objective: This session will deal with the establishment of research modalities for the study of oral analgetic compounds in humans and will demonstrate the all-purpose analgetic activity of the nonsteroidal, anti-inflammatory drugs in addition to their antiarthritic activity.

8:30 A.M. **New Aspects of Iron Deficiency and Iron Overload** (Room 540)

SIMON POLLACK, M.D., Bronx

Professor of Medicine,
Albert Einstein College of Medicine

Objective: The objective of this presentation will be to explain current concepts of iron absorption and utilization, and the diagnosis and treatment of iron deficiency and iron overload. Pitfalls in the use of available tests will be described and the strategy for making a diagnosis in the partially treated patient will be explored.

8:30 A.M. **Fluid and Electrolytes** (Room 543)

ARNOLD ABERMAN, M.D., Toronto, Canada

Chief of Medicine, Mt. Sinai Hospital,
University of Toronto Faculty of Medicine

Objective: An approach to fluid and electrolyte management in the hospitalized patient based on the various compartments of the body will be presented. Particular emphasis will be placed on hypo- and hyper-natremia.

Allergy and Immunology

10:00 A.M.–1:00 P.M.
BEEKMAN PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

HEALTH CARE TODAY

RENATA FRENKEL, M.D., New York City, *Moderator*

Associate in Clinical Pediatrics, Columbia University College of Physicians and Surgeons; Chief of Allergy, Manhattan Eye, Ear and Throat Hospital

Objective: This program is designed not only for the Allergist, but for all Primary Care Physicians, with the purpose of updating them in the diagnosis and management of common allergic problems presented in office practice.

10:00 A.M. **Indications for Immunotherapy**

EARL B. BROWN, M.D., New York City

Assistant Professor, Clinical Medicine, Columbia University College of Physicians and Surgeons; Chief of Allergy, Lenox Hill Hospital

10:40 A.M. **Controversial Practices in Allergy**

MICHAEL H. GRIECO, M.D., New York City

Director, R. A. Cocke Institute of Allergy, St. Lukes-Roosevelt Hospital Center; Professor of Clinical Medicine, Columbia University College of Physicians and Surgeons

11:20 A.M. **Cooperative Management of Asthma**

LAWRENCE T. CHIARAMONTE, M.D., Brooklyn

Director, Department of Allergy and Immunology, The Long Island College Hospital; Clinical Assistant Professor, State University of New York Downstate Medical Center

11:55 A.M. **New Advances in Detection of Penicillin Allergy**

GILLIAN M. SHEPHERD, M.D., New York City

Assistant Professor of Medicine, Cornell University Medical College; Assistant Attending Physician, New York Hospital

12:25 P.M. **Diagnosis of Immunodeficiency**

NICHOLAS T. MACRIS, M.D., New York City

Chief, Allergy and Immunology Section, Department of Medicine, Lenox Hill Hospital; Clinical Associate Professor of Medicine, New York Hospital-Cornell Medical Center

(*continued*)

GENERAL SESSION
The Physician As A Witness

10:00 A.M.–1:00 P.M.
SUTTON PARLOR NORTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

JUDGE JACOB D. FUCHSBERG, New York City, *Moderator*

Senior Partner, Jacob D. Fuchsberg Law Firm; Former Justice, Court of Appeals, State of New York; Former President, American Trial Lawyers Association

OBJECTIVE: TO TEACH THE PHYSICIAN HOW TO HANDLE HIMSELF IN COURT.

10:00 A.M. **Introduction**

MAXWELL SPRING, M.D., F.A.C.P., Bronx

Associate Chairman, Scientific Program Committee, Medical Society of the State of New York; Clinical Associate Professor of Medicine, New Jersey Medical School

10:05 A.M. **The Physician's Proper Approach to Testifying**

IRVIN KLEIN, M.D., New York City

Medical Director, Workers' Compensation Board of New York (Retired); Former President, American College of Occupational Medicine

11:00 A.M. **The Physician and Malpractice**

HERBERT BERGER, M.D., Richmond

Professor of Clinical Medicine, New York Medical College; Former Vice-President, Medical Society of the State of New York and New York Academy of Medicine

12:00 NOON–1:00 P.M. **Trial Preparation for the Medical Expert**

FRED QUELLER, Esq., New York City

President of New York State Trial Lawyers Association; Certified as Civil Trial Advocate In Who's Who in American Law

---

ANNUAL MEETING
Women's Medical Society of New York State/Family Practice
Joint Meeting With
American Medical Women's Association Branch 18
and
New York State Academy of Family Physicians

10:00 A.M.–1:00 P.M.
SUTTON PARLOR CENTER

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

CLINICAL PROBLEMS IN FAMILY PRACTICE

PATRICIA FORD, M.D., Schenectady, *Moderator*

Resident Physician, Family Practice (Second Year), St. Clare's Hospital

Objective: This program is meant to familiarize physicians with areas of clinical medicine which confront the primary care physician almost daily. It should broaden the diversity of the physicians' knowledge and increase his/her ability to manage common problems.

10:00 A.M. **A Sample Case Illustrating the Family Practitioners' Continuity of Care in Obstetrics and Care of the Neonate**

MARY VERDON, M.D., Johnson City

Resident Physician, Family Practice (Second Year), Wilson Hospital

10:25 A.M. **Case Presentation: Staphylococcal Pneumonia and Its Sequelae**

PATRICIA FORD, M.D., Schenectady

10:50 A.M. **Case Presentation: Primary Hypoventilation Syndrome**

JOHN SABIA, M.D., Schenectady

Resident Physician, Family Practice (First Year), St. Clare's Hospital

11:15 A.M. **Discussion**

11:25 A.M. JEANNE SMITH, M.D., New York City, *Moderator*

11:30 A.M. **Pathology and Treatment of Irrational Guilt Syndromes in Family Practice**

JEANNE SMITH, M.D., F.A.A.P., New York City

Fellow, American Academy of Pediatrics and American Academy of Psychoanalysis

12:15–1:00 P.M. **Radioimmunoassay in Clinical Medicine**

ROSALYN S. YALOW, Ph.D., Bronx

Senior Medical Investigator, Veterans Administration Medical Center; Nobel Prize Winner in Medicine and Physiology, 1977 ■

1985 ANNUAL CME ASSEMBLY

TECHNICAL EXHIBITS

RHINELANDER GALLERY
NEW YORK HILTON

Accumedic Computer Systems, Inc. New York, New York	**Booth 62**
AIS Corp. Bridgewater, New Jersey	**Booth 41**
American Express Co. New York, New York	**Booth 54**
Blue Cross/Blue Shield of Greater New York New York, New York	**Booth 59**
Brentwood Instruments, Inc. Torrance, California	**Booth 43**
Burlington Systems Inc. Burlington, Vermont	**Booth 56**
Carann Leasing Corporation Massapequa Park, New York	**Booth 74**
Damon Clinical Laboratories Needham Heights, Massachusetts	**Booth 65**
Encyclopaedia Britannica USA Chicago, Illinois	**Booth 64**
EPIC Residential Network, Inc. Falls Church, Virginia	**Booth 48**
Equitable Life Assurance Society New Hyde Park, New York	**Booth 66**
FBL Medical Computer Specialists Fairfield, New Jersey	**Booth 29**
Gibraltar Securities Florham Park, New Jersey	**Booth 57**
Group Health Incorporated New York, New York	**Booth 31**
Hoechst-Roussel Pharmaceuticals, Inc. Somerville, New Jersey	**Booth 63**
International Preferred Assets Coppell, Texas	**Booth 40**
Marketing Communications, Inc. Brooklyn, New York	**Booth 28**
Marsh & McLennan Group Associates New York, New York	**Booth 42**
Marsh & McLennan Group Associates New York, New York	**Booth 68**
Mead Johnson Nutritional Division Evansville, Indiana	**Booth 60**
Medical Business Services New York, New York	**Booths 69 and 70**
Medical Liability Mutual Insurance Co. **Donald J. Fager & Assoc., Inc.** New York, New York	**Booth 44**
Metpath Inc. Teterboro, New Jersey	**Booth 1**
New York Army National Guard Albany, New York	**Booth 39**
Pfizer Pharmaceuticals New York, New York	**Booth 35**
S. A. Ramirez & Co., Inc. New York, New York	**Booth 67**

Continued next page

Medical Society of the
State of New York

Annual Dinner Dance

in honor of
Allison B. Landolt, M.D.
President

Thursday
June 6, 1985
Trianon Ballroom
The New York Hilton

53rd Street & Avenue of America

Cocktail Hour 7 P.M.	Superlative Dinner

Subscription $70.00 per person

Reservation Form

Make check payable to:
Medical Society of the State of New York
420 Lakeville Road
Lake Success, New York 11042

Please send me _______ tickets for the
Annual Dinner Dance on June 6, 1985
Check enclosed for $__________

PLEASE NOTE: Reservations and ticket sales will close at 12 Noon on Thursday, June 6, one hour before the House Convenes.

Name______________________________________

Address____________________________________

____________________________Zip____________

Schering Laboratories Bloomfield, New Jersey	**Booth 47**
Shearson Lehman/American Express Inc. White Plains, New York	**Booth 61**
U. S. Air Force Randolf Air Force Base, Texas	**Booth 73**
U. S. Army Medical Department Washington, D.C.	**Booth 27**
The Upjohn Company Garden City, New York	**Booth 36**
USV Laboratories Tarrytown, New York	**Booth 55**
Whittman-Burke Refining Corp. Midland Park, New Jersey	**Booth 71**
Winthrop-Breon Laboratories Cherry Hill, New Jersey	**Booth 58**

■

Note: Exhibitors who are not listed here will appear in the final program book which will be distributed at the CME Assembly registration desk during the meeting.

Advance Registration Form

Complete and return this form to the Medical Society address as indicated below.

1985 Annual CME Assembly, June 7–9

The New York Hilton, New York City

PLEASE PRINT

NAME ______________________________
Last *First*

Address ______________________________

City/State/Zip ______________________________

Medical Specialty ______________________________

REGISTRATION FEE

Please check category

☐ Non-Member Physician	$50.00
☐ Allied Profession	$10.00

Please check category

- ☐ Nurse
- ☐ Physician Assistant
- ☐ Dentist
- ☐ Lab Technician
- ☐ PT/OT
- ☐ Other (specify) ______________

NO FEE

- ☐ MSSNY Active Member
- ☐ MSSNY Life Member
- ☐ Resident/Intern/Fellow
- ☐ Medical Student
- ☐ Medical Assistant
- ☐ Speaker
- ☐ Spouse/Family
- ☐ News Media/Medical Writer

MEET THE PROFESSOR—Continental Breakfast Meetings.

Fee: $35.00 per session. Time: 8:00 a.m.-10:00 a.m. Please indicate session(s) attending. Each session is limited to 30 persons.

Friday, June 7

- ☐ Charles Hollander, M.D.
- ☐ Harold Itskovitz, M.D.
- ☐ Harold Lebovitz, M.D.
- ☐ Donald Louria, M.D.
- ☐ Carroll Leevy, M.D.

Sunday, June 9

- ☐ Arnold Aberman, M.D.
- ☐ Michael Grieco, M.D.
- ☐ Thomas Kantor, M.D.
- ☐ Simon Pollack, M.D.
- ☐ Anthony Rebuck, M.D.

(see this issue for subjects to be presented by each Professor)

REMITTANCE

Registration Fee $ ____________
Meet the Professor session(s) $ ____________
TOTAL $ ____________

Please make check payable to:
Medical Society of the State of New York, and return this form with remittance to: Division of Meeting Services, MSSNY, 420 Lakeville RD., P.O. Box 5404, Lake Success, N.Y. 11042

Important: Your ID Badge will be held at the MSSNY Registration Desk located in the Exhibit Hall, on the second floor of the New York Hilton.

Meet the Professor – June 7th and 9th

at the Medical Society of the State of New York's Annual CME Assembly, June 7-9, 1985, the New York Hilton, New York City. Five continental breakfast meetings, 2 hours each, will be offered on Friday and Sunday mornings from 8:00 a.m.-10:00 a.m. Sessions are limited to 30 attendees each, and there is a $35.00 fee. Please see the advance registration form in this section.

Hollander

Itskovitz

Lebovitz

Louria

Leevy

Aberman

FRIDAY, JUNE 7

Charles Hollander, M.D.

Professor of Medicine & Chief of Endocrinology, New York University School of Medicine, New York City. To provide a very brief review of our understanding of the physiology and pathophysiology of thyroid, pituitary and hypothalamic disorders.

Harold D. Itskovitz, M.D.

Professor of Medicine & Pharmacology, New York Medical College, Valhalla, New York. To familiarize the physician with new drugs used to treat hypertension; to stress changing concepts concerned with etiology, work-up and management of hypertension.

Harold E. Lebovitz, M.D.

Chief, Endrocrinology & Metabolism, SUNY, Downstate Medical Center, Brooklyn, New York. To provide an understanding of the pathophysiology of the two major types of diabetes, and to develop a rational set of goals for the treatment of patients with diabetes.

Donald Louria, M.D.

Professor & Chairman, Department of Preventive Medicine & Community Health, New Jersey Medical School, College of Medicine & Dentistry of New Jersey, Newark, New Jersey. To develop a programatic approach to the seriously ill patient with a putative infection.

Carroll Leevy, M.D.

Professor & Chairman, Department of Medicine, New Jersey Medical School, College of Medicine & Dentistry of New Jersey, Newark, New Jersey. Immunologic abnormalities are key factors in pathogenesis of hepatitus due to infections, alcoholism, and drug induced toxicity. Prevention, diagnosis and treatment have been greatly facilitated by recent advances in genetics, availabilty of monoclonal antibodies and the use of recombinant D.N.D. (Deoxyribonucleic Acid) technology.

SUNDAY, JUNE 9

Arnold Aberman, M.D.

Chief of Medicine, Mt. Sinai Hospital, University of Toronto Faculty of Medicine, Toronto, Canada. To approach the fluid and electrolyte management in the hospitalized patient with particular emphasis on hypo and hypernatremia.

Continued on next page

Meet the Professor - continued

Michael H. Grieco, M.D.

Director, R.A. Cooke Institute of Allergy, St. Luke's-Roosevelt Hospital Center, New York City. A review of current concepts concerning the immunologic and nonimmunologic mechanisms involved on pathogenesis of bronchial asthma, and review the management of ambulatory asthmatics.

Grieco

Kantor

Thomas Kantor, M.D.

Professor of Medicine; Chief, Division of Rheumatology, New York University School of Medicine, New York City. This session will demonstrate the all-purpose analgetic activity of the nonsteroidal, anti-inflammatory drugs in addition to their antiarthritic activity.

Simon Pollack, M.D.

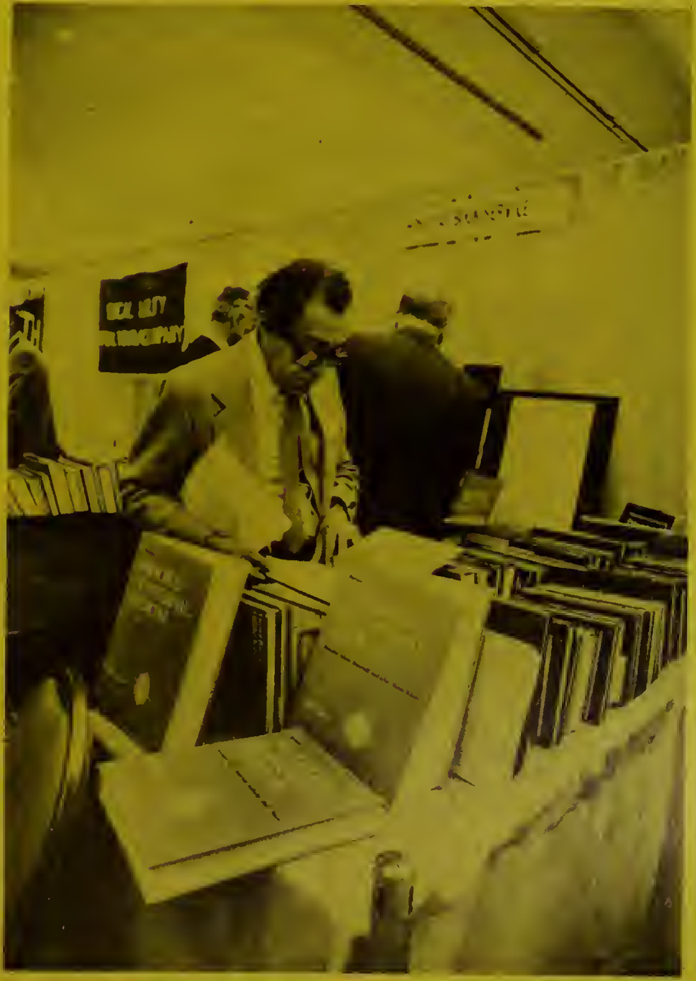
Professor of Medicine, Albert Einstein College of Medicine, Bronx, New York. The objective of this presentation will be to explain current concepts of iron absorption and utilization, and the diagnosis and treatment of iron deficiency and iron overload.

Pollack

Rebuck

Anthony Rebuck, M.D.

Professor of Medicine, University of Toronto Faculty of Medicine, Toronto, Canada. Physicians attending this session will be made familiar with the proper use of oxygen in the every day care of caridac and pulmonary patients.



DOCTOR:

is your Medical Assistant keeping in step with you?

As medical practice becomes more complicated and more highly specialized, you need more highly trained medical assistants in your office.

Membership in the AMERICAN ASSOCIATION OF MEDICAL ASSISTANTS will help your assistants keep up-to-date and informed of new ideas and techniques. AAMA's continuing education program offers workshops and seminars that will enhance the professionalism of your office employees.

As the first professional organization for medical assistants (founded 1956), AAMA pioneered in developing the only certification program in this field. A medical assistant who successfully completes the examination is identified as a Certified Medical Assistant (CMA). More than 8,000 certificates have been earned since the first examination was given in 1963.

The AAMA pioneered in the development of curriculum standards for medical assisting programs. The American Medical Association, in collaboration with AAMA, is recognized as an official accrediting agency for such programs by the U.S. Office of Education.

On five different occasions the AMA House of Delegates has passed resolutions commending the objectives of the AAMA, endorsing its functions, and urging every physician to encourage medical assistants to join the association in order to benefit from its educational programs.

To help your medical assistants do a better job of helping you, urge them to join AAMA—the professional association dedicated to their continuing education. Fill in the attached coupon and mail it today. Your practice deserves the best!

I wish to inquire about membership for my medical assistant in the American Association of Medical Assistants, Inc. Please send more information to:

Name____________________

Business Address____________________

City/State/Zip____________________

County________________Phone________________

Names and Addresses of Assistants____________________

Clip and mail to: AAMA, NYSS, Inc., c/o Department of Information, Medical Society State of New York, 420 Lakeville Road, Lake Success, New York 11042.

Have you looked at THE LANCET lately?

There's a lot in it for you!

Each week, *The Lancet* cover tells you "at-a-glance" what you'll find inside — to help you plan your reading!

The focal point of each issue— articles you'll find timely, relevant, and applicable to your practice

medical news briefs written by The Lancet editors - fully referenced for further reading

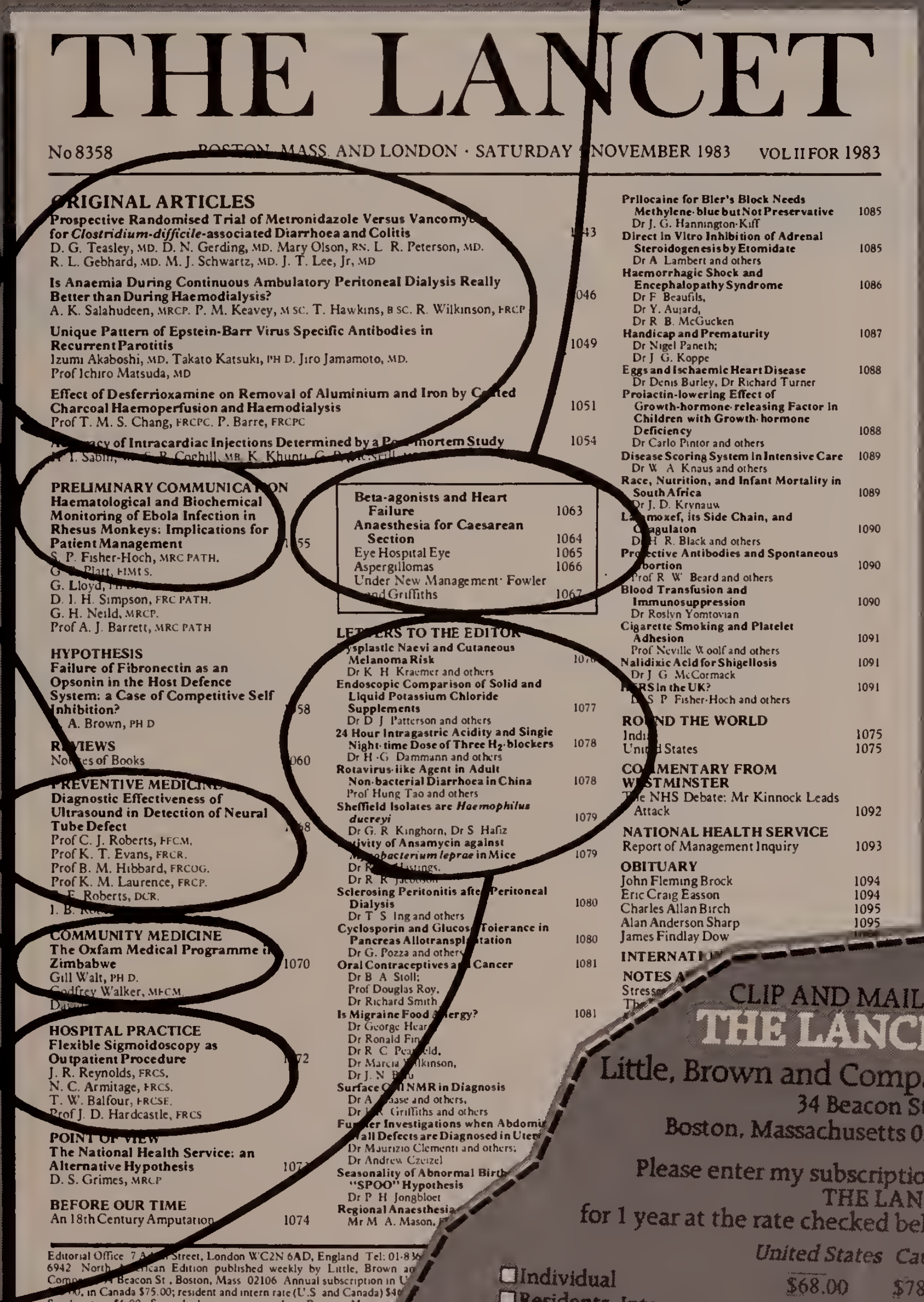
occasional early reports on breakthroughs

tips on improving your diagnostic skills

ideas and trends from around the world

critical assessments of current practice

important comments from readers - your colleagues from around the world - on issues that can affect your practice



THE LANCET

No 8358 BOSTON, MASS. AND LONDON · SATURDAY NOVEMBER 1983 VOL II FOR 1983

ORIGINAL ARTICLES

Prospective Randomised Trial of Metronidazole Versus Vancomy for *Clostridium-difficile*-associated Diarrhoea and Colitis 43
D. G. Teasley, MD. D. N. Gerding, MD. Mary Olson, RN. L R. Peterson, MD. R. L. Gebhard, MD. M. J. Schwartz, MD. J. T. Lee, Jr, MD

Is Anaemia During Continuous Ambulatory Peritoneal Dialysis Really Better than During Haemodialysis? 046
A. K. Salahudeen, MRCP. P. M. Keavey, M SC. T. Hawkins, B SC. R. Wilkinson, FRCP

Unique Pattern of Epstein-Barr Virus Specific Antibodies in Recurrent Parotitis 1049
Izumi Akaboshi, MD. Takato Katsuki, PH D. Jiro Jamamoto, MD. Prof Ichiro Matsuda, MD

Effect of Desferrioxamine on Removal of Aluminium and Iron by Coated Charcoal Haemoperfusion and Haemodialysis 1051
Prof T. M. S. Chang, FRCPC. P. Barre, FRCPC

acy of Intracardiac Injections Determined by a Post-mortem Study 1054

PRELIMINARY COMMUNICATION

Haematological and Biochemical Monitoring of Ebola Infection in Rhesus Monkeys: Implications for Patient Management 55
S. P. Fisher-Hoch, MRC PATH.
G. Platt, FIMLS.
G. Lloyd,
D. I. H. Simpson, FRC PATH.
G. H. Neild, MRCP.
Prof A. J. Barrett, MRC PATH

HYPOTHESIS

Failure of Fibronectin as an Opsonin in the Host Defence System: a Case of Competitive Self Inhibition? 58
A. Brown, PH D

REVIEWS

Notices of Books 060

PREVENTIVE MEDICINE

Diagnostic Effectiveness of Ultrasound in Detection of Neural Tube Defect 68
Prof C. J. Roberts, FFCM.
Prof K. T. Evans, FRCR.
Prof B. M. Hibbard, FRCOG.
Prof K. M. Laurence, FRCP.
E. Roberts, DCR.
I. B.

COMMUNITY MEDICINE

The Oxfam Medical Programme in Zimbabwe 1070
Gill Walt, PH D.
Godfrey Walker, MFCM.

HOSPITAL PRACTICE

Flexible Sigmoidoscopy as Outpatient Procedure 72
J. R. Reynolds, FRCS.
N. C. Armitage, FRCS.
T. W. Balfour, FRCSE.
Prof J. D. Hardcastle, FRCS

POINT OF VIEW

The National Health Service: an Alternative Hypothesis 107
D. S. Grimes, MRCP

BEFORE OUR TIME

An 18th Century Amputation 1074

Beta-agonists and Heart Failure 1063
Anaesthesia for Caesarean Section 1064
Eye Hospital Eye 1065
Aspergillomas 1066
Under New Management: Fowler and Griffiths 1067

LETTERS TO THE EDITOR

ysplastic Naevi and Cutaneous Melanoma Risk 107
Dr K H Kraemer and others
Endoscopic Comparison of Solid and Liquid Potassium Chloride Supplements 1077
Dr D J Patterson and others
24 Hour Intragastric Acidity and Single Night-time Dose of Three H_2-blockers 1078
Dr H-G Dammann and others
Rotavirus-like Agent in Adult Non-bacterial Diarrhoea in China 1078
Prof Hung Tao and others
Sheffield Isolates are *Haemophilus ducreyi* 1079
Dr G. R Kinghorn, Dr S Hafiz
ivity of Ansamycin against *obacterium leprae* in Mice 1079
Dr R Hastings,
Dr R R
Sclerosing Peritonitis after Peritoneal Dialysis 1080
Dr T S Ing and others
Cyclosporin and Glucose Tolerance in Pancreas Allotransplantation 1080
Dr G. Pozza and others
Oral Contraceptives and Cancer 1081
Dr B A Stoll;
Prof Douglas Roy,
Dr Richard Smith
Is Migraine Food Allergy? 1081
Dr George Hear
Dr Ronald Fin
Dr R C Pea ld,
Dr Marcia Wilkinson,
Dr J. N u
Surface NMR in Diagnosis
Dr A aase and others,
Dr Griffiths and others
Further Investigations when Abdomi all Defects are Diagnosed in Uter
Dr Maurizio Clementi and others;
Dr Andrew Czeizel
Seasonality of Abnormal Birth "SPOO" Hypothesis
Dr P H Jongbloet
Regional Anaesthesia
Mr M A. Mason,

Prilocaine for Bier's Block Needs Methylene-blue but Not Preservative 1085
Dr J. G. Hannington-Kiff
Direct In Vitro Inhibition of Adrenal Steroidogenesis by Etomidate 1085
Dr A Lambert and others
Haemorrhagic Shock and Encephalopathy Syndrome 1086
Dr F Beaufils,
Dr Y. Aujard,
Dr R B. McGucken
Handicap and Prematurity 1087
Dr Nigel Paneth;
Dr J G. Koppe
Eggs and Ischaemic Heart Disease 1088
Dr Denis Burley, Dr Richard Turner
Prolactin-lowering Effect of Growth-hormone-releasing Factor in Children with Growth-hormone Deficiency 1088
Dr Carlo Pintor and others
Disease Scoring System in Intensive Care 1089
Dr W A Knaus and others
Race, Nutrition, and Infant Mortality in South Africa 1089
Dr J. D. Krynauw
Latamoxef, its Side Chain, and Coagulation 1090
Dr H R. Black and others
Protective Antibodies and Spontaneous Abortion 1090
Prof R W Beard and others
Blood Transfusion and Immunosuppression 1090
Dr Roslyn Yomtovian
Cigarette Smoking and Platelet Adhesion 1091
Prof Neville Woolf and others
Nalidixic Acid for Shigellosis 1091
Dr J G McCormack
RS in the UK? 1091
S P Fisher-Hoch and others

ROUND THE WORLD

India 1075
United States 1075

COMMENTARY FROM WESTMINSTER

The NHS Debate: Mr Kinnock Leads Attack 1092

NATIONAL HEALTH SERVICE

Report of Management Inquiry 1093

OBITUARY

John Fleming Brock 1094
Eric Craig Easson 1094
Charles Allan Birch 1095
Alan Anderson Sharp 1095
James Findlay Dow

INTERNAT

NOTES A
Stress
Th

Editorial Office 7 A Street, London WC2N 6AD, England Tel: 01-83 6942 North American Edition published weekly by Little, Brown a Com Beacon St, Boston, Mass 02106 Annual subscription in U , in Canada $75.00; resident and intern rate (U.S and Canada) $4 Single copy $6 00 Second class postage paid at Boston, Mass, additional mailing offices. © The Lancet Ltd, 5 Nov 1983 Notif Form 3579 is to be mailed to The Lanced, 34 Beacon Street, Bo 02106

THE WHOLE OF THE LITERARY MATTER IN THE LANC

SUBSCRIBE TODAY!

CLIP AND MAIL TO:
THE LANCET
Little, Brown and Company
34 Beacon Street
Boston, Massachusetts 02106

Please enter my subscription to THE LANCET for 1 year at the rate checked below.

	United States	Canada
☐ Individual	$68.00	$78.00*
☐ Residents, Interns, Students, Nurses	$46.50	$46.50*

*U.S. Dollars

Payment MUST accompany order

Please print clearly:

Name ____________________

Address ____________________

City, State, Zip ____________________

Specialty ____________________

M

Classified Advertising

New York State Journal of Medicine

GENERAL INFORMATION

All classified advertisements are payable in advance, except for government agencies and advertising agencies; a 15% commission to recognized advertising agencies.

Frequency: Monthly
Mailing date: 15th of the month
Closing date: 30 days before mailing date

Circulation: 27,000 (mainly physicians & medical libraries)

DISPLAY ADVERTISING

	1 time	*6 time*	*12 time*	*Width*		*Depth*
1/4-page	$450	$400	$350	3-1/2″	×	4-7/8″
1/6-page	210	190	170	2-1/8″	×	4-7/8″
1/8-page	190	170	150	3-1/2″	×	2-3/8″
1/16-page	95	85	75	3-1/2″	×	1-1/8″
1/18-page	70	60	50	2-1/8″	×	1-1/2″

PARAGRAPH ADVERTISING

The first several words are set in bold face caps.

Rates: $25.00 per insertion for 50 words or less; additional words are 30 cents each. On request, Dept. numbers will be assigned by the Journal for replies to be forwarded to the advertiser. Dept. numbers are 50 cents per insertion. There is no frequency discount for paragraph ads.

Counting Words: Two initials, each abbreviation, figures consisting of 5 numerals or less, are counted as separate words. For replies, your name and address, or telephone number should appear at the end of the ad and be counted in the total number of words.

For further information, please call the Advertising Department at 516-488-6100.

CLASSIFIED ADVERTISING ORDER FORM

New York State Journal of Medicine
Attn: Classified Advertising
420 Lakeville Rd., P.O. Box 5404
Lake Success, N.Y. 11042

Date____________________

Name____________________________________

Payment enclosed: $____________

Address__________________________________

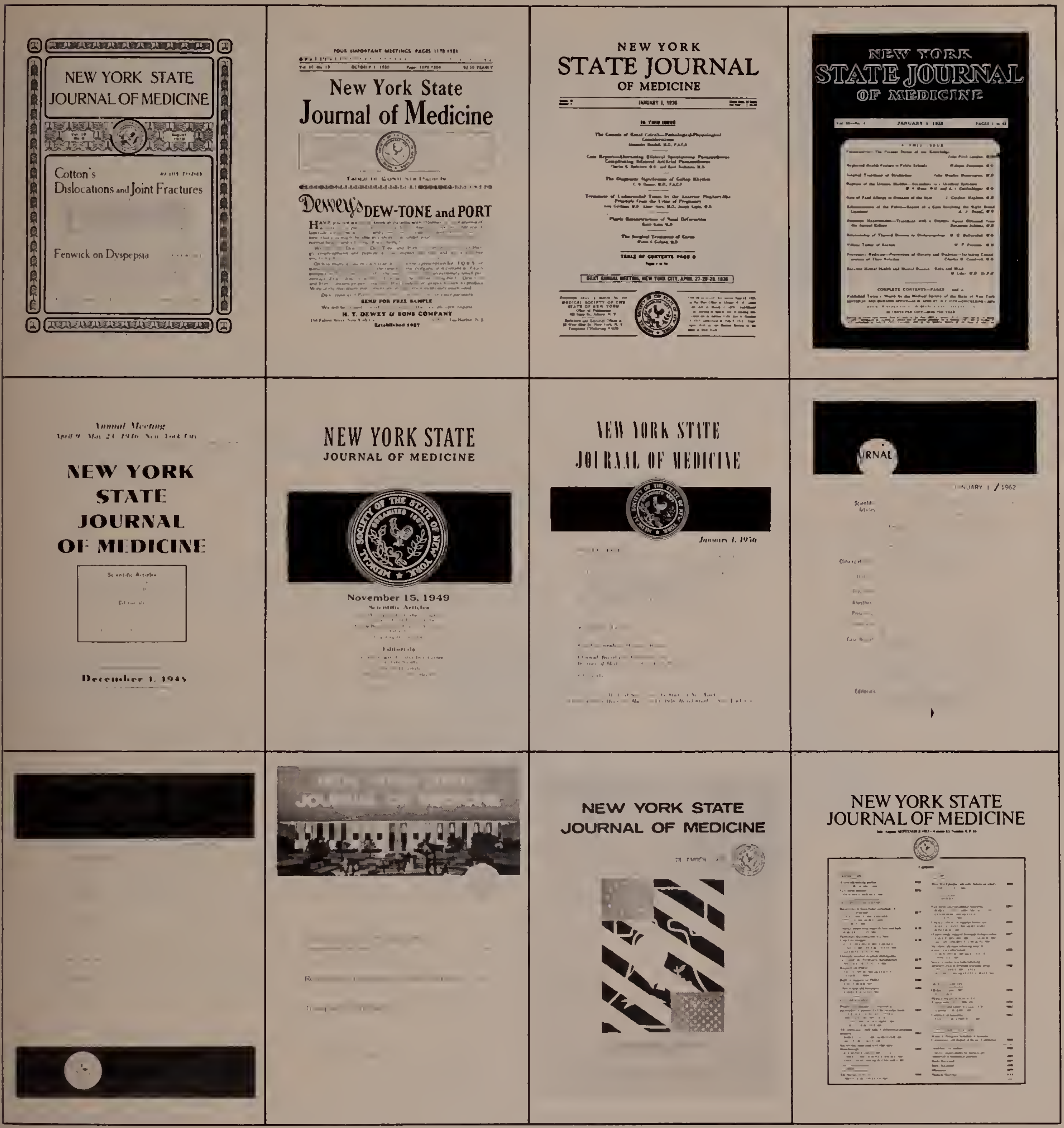
Number of Insertions: __________

City/State/Zip_______________________________

Assign Dept. Number: __________

Please make check payable to New York State Journal of Medicine.

Cover to cover . . . . always good reading.



NEW YORK STATE JOURNAL OF MEDICINE

Cotton's Dislocations and Joint Fractures

Fenwick on Dyspepsia

New York State Journal of Medicine

Dewey's DEW-TONE and PORT

SEND FOR FREE SAMPLE

H. T. DEWEY & SONS COMPANY

Established 1857

NEW YORK STATE JOURNAL OF MEDICINE

JANUARY 1, 1936

IN THIS ISSUE

NEW YORK STATE JOURNAL OF MEDICINE

JANUARY 1, 1938

NEW YORK STATE JOURNAL OF MEDICINE

December 1, 1945

NEW YORK STATE JOURNAL OF MEDICINE

November 15, 1949

Scientific Articles

Editorials

NEW YORK STATE JOURNAL OF MEDICINE

NEW YORK STATE JOURNAL OF MEDICINE

NEW YORK STATE JOURNAL OF MEDICINE

In 1985, the *New York State Journal of Medicine* promises to provide timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news. The journal will make a welcome gift to a colleague or student in the United States or abroad.

A free copy of the December 1983 issue, a special issue on smoking, will be included with each subscription while the supply lasts.

SUBSCRIPTION ORDER FORM Clip and mail

Circulation Department
New York State Journal of Medicine
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042

Name______________________________

Address______________________________

____

	1 Year	2 Years	3 Years
U.S. & Canada	☐ $25	☐ $45	☐ $60
Foreign	☐ $30	☐ $55	☐ $75

Payment required with order.

THE BEST INVESTMENT YOU'LL EVER MAKE IN YOUR PRACTICE!

A Successful "Discreet" Approach To Practice Marketing

A new seminar program presented through Practice Marketing Seminars by D. BAKER JACOBS, the creator of the highly-acclaimed PRACTICE DEVELOPMENT AND EXPANSION PROGRAM.

> "An excellent job of compacting into a small package lots of valuable information."
>
> — **AMERICAN ACADEMY OF FAMILY PHYSICIANS**

> "An important contribution for doctors interested in ethically expanding their practices."
>
> — **INDIANA STATE MEDICAL ASSOCIATION**

> ". . . a step-by-step map for those unfamiliar with the territories of media, image and public awareness."
>
> —**DELAWARE MEDICAL JOURNAL**

The seminar is a three hour intensive program detailing the essentials of developing a Discreet Marketing action plan for professional practice growth. Cost for the seminar is $145.00. A variety of convenient session times are available.

Learn to:

- Establish the appropriate practice image.
- Identify your prospective patients.
- Create public awareness for your practice.
- Use awareness to attract new patients.
- Develop a program for patient relations.
- Increase word-of-mouth referrals.
- Use your expertise to generate exposure.

Registration is limited so call or write Practice Marketing Seminars at 305 Madison Avenue, Suite 1338, New York, NY 10165 (212) 697-9110 for additional information about available times and locations.

From Blue Cross and Blue Shield of Greater N.Y.

The last claim form!

Introducing

Amicus 1™

MEDICAL OFFICE MANAGEMENT SYSTEM

A computer system that does something you could never do before

For over 50 years, Blue Cross and Blue Shield of Greater New York has been the most consistently innovative force in helping physicians and patients meet the financial demands of progressive medical care. Now, with advances in the technology of computer networking, we can help you do something never before possible. Amicus 1 lets you transmit claims directly by phone to our computer for faster, more efficient processing of our medical and Medicare B* claims.

Direct communication provides better service

Just as direct communication between people is preferred for accuracy, computer-to-computer communication reduces the potential for errors and delays. When you purchase Amicus 1, you gain significant benefits. Faster collections and fewer errors. Greater productivity. And significant relief of cash flow pain.

Handles most of your accounting and financial management functions

Of course, Amicus 1 does more than just send claims to our computer. It assists you in all your billing and accounts receivable needs. It provides up to the minute financial reports on your practice. And more. All with just a few short entries.

A complete system featuring the IBM® Personal Computer XT

We've developed a ready-to-use system featuring an IBM Personal Computer XT. In addition, Amicus 1 includes a high speed printer; a tape backup storage unit; and a fast modem for telephone data transmission and access to medical and financial services. The Amicus 1 system includes installation with a three-day training program for your office staff. It also includes an on-site service contract plus a special telephone response line available during business hours.

Fill out your last claim form.

Just complete and mail the coupon below. Or, call us at **212 490-4180** to learn how Amicus 1 can help reduce your paperwork, increase your productivity and improve your cash flow.

Tell me how Amicus 1 can work for my practice.

☐ Yes, I'd like to see a personal demonstration.

☐ Please send me more information. NYSJM

1. NAME

2. SPECIALTY

3. ADDRESS *(Street, city, state, ZIP code)*

4. TELEPHONE ( )

5. OFFICE MANAGER

Send completed form to: **Medical Business Services**
P.O. Box 1707 Grand Central Station
New York, N.Y. 10163

Medical Business Services
Div. of Blue Cross® Blue Shield® of Greater New York

*Applies to the 16 counties where Blue Cross and Blue Shield of Greater N.Y. is the Medicare carrier
IBM® is a registered trademark of International Business Machines Corporation
Amicus 1™ is a trademark of Blue Cross and Blue Shield of Greater New York

A defense against cancer can be cooked up in your kitchen.

There is evidence that diet and cancer are related. Some foods may promote cancer, while others may protect you from it.

Foods related to lowering the risk of cancer of the larynx and esophagus all have high amounts of carotene, a form of Vitamin A which is in cantaloupes, peaches, broccoli, spinach, all dark green leafy vegetables, sweet potatoes, carrots, pumpkin, winter squash, and tomatoes, citrus fruits and brussels sprouts.

Foods that may help reduce the risk of gastrointestinal and respiratory tract cancer are cabbage, broccoli, brussels sprouts, kohlrabi, cauliflower.

Fruits, vegetables and whole-grain cereals such as oatmeal, bran and wheat may help lower the risk of colorectal cancer.

Foods high in fats, salt- or nitrite-cured foods such as ham, and fish and types of sausages smoked by traditional methods should be eaten in moderation.

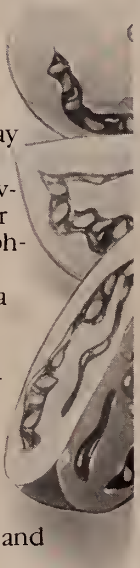
Be moderate in consumption of alcohol also.

A good rule of thumb is cut down on fat and don't be fat. Weight reduction may lower cancer risk. Our 12-year study of nearly a million Americans uncovered high cancer risks particularly among people 40% or more overweight.

Now, more than ever, we know you can cook up your own defense against cancer. So eat healthy and be healthy.

No one faces cancer alone.

AMERICAN CANCER SOCIETY®



JUNE 15, 1985
5th Annual Advances in Gastroenterology
Golden Nugget Hotel
Atlantic City, New Jersey

Sponsored by the Gastrointestinal Section of the Hospital of the University of Pennsylvania
and
the Continuing Medical Education Department of the Underwood Memorial Hospital, Woodbury, New Jersey

Category 1 credit offered

Information: Registration Supervisor, SLACK, Incorporated, 6900 Grove Road, Thorofare, New Jersey 08086, 609-848-1000.

ORTHOPEDIC SURGEON

Orthopod needed full time for my clinics in Rochester and Buffalo to perform Orthopedic Disability Evaluations.

Kick the hassle of practice, hospitals, etc. Enjoy banker's hours and maintain high income.

Write with CV to H. Lewis, M.D., 3641 Quail Hollow, Bloomfield Hills, Michigan, 48013.

PRACTICES AVAILABLE

INTERNAL MEDICINE practice for sale. Well-established in Nassau County. Home-office, fully equipped for one or two physicians. Telephone (516) 747-3072.

MEDICAL PRACTICES FOR SALE—Are you contemplating selling or buying a medical practice? We can help. PRACTICE FINDERS, INC. 45 East 89th Street, New York, NY 10028, Phone (212) 860-2206.

"Why your investments aren't as successful as you are."

Face it. Few problems are as complex as the management of your practice, money and taxes. And few health care practitioners have the extensive specialized expertise necessary for success in precisely these disciplines. Expertise which is vital to achieving financial growth, and, ultimately, independence.

As one of the health care practitioners who has been fortunate enough to gain these crucial financial skills, I'd like to share a few observations on why so many otherwise successful practitioners don't have similar success when it comes to their investments.

Strategies to make your earnings grow

A successful practice alone is not enough to guarantee your financial independence. That requires a careful balance of prudent, disciplined tax planning and professional management of your retirement assets.

Too many of you have relied far too much on the stock market...and paid the price for its unpredictability. Why assume such risk–when you can pursue a conservative, non-stock market approach that provides for a reliable, real rate of return? A diversified portfolio based on short-term guaranteed investment vehicles, as well as real estate investments designed specifically for retirement plans, can satisfy both your immediate and future financial needs.

Call in the right professionals now

Deciding how to invest isn't easy. Financial strategies present a complex picture–but the right professionals can help you focus in on the best solutions for you. I've compiled "25 Investment Pitfalls and How the Health Care Practitioner Can Avoid Them." Your phone call or coupon will bring these valuable insights free of charge–and start you on the path to making more of what you earn with the help of our team of qualified specialists.

So call me, Dr. Randy Sarantos, or my colleagues, Dr. David Raucher, or Jay Cruice, Certified Financial Planner, today. Whatever your concerns–practice evaluation, investment counseling, or any other aspect of personal financial planning–one firm does have the financial solutions for the health care practitioner...Sarantos & Company.

Sarantos & Company, Inc.
Registered investment advisors
800-223-0164 212-484-9759

"Total financial planning for the health care practitioner"

Mail To: Sarantos & Company, Inc.
733 Third Avenue, Suite 600
New York, NY 10017

☑ YES, send me my free copy of:
"25 Investment Pitfalls and How the Health Care Practitioner Can Avoid Them."

Also send me information on:
☐ Personal Financial Planning
☐ Investment Management for Retirement Plans

Name______________________________

Address______________________________

City____________ State________ Zip________

Phone: Office (   )________ Home (   )________

NYJM-385

THIRD ANNUAL CONFERENCE ON MATERNAL MORTALITY AND MORBIDITY of NEW YORK COUNTY MEDICAL SOCIETY

to be held

Wednesday, April 24, 1985 — 7 PM to 10 PM, NO FEE

at

The New York Academy of Medicine, 2 East 103rd St., NYC 10029

Albert Altchek, MD, Moderator
Chairman, Committee on Maternal Welfare, New York County Medical Society

PROGRAM

WELCOME: Hugh Clark Davidson, MD, President, New York County Medical Society

INTRODUCTION: David J. Sencer, MD, MPH, Commissioner of Health, City of New York

HISTORY OF MATERNAL MORTALITY STUDY IN NEW YORK: Bernard J. Pisani, MD,
Director Emeritus, St. Vincent's Hospital, New York City;
President, New York Academy of Medicine

MATERNAL MORTALITY IN NEW YORK CITY: Wendy Chavkin, MD, MPH,
Director, Maternity Services & Family Planning,
New York City Department of Health

Allan Rosenfield, MD, Moderator
Professor & Acting Chairman, Department of Obstetrics & Gynecology;
Director, Center for Population and Family Health
Columbia University College of Physicians & Surgeons

THIRTY YEARS OF MATERNAL MORTALITY IN MASS.: John Figgis Jewett, MD,
Clinical Professor of Obstetrics and Gynecology,
Emeritus, Harvard Medical School;
Chairman, Committee on Maternal Welfare, Mass. Medical Society

PANEL DISCUSSION: Previous Speakers &
Sally Faith Dorfman, MD, MSHSA, Reproductive Health Epidemiologist

Jean Pakter, MD, Consultant to the Commissioner of Health, NYC Department of Health;
Consultant in Maternal Child Health, Center for Population and Family Health

CONCLUSION: Albert Altchek, MD

This program is open without fee to members of the medical and allied professions. No advanced registration is required.

Why not enjoy a pleasant buffet supper before the conference from 5:45 PM to 6:45 PM. Invite a colleague or a resident. Buffet fee $10 per person—call the New York Academy of Medicine at 212-876-8200. Buffet open to members of all medical and allied professions.

This program is acceptable for TWO hours of Category I credit for the Physician's Recognition Award of the AMA. This meeting has been approved for 3 cognates, Formal Learning, by the American College of Obstetricians and Gynecologists.

CO-SPONSORS

- **New York Academy of Medicine, Section on Ob and Gyn**
  Harold Schulman, Chairman;
  Stanley Zinberg, Secretary
- **New York County Medical Society**
  Hugh Clark Davidson, President;
  Michael H. Grieco, Secretary
- **Medical Society of the State of New York**
  Allison Landolt, President;
  John H. Carter, Secretary
- **New York Gynecological Society**
  Kevin D. Reilly, President;
  David F. James, Secretary
- **American College of Obstetricians and Gynecologists**
  Richard H. Schwartz, Chairman District II; Gerson Weiss, Chairman Section I

FOR PHYSICIANS AND SURGEONS WITH A PART-TIME EXPOSURE ONLY

Now offering Medical Malpractice Insurance on an ***occurrence basis*** at drastically reduced rates thru Frontier Insurance Co., Monticello, NY.

With the tail on claims made policies, you might lose your shirt.

Call or write for further information and application.

MEDICAL MQ QUADRANGLE

Medical Quadrangle, Inc.
P. O. Box 456
318 Bedford Road
Bedford Hills, New York 10507
914 666-0170
212 829-3654
"Specializing in Medical Liability Insurance"

Index to Advertisers

Ayerst Laboratories 11A,12A,13A,14A,58A,59A,60A
Blue Cross/Blue Shield of Greater NY 49A
Classified Advertising ... 7A,18A,50A,53A,57A
Eastman Kodak 3A,4A,5A
Eli Lilly and Co. .... 2nd Cover
D. Baker Jacobs 48A
Marion Laboratories 1A,2A
Medical Quadrangle 53A
A.H. Robins 8A
Roche Laboratories .... 54A,55A,56A 3rd Cover, 4th Cover
Sarantos and Company 51A
Stuart Pharmaceuticals 15A,16A
Upjohn Company 10A
Wyeth Laboratories 17A,18A

NATIONAL EMERGENCY SERVICES, INC. (N.E.S.)

Directorships, full- and part-time emergency department opportunities are available throughout New York State and the country for primary care physicians with ER experience. Enjoy a variety of practice settings, competitive remuneration, paid malpractice insurance, and flexible scheduling. For information and application materials contact the recruitment department at (516) 365-4010, (Outside N.Y. State 1-800-645-4848) or send C.V. to N.E.S., One Hollow Lane, Suite 304, Lake Success, N.Y. 11042.

THE NEW JEWISH HOME OF ROCHESTER SEEKS A MEDICAL DIRECTOR

The new Jewish Home of Rochester, moving to a 362 bed Skilled Nursing and Health Related modern facility in June of 1985 in suburban Rochester, is looking for a physician licensed to practice medicine in New York State who seeks an opportunity to direct our medical activities.

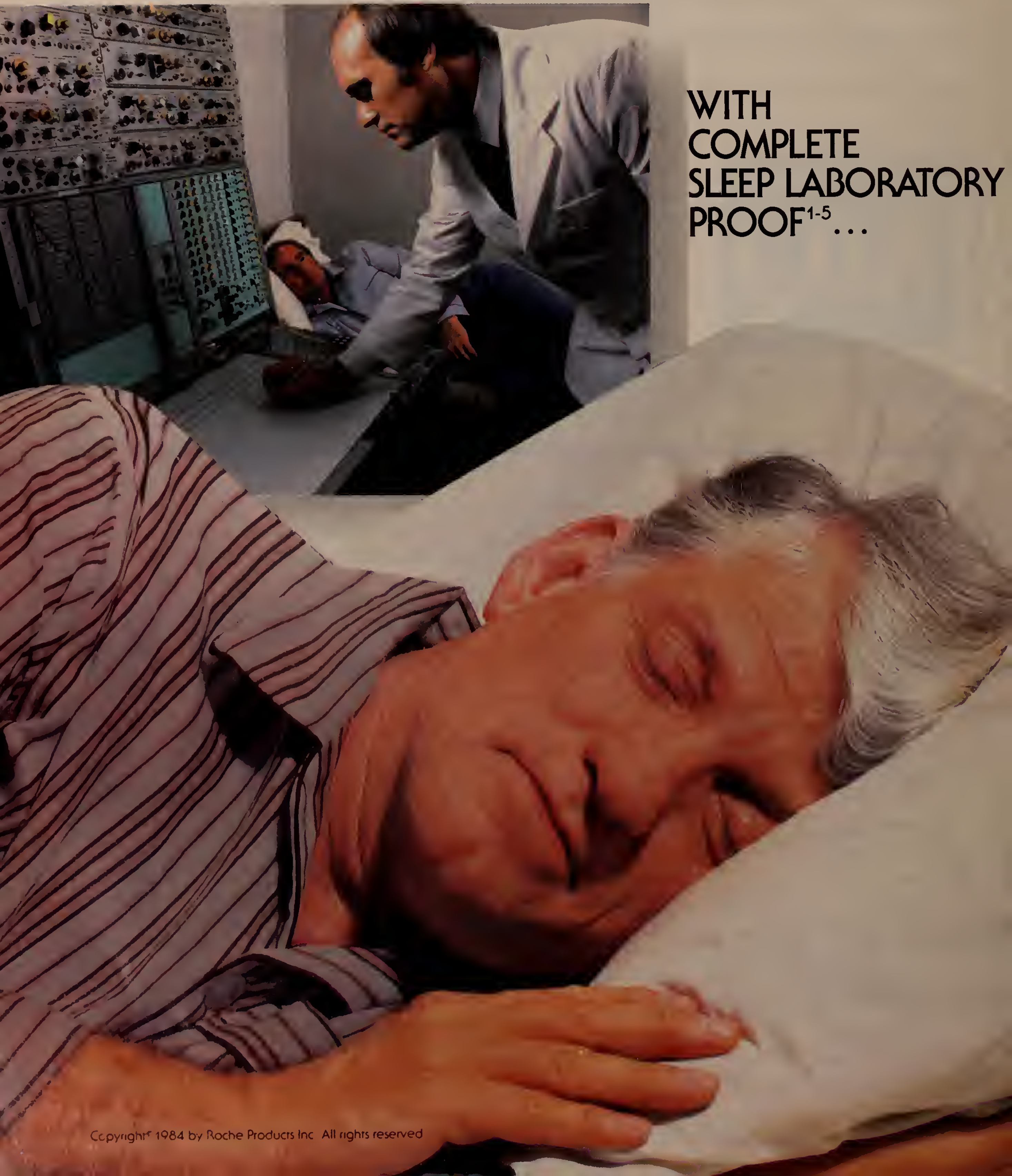
Applicants should have a keen interest in geriatric medicine with a vision toward a future teaching environment. A university appointment is anticipated.

Although full time is preferred, 25 hour applicants will be considered.

We offer an attractive salary and benefits package commensurate with experience.

Please send curriculum vita to: MEDICAL MATTERS COMMITTEE, ATTN: ADMINISTRATION, JEWISH HOME OF ROCHESTER, 1180 ST. PAUL STREET, ROCHESTER, NEW YORK 14621.

ONLY DALMANE®
flurazepam HCl/Roche
IMPROVES SLEEP...



WITH
COMPLETE
SLEEP LABORATORY
PROOF[1-5]...

Copyright© 1984 by Roche Products Inc All rights reserved

IN ALL THESE WAYS

- Rapid sleep onset[1-6]
- Effective for middle-of-the-night and early-morning awakenings[2,5]
- Undiminished efficacy for at least 28 nights[2-4]
- Patients usually awake rested and refreshed[7-9]
- Avoids rebound insomnia after discontinuation of therapy[3,5,10-12]

Caution patients about driving, operating hazardous machinery or drinking alcohol during therapy. Limit dose to 15 mg in elderly or debilitated patients. Contraindicated during pregnancy.

AND THE PREDICTABILITY THAT COMES WITH EXPERIENCE

FOR EFFECTIVE RELIEF OF INSOMNIA

DALMANE® ⓘⓥ

flurazepam HCl/Roche

STANDS APART

15-MG/30-MG CAPSULES

See next page for references and summary of product information.

References: **1.** Kales J *et al: Clin Pharmacol Ther* 12:691-697, Jul-Aug 1971. **2.** Kales A *et al: Clin Pharmacol Ther 18*:356-363, Sep 1975. **3.** Kales A *et al. Clin Pharmacol Ther 19* 576-583, May 1976. **4.** Kales A *et al: Clin Pharmacol Ther* 32:781-788, Dec 1982. **5.** Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc* 27:541-546, Dec 1979. **6.** Kales A, Kales JD: *J Clin Pharmacol* 3:140-150, Apr 1983. **7.** Monti JM: *Methods Find Exp Clin Pharmacol* 3:303-326, May 1981. **8.** Greenblatt DJ *et al: Sleep* 5 (Suppl 1):S18-S27, 1982. **9.** Kales A *et al: Pharmacology* 26:121-137, 1983. **10.** Greenblatt DJ, Allen MD, Shader RI: *Clin Pharmacol Ther 21*:355-361, Mar 1977. **11.** Zimmerman AM: *Curr Ther Res 13*:18-22, Jan 1971. **12.** Amrein R *et al: Drugs Exp Clin Res* 9(1):85-99, 1983.

Dalmane® Ⓒ
flurazepam HCl/Roche

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended. Repeated therapy should only be undertaken with appropriate patient evaluation.

Contraindications: Known hypersensitivity to flurazepam HCl; pregnancy. Benzodiazepines may cause fetal damage when administered during pregnancy. Several studies suggest an increased risk of congenital malformations associated with benzodiazepine use during the first trimester. Warn patients of the potential risks to the fetus should the possibility of becoming pregnant exist while receiving flurazepam. Instruct patient to discontinue drug prior to becoming pregnant. Consider the possibility of pregnancy prior to instituting therapy.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided, with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg recommended initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl

ROCHE®
Roche Products Inc.
Manati, Puerto Rico 00701

ONONDAGA

Apr 11–12. **Toward Quality Care—an Accreditation Standards Workshop for Ambulatory Health Care.** Syracuse. 10 Cat 1 credits. Info: JCAH, 875 N. Michigan Ave, Chicago, IL 60611.

ROCKLAND

Apr 26. **Interesting Non-Invasives.** Nyack. Info: Ms Myrna Hahn, Nyack Hospital, N. Midland Ave, Nyack, NY 10960. Tel: (914) 358-6200, ext. 2509.

SCHENECTADY

Apr 4. **Management of the Patient with Peripheral Vascular Disease and Amputee Rehabilitation.** Info: Barbara G. Post, Regional Medical Education Office, Sunnyview Hospital & Rehabilitation Center, 1270 Belmont Ave, Schenectady, NY 12308. Tel: (518) 382-4595.

AROUND THE NATION

ARIZONA

Apr 1–3. **Practical Cardiology for the Family Physician—1985.** *Apr 10–12.* **You're the Expert: Crucial Cardiac Decisions and the Nurse Specialist.** Info: American College of Cardiology, Extramural Programs Dept. 9111, Old Georgetown Rd, Bethesda, MD 20814. Tel: (301) 897-5400, ext. 230.

CALIFORNIA

Apr 12–14. **OB/GYN and Abdominal Sonography: Update '85.** San Francisco. Info: Dept of Radiology, University of California, San Francisco, CA 94143. Tel: (415) 666-5731.

Apr 14–19. **Fifth Advanced Workshop of Microsurgery in Gynecology.** *Apr 18–21.* **10th Annual Workshop of Basic Microsurgery in Gynecology.** University of California, Irvine. Info: Jordan M. Phillips, MD, 13021 East Florence Ave, Santa Fe Springs, CA 90670. Tel: (213) 862-8181.

Apr 18–20. **Allergy/Immunology for the Practicing Physician.** San Francisco. 18 Cat 1 credits. Info: Extended Programs in Medical Education, Room 569-U, University of California, San Francisco, CA 94143. Tel: (415) 666-4251.

Apr 27–28. **The Cutting Edge—1985: Clinical Applications of Kohut's Theories of Narcissism.** San Diego. 13 Cat 1 credits. Info: Office of CME, University of California San Diego School of Medicine, M-017, La Jolla, CA 92093. Tel: (619) 452-3940.

DISTRICT OF COLUMBIA

Apr 18–21. **Youth and Alcohol: Trends in Public Policies.** Omni Shoreham Hotel. Info: Forum Coordinator, National Council on Alcoholism, 12 West 21st St, New York, NY 10010. Tel: (212) 206-6770.

MARYLAND

Apr 15–17. **Current Applications of Echocardiography: M-Mode, Two-Dimensional and Doppler Techniques.** *Apr 25–27.* **Pediatric Electrocardiography, Electrophysiology and Pacing.** Info: American College of Cardiology Learning Center Registrar, 9111 Old Georgetown Rd, Bethesda, MD 20814. Tel: (301) 897-5400, ext 241.

VIRGINIA

Apr 19–21. **Emergency Medicine for the Primary Care Physician.** Williamsburg. 16½ Cat 1 credits. Info: Kathy Martin, CME, Box 48, MCV Station, Richmond, VA 23298. Tel: (804) 786-0494.

AROUND THE WORLD

BERMUDA

Apr 15–17. **The Hand Wound and its Treatment.** 18 Cat 1 credits. Info: American Society for Surgery of the Hand, 3025 South Parker Road, Suite 65, Aurora, CO 80014. Tel: (303) 755-4588.

PRACTICES AVAILABLE—CONT'D

SELLING PRACTICES IS OUR ONLY BUSINESS—Want to maximize the return on your investment? Before you buy or sell, call for a consultation to assure the best financial and transfer terms. We guide you through the entire sales process from initial meeting to closing. Serving 100 mile radius of New York City. Countrywide Business Brokerage, Inc., 319 East 24th Street, Suite 23-G, New York, NY 10010 (212) 686-7902.

FOR SALE LONG ESTABLISHED GENERAL PRACTICE with two story residence and attached office. Corner plot, separate entrances and addresses. Nassau County, New York, South Shore. Many extras, will introduce. Reply Dept. 380 c/o NYSJM.

FAMILY PRACTICE AVAILABLE WITH OFFICE BUILDING: Fully equipped. Located Ballston Spa, N.Y. near Albany, N.Y. Doctor retiring after 35 years. Information from Norman Fox, 404 Broadway, Saratoga Springs, N.Y. 12866. (518) 587-7778.

EQUIPMENT

PVR MACHINE FOR SALE—Life sciences pulse volume recorder, PVR IV, 2 years old, light usage, in excellent condition. Best offer. $18,000 new. Call (212) 549-3726 with offer or for further details.

REAL ESTATE FOR SALE OR RENT

MARCO ISLAND, FLORIDA. Two-bedroom condominium, eleventh floor; on Gulf. Beautifully decorated and furnished. For lease. Call (513) 891-1263.

WANTED: MEDICAL OFFICE BUILDINGS. As principals we are seeking "existing buildings"—fully tennanted by physicians, dentists and health care services. "To be built" medical facilities as a joint venture with either an institution or prominent area physician. Contact: Paul Gellert, Gelco Associates, 155 West 68th St., N.Y., N.Y. 10023; (212) 223-1130.

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

BRONX—Professional lobby apartment, highly desirable location. Reasonable sublet. Waiting room, 2 consulting rooms, (B&K). Ideal Psychotherapist. Call Mrs. Montagno (718) 894-7958.

OZONE PARK, QUEENS: Basement of busy Medical Building, stable community, for any medical related activity. 3,400 square feet available. Will subdivide to suit and consider any arrangements. Very favorable terms, great opportunity. Call (718) 848-6790 days or (718) 835-5259 nights and weekends.

REAL ESTATE FOR SALE OR RENT—CONT'D

LEASE FURNISHED OR UNFURNISHED MEDICAL OFFICE in a medical building in Eastchester, NY. Easily accessible to hospitals and parkways. Reasonable. Call (212) 792-5566.

FOREST HILLS, NEW YORK: Original owner, custom built office and home. Ten room house plus five and half room fully equipped office of former internist. Ideal location-accessible to subway and buses. Call after March 18th (718) 268-1255.

UPSTATE, NY. Are you doctor that would like a home adjacent to office on an acre in Manlius, NY (suburban Syracuse)? The house has 4 bedrooms, mulitple fireplaces, 2 car garage, gameroom. Office has 2 examining rooms, waiting room, office, ample parking. Retiring doctor. $105,000. Call Ellie Hayman, broker, (315) 446-8291. Gallinger Real Estate/Better Homes and Gardens, P.O. Box 66, Syracuse, NY 13209.

MISCELLANEOUS

JUNE 10-14, 1985, UPDATE YOUR MEDICINE 1985. Association of Practicing Physicians of the New York Hospital and Cornell University Medical College. 37 hours Category 1 credit, 49 if both optional workshops are taken. 9 major review lectures, 16 workshops, 2 Meet the Professor luncheons, Grand Rounds and optional practical workshops in breast/pelvic and male genitorectal examinations. Held at New York Hospital-Cornell, 1300 York Avenue at 69th Street, New York. Information: Office of CME, (212) 472-6119 or 5600. Dr. Lila Wallis, Course Director. Also call us for information about Update Your Medicine, Ithaca course July 22-26, 1985.

PATIENT NEWSLETTER: Show patients you care by keeping in touch! Send them our quarterly health promotion newsletter. As low as $1 per year. Customized with your name, address and optional office column. Mailing service. Free sample. RX: LIVE WELL, 6809 Santa Maria Lane, Dallas, Texas 75214-2847, or call (214) 821-5218.

ARCHITECTURE & PLANNING, Comprehensive services from analysis of alternative office locations through design and construction. Experienced in both new construction and renovation. Lewis & Gould (212) 807-6588.

TAX ACCOUNTANT-ATTORNEY, formerly with I.R.S., announces his availability to prepare taxes, accounting services, advise on tax shelters, pension plans and represent buyers or sellers of medical practices. I have other doctor clients. Gerald Goldberg, (212) 406-1156 and (516) 365-5679.

1985 CME CRUISE/CONFERENCES on Selected Medical Topics—Caribbean, Mexican, Hawaiian, Alaskan, Mediterranean. 7-14 days year-round. Approved for 20-24 CME Cat. 1 credits (AMA/PRA) & AAFP prescribed credit. Distinguished professors. Fly Roundtrip Free on Caribbean, Mexican & Alaskan Cruises. Excellent group fares on finest ships. Registration Limited. Pre-scheduled in compliance with present IRS requiremnets. Information: International Conferences, 189 Lodge Ave., Huntington Station, NY 11746. (516) 549-0869.

MISCELLANEOUS—CONT'D

DOCTOR, YOU CAN'T BEAT the quality or the price! Holter Monitor Scanning Service. Physician owned, trained and supervised. No contracts to sign. We can arrange for lease/purchase of Holter equipment. Why are you paying more and getting less? Now using UP Service for faster turn around time. DCG Interpretation (313) 879-8860.

TAX ATTORNEY AND PENSION PLAN Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professional incorporation—No insurance required—references upon request—Jack Wachstock, PC, 3000 Marcus Ave., Lake Success, N.Y. 11530 (516) 437-4848.

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate, with no points, fees or charges of any kind. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

MEDSTAR: THE CUSTOMIZED COMPUTER SYSTEM for medical office management. Designed by a physician and his staff. Completely customized for each practice. Affordable, Reliable, Easy to use. Leasing available. Medstar, 363 Route 111, Smithtown, NY 11787. (516) 360-1422.

ARCHITECTURAL DESIGN AND CONSTRUCTION: Designers, builders of custom professional offices. Consulting services available. Whether you're opening a new office, relocating, remodeling or expanding an existing office in New York City area and adjacent New Jersey counties. Donald Young, Inc. Tel: (718) 948-5191.

AVOID MALPRACTICE SUITS—Free 30 Day Trial "Guidelines For Medical and Surgical Emergencies". Write professional type orders for laboratory and treatment. Masson Publishing Company—U.S.A., 133 East 58th Street, New York, NY 10022.

EXPLORE FABULOUS AEGEAN SHORES: Hone sailing skills with English skipper on racy Rival 34. Martin Geller, M.D., 474 1st Street, Brooklyn, NY 11215 (718) 830-1344.

Angina Protection with Benefits for a Lifetime

ONCE-DAILY CONTROL WITH HEART-SAVING BENEFITS

By reducing heart rate and cardiac contractility, INDERAL LA helps protect the heart from the potentially serious and debilitating consequences of ischemia. A highly effective antianginal agent for around-the-clock control of symptoms, INDERAL LA also provides cardiovascular protection for a sense of security in the years ahead.

PROTECTION AND EXPERIENCE NO CALCIUM BLOCKER CAN MATCH

Unlike calcium blockers, INDERAL LA—either alone or with a nitrate—is recommended for early treatment of angina in the majority of patients. Equally important, INDERAL LA delivers the proven performance and safety profile of INDERAL tablets—confirmed by millions of patients during 16 years of clinical use.

START WITH 80 MG ONCE DAILY

Dosage may be increased to 160 mg once daily, as needed, to achieve optimal control. INDERAL LA should not be used in congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma. Please see next page for further details and brief summary of prescribing information.

ONCE-DAILY **INDERAL LA**
(PROPRANOLOL HCl) LONG ACTING CAPSULES

Ayerst.

The appearance of INDERAL LA capsules is a registered trademark of Ayerst Laboratories

JUST ONCE EACH DAY FOR SIMPLIFIED CORE THERAPY IN ANGINA

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl) *LONG ACTING CAPSULES*

Just once each day...

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*
INDERAL* LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules.

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain. Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use. Effects on plasma volume appear to be minor and somewhat variable. INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction. Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential. The significance of the membrane action in the treatment of arrhythmias is uncertain.

The mechanism of the antimigraine effect of propranolol has not been established. Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient. But there are also situations in which sympathetic stimulation is vital. For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction. Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

Propranolol is not significantly dialyzable

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock; 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)— PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g., dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA: Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General:* Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL is not indicated for the treatment of hypertensive emergencies.

Beta adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure.

Clinical Laboratory Tests. Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy: Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose

There are no adequate and well-controlled studies in pregnant women INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman

Pediatric Use Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular bradycardia, congestive heart failure, intensification of AV block, hypotension, paresthesia of hands; thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type

Central Nervous System. lightheadedness, mental depression manifested by insomnia, lassitude, weakness, fatigue, reversible mental depression progressing to catatonia, visual disturbances; hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics

Gastrointestinal. nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory bronchospasm

Hematologic: agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous: alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval.

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established.

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

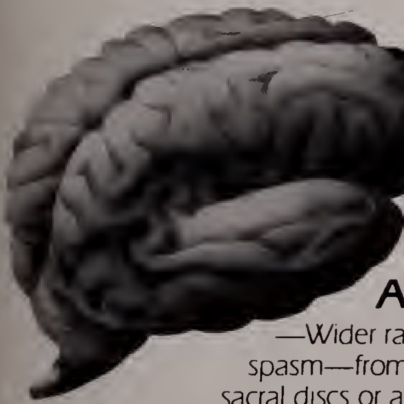
*The appearance of INDERAL LA capsules is a registered trademark of Ayerst Laboratories.

8833/384

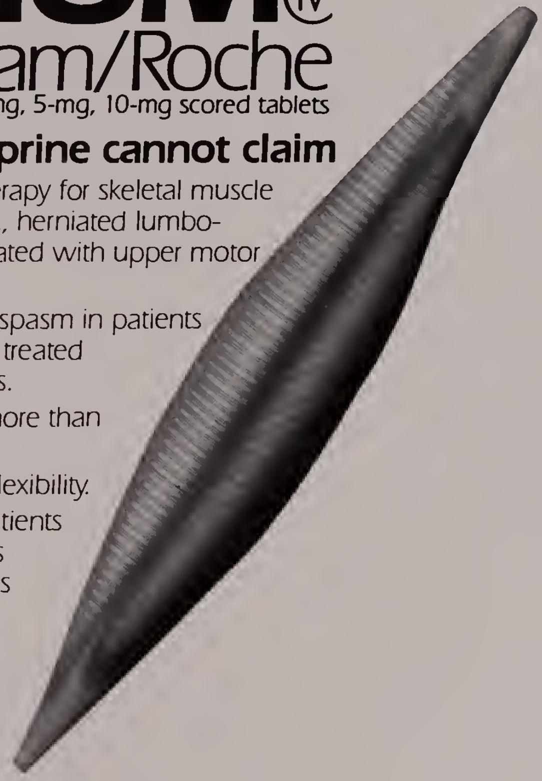
Ayerst.
AYERST LABORATORIES
New York, N.Y. 10017

THE SPASM/PAIN/SPASM CYCLE

In skeletal muscle spasm due to local pathology, responsive to the distinct actions of



Adjunctive
VALIUM® ⓘⓥ
diazepam/Roche
2-mg, 5-mg, 10-mg scored tablets



Advantages cyclobenzaprine cannot claim

—Wider range of indications as adjunctive therapy for skeletal muscle spasm—from spasm due to local pathology (*e.g.,* herniated lumbosacral discs or acute muscle strain) to spasm associated with upper motor neuron disorders (*e.g.,* cerebral palsy and paraplegia).

—May be used adjunctively for relieving skeletal muscle spasm in patients with hyperthyroidism, cardiac patients and patients being treated with anticholinergics or guanethidine-type antihypertensives.

—Can be administered to patients less than 15 years and more than 6 months of age.

—Scored tablets in three strengths provide greater dosage flexibility.

Since drowsiness, fatigue and ataxia sometimes occur, patients should be cautioned against driving or operating hazardous machinery and should also be advised against simultaneous ingestion of alcohol.

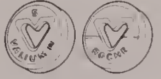
References: 1. Rankin EA: *Contin Educ 3*(1):46-50, Jan 1975. **2.** When muscle spasm hobbles your patient. *Patient Care 8*(11):20-37, Jun 1, 1974.

Valium® (diazepam/Roche) ⓘⓥ
Before prescribing, please consult complete product information, a summary of which follows:
Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic Symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal, adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not as sole therapy)
The effectiveness of Valium in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies The physician should periodically reassess the usefulness of the drug for the individual patient
Contraindicated: Known hypersensitivity to the drug Children under 6 months of age Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy
Warnings: Not of value in psychotic patients Caution against hazardous occupations requiring complete mental alertness When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication, abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures Advise against simultaneous ingestion of alcohol and other CNS depressants Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months After extended therapy gradually taper dosage Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

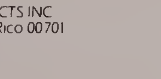
Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed, drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action Usual precautions indicated in patients severely depressed or with latent depression, or with suicidal tendencies Observe usual precautions in impaired renal or hepatic function Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation
The clearance of Valium and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration. The clinical significance of this is unclear
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported, should these occur, discontinue drug Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy
Dosage: Individualize for maximum beneficial effect *Adults* Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d to q i d alcoholism, 10 mg t.i.d. or q i.d in first 24 hours, then 5 mg t.i.d. or q i d as needed, adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d or q.i.d., adjunctively in convulsive disorders, 2 to 10 mg b.i.d to q i.d. *Geriatric or debilitated patients* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated (See Precautions) *Children* 1 to 2½ mg t.i.d. or q i.d initially, increasing as needed and tolerated (not for use under 6 months).
How Supplied: For oral administration, round, scored tablets with a cut out "V" design—2 mg, white, 5 mg, yellow, 10 mg, blue— bottles of 100 and 500, Prescription Paks of 50, available in trays of 10. Tel-E-Dose® packages of 100, available in boxes of 4 reverse-numbered cards of 25, and in boxes containing 10 strips of 10

Imprint on tablets
2 mg—2 VALIUM® (front)
ROCHE (scored side)

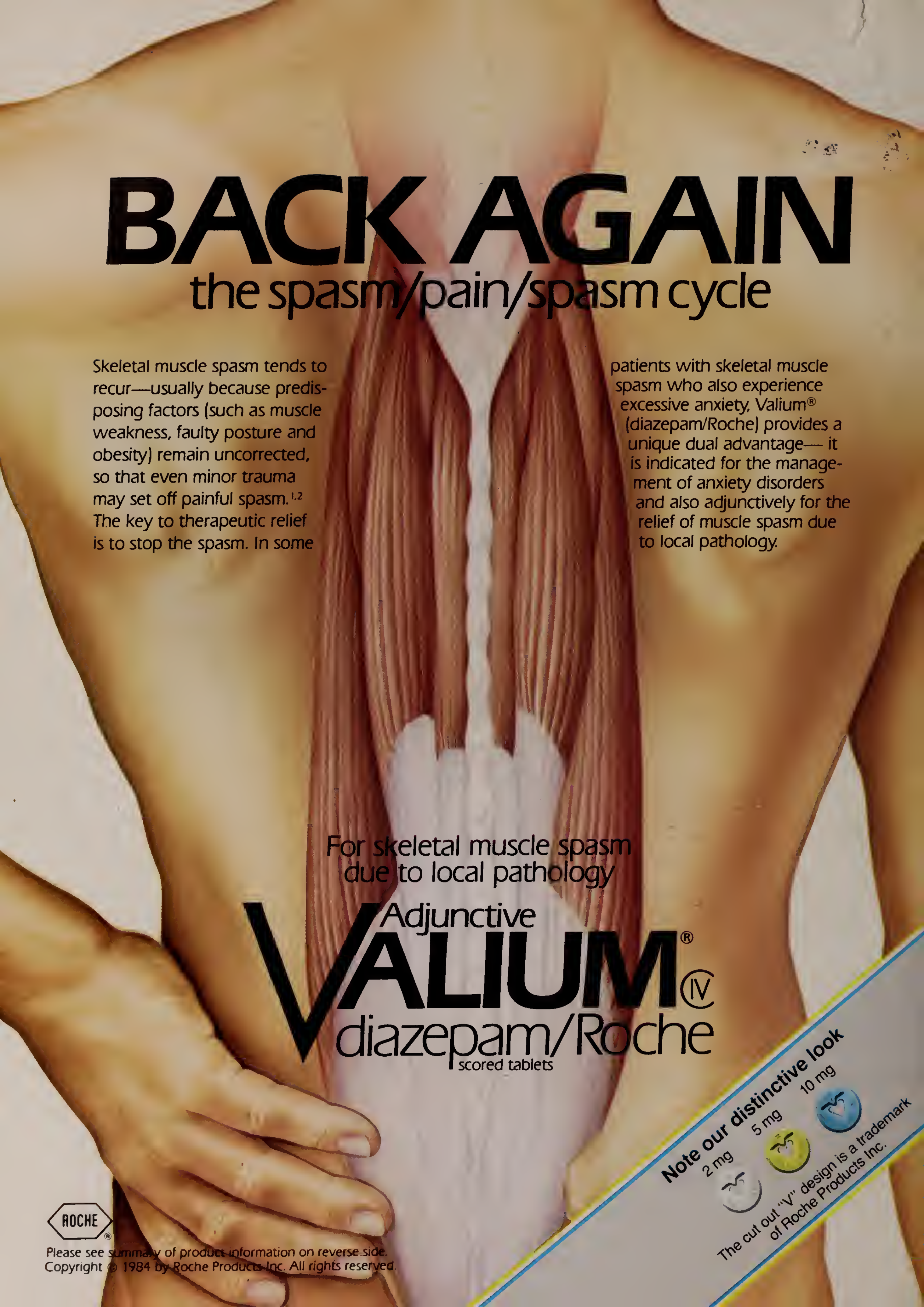


5 mg—5 VALIUM® (front)
ROCHE (scored side)

10 mg—10 VALIUM® (front)
ROCHE (scored side)



ROCHE®
ROCHE PRODUCTS INC
Manati, Puerto Rico 00701



BACK AGAIN

the spasm/pain/spasm cycle

Skeletal muscle spasm tends to recur—usually because predisposing factors (such as muscle weakness, faulty posture and obesity) remain uncorrected, so that even minor trauma may set off painful spasm.[1,2] The key to therapeutic relief is to stop the spasm. In some patients with skeletal muscle spasm who also experience excessive anxiety, Valium® (diazepam/Roche) provides a unique dual advantage— it is indicated for the management of anxiety disorders and also adjunctively for the relief of muscle spasm due to local pathology.

For skeletal muscle spasm due to local pathology

Adjunctive
VALIUM® Ⓘⓥ
diazepam/Roche
scored tablets

The cut out "V" design is a trademark of Roche Products Inc.

ROCHE®

Please see summary of product information on reverse side.

Copyright © 1984 by Roche Products Inc. All rights reserved.

STACKS

Annual CME Assembly, June 7–9, New York Hilton Hotel

(See March issue for Preliminary Program)

NEW YORK STATE JOURNAL OF MEDICINE

APRIL 1985 • Volume 85, Number 4

MEDICAL SOCIETY OF THE STATE OF NEW YORK ORGANIZED 1807

MINORITIES AND MEDICINE

Contents

Racial equality in the American health care system: an unfulfilled ideal 125
ANDREW D. McBRIDE, MD

Legal and administrative barriers to health care 126
BETH J. LIEF, ESQ

Maternal and child health care: A deepening crisis 128
MARIAN WRIGHT EDELMAN

A comparison of selected health indicators for black and white children in New York State 131
LINDA A. RANDOLPH, MD, SYLVIA RIVERS, RN EdD

The restraint of progress: Declining participation of blacks and other minorities in medical schools 135
LEON JOHNSON, Jr, DEd

Black-Jewish relationships: access to health services and health careers 137
SAMUEL WOLFE, MD, DrPH

Inequality in the availability of black physicians 139
DAVID McBRIDE, PhD

Eugene Percy Roberts, MD: Black physician and leader 143
DENNIS C. DICKERSON, PhD

The black woman physician 145
NORMA J. GOODWIN, MD

Minorities and New York medical schools: Past failures and the prospect for future successes 148
ROBERT K. WATTS, MSW, JAMES J. McCORMACK, PhD

Black doctors on television 151
J. FRED MacDONALD, PhD

The status of Asian Americans in the health care delivery system in New York 153
SETSUKO MATSUNAGA NISHI, PhD, CHARLES PEI WANG, MSW

Gastrointestinal malignancies in black Americans 157
WILLIAM McBRIDE, MD, GARY C. RICHTER, MD

Hypertension in blacks: Is it a different disease? 160
LEONARD G. MEGGS, MD

Sarcoidosis: focus on the black patient 162
RONALD P. DANIELE, MD

Coronary heart disease in black Americans 164
JAY BROWN, MD

BOOK REVIEWS 167

HOUSE OF DELEGATES 171

Sessions; Officers, Councilors, and Trustees; Members; Reference and Nominating Committees; Report of the Committee on Bylaws

MEDICAL MEETINGS AND LECTURES 8A

Healthy behavior is not a question of individual versus government responsibility. It is a natural outgrowth of the interaction between an individual's outlook on life, his self-esteem, and his opportunities, and government's role as a facilitator.

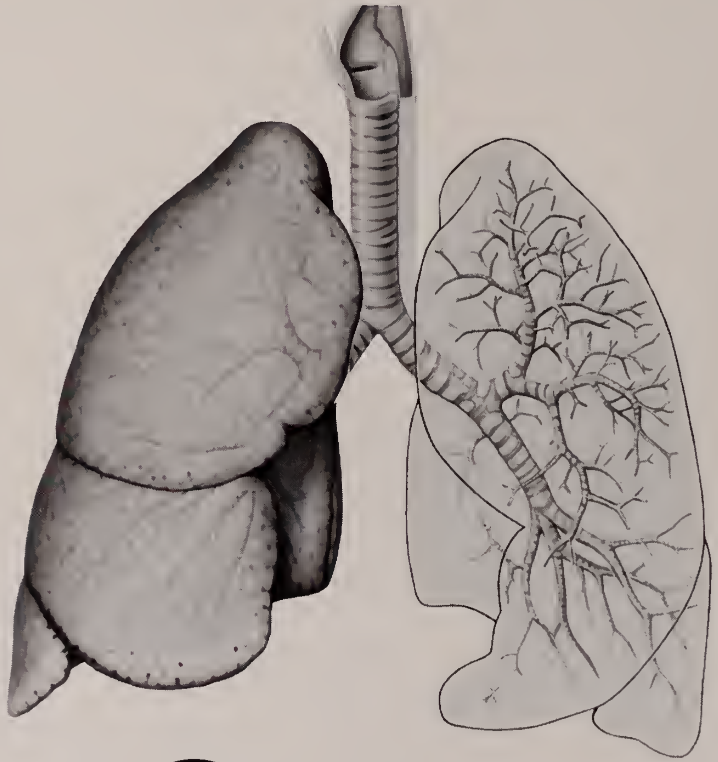
Individuals have a personal responsibility to practice good health habits; however, it is society's responsibility to provide the social and economic conditions, as well as the access to medical care that result in a healthy people.

M.D.S.

LIBRARY OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA
APR 22 1985

Library of the College Physicia
of Philadelphia Dec. 85
19 S. 22nd St.
Phila, PA 19103

Consider the causative organisms...



Ceclor®
cefaclor
250-mg Pulvules® t.i.d.

offers effectiveness against the major causes of bacterial bronchitis

H. influenzae, *H. influenzae*, *S. pneumoniae*, *S. pyogenes*
(ampicillin-susceptible) (ampicillin-resistant)

Brief Summary Consult the package literature for prescribing information

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae* and *S pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor

Contraindication Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

Warnings IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES

Antibiotics including Ceclor should be administered cautiously to any patient who has demonstrated some form of allergy particularly to drugs

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins and cephalosporins), therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics Such colitis may range in severity from mild to life-threatening

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone In moderate to severe cases management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by C *difficile* Other causes of colitis should be ruled out

Precautions: *General Precautions* – If an allergic reaction to Ceclor® (cefaclor, Lilly) occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e g , pressor amines, antihistamines, or corticosteroids

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms Careful observation of the patient is essential If superinfection occurs during therapy, appropriate measures should be taken

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

Ceclor should be administered with caution in the presence of markedly impaired renal function Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly)

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis

Usage in Pregnancy – Pregnancy Category B – Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor® (cefaclor, Lilly) There are, however, no adequate and well-controlled studies in pregnant women Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers – Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses Average levels were 0 18, 0 20, 0 21, and 0 16 mcg/ml at two, three, four, and five hours respectively Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor is administered to a nursing woman.

Usage in Children – Safety and effectiveness of this product for use in infants less than one month of age have not been established

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70)

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment Nausea and vomiting have been reported rarely

Hypersensitivity reactions have been reported in about 1 5 percent of patients and include morbilliform eruptions (1 in 100) Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor Such reactions have been reported more frequently in children than in adults Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy No serious sequelae have been reported Antihistamines and corticosteroids appear to enhance resolution of the syndrome

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain – Transitory abnormalities in clinical laboratory test results have been reported Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician

Hepatic – Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40)

Hematopoietic – Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40)

Renal – Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200)

[061782R]

Note Ceclor® (cefaclor, Lilly) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever See prescribing information

© 1984, ELI LILLY AND COMPANY

Lilly

Additional information available to
the profession on request from
Eli Lilly and Company
Indianapolis, Indiana 46285

Eli Lilly Industries, Inc
Carolina Puerto Rico 00630

400441

NEW YORK STATE JOURNAL OF MEDICINE

LIBRARY, ARCHIVES, AND PUBLICATIONS COMMITTEE

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
ELIZA H. CALDWELL, MD
URSULA MATULONIS (Medical Student)
GERALD P. MURPHY, MD
RICHARD B. NOLAN, MD
CARL E. POCHEDLY, MD
JOHN T. PRIOR, MD

Editor	ALAN BLUM, MD
Deputy Editor and Book Review Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Managing Editor	VICKI GLASER
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Administrative Assistant	IMELDA MORRISSEY
Editorial Assistant	JEANNE CONNERY
Librarian	ELLA ABNEY
Library Assistant	ELEANOR BURNS
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1985

RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MARK C. HOMONOFF, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. Synergistic Media Sales, 50 East 42nd St., Suite 504, New York, N.Y. 10017. Telephone: (212) 682-2200.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

ALLISON B. LANDOLT, MD, Westchester	*President*
BERNARD J. PISANI, MD, New York	*Past-President*
DANIEL F. O'KEEFFE, MD, Warren	*President-Elect*
JOHN A. FINKBEINER, MD, New York	*Vice-President*
JOHN H. CARTER, MD, Albany	*Secretary*
GEORGE LIM, MD, Oneida	*Assistant Secretary*
CHARLES D. SHERMAN, JR, MD, Monroe	*Treasurer*
MORTON KURTZ, MD, Queens	*Assistant Treasurer*
JOHN L. CLOWE, MD, Schenectady	*Speaker*
CHARLES N. ASWAD, MD, Broome	*Vice-Speaker*

Councilors

Term Expires 1985	JAMES H. COSGRIFF, JR, MD, Erie SAMUEL M. GELFAND, MD, Nassau RICHARD A. HUGHES, MD, Warren ANTONIO F. LASORTE, MD, Broome
Term Expires 1986	ROBERT A. MAYERS, MD, Westchester THOMAS D. PEMRICK, MD, Rensselaer RALPH E. SCHLOSSMAN, MD, Queens STANLEY A. STECKLER, MD, Suffolk
Term Expires 1987	DAVID M. BENFORD, MD, New York DUANE M. CADY, MD, Onondaga JAMES M. FLANAGAN, MD, Wayne M. THEODORE TANENHAUS, MD, Brooklyn

Trustees

CARL GOLDMARK, JR, MD, New York, *Chairman*
G. REHMI DENTON, MD, Albany
RALPH M. SCHWARTZ, MD, New York
EDGAR P. BERRY, MD, New York
RICHARD D. EBERLE, MD, Onondaga
JOSEPH F. SHANAPHY, MD, Richmond
VICTOR J. TOFANY, MD, Monroe

Executive Vice-President	GEORGE J. LAWRENCE, JR, MD
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Associate	IRMA A. ERICKSON
Director, Division of Scientific Publications	ALAN BLUM, MD
Director, Division of Management Services	GEORGE FORREST, JR
Assistant Director, Division of Governmental Affairs	JERRY S. HOFFMAN, ESQ
Director, Division of Communications	EDWARD HYNES
Director, Division of Insurance and Membership Benefits	BERNARD M. JACKSON
Director, Division of Membership	EUNICE SKELLY
Director, Division of Finance	PHILIP G. KEATING
Asst. Director, Division of Scientific Activities	ELIZABETH DAEPP
Director, Division of Advertising-Sales and Meeting Services	ROBERT MILLER
General Counsel	DONALD R. MOY, JD
Director, Division of Computer Services	IVAN H. NAJMAN
Acting Director, Division of Medical Services	MARIO MENGHINI
Director, Division of Long-Range Planning	DEBORAH R. SIEGEL
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. Editorial material published in the *Journal* is covered by copyright. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

Angina Protection with Benefits for a Lifetime

ONCE-DAILY CONTROL WITH HEART-SAVING BENEFITS

By reducing heart rate and cardiac contractility, INDERAL LA helps protect the heart from the potentially serious and debilitating consequences of ischemia. A highly effective antianginal agent for around-the-clock control of symptoms, INDERAL LA also provides cardiovascular protection for a sense of security in the years ahead.

PROTECTION AND EXPERIENCE NO CALCIUM BLOCKER CAN MATCH

Unlike calcium blockers, INDERAL LA—either alone or with a nitrate—is recommended for early treatment of angina in the majority of patients. Equally important, INDERAL LA delivers the proven performance and safety profile of INDERAL tablets—confirmed by millions of patients during 16 years of clinical use.

START WITH 80 MG ONCE DAILY

Dosage may be increased to 160 mg once daily, as needed, to achieve optimal control. INDERAL LA should not be used in congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma. Please see next page for further details and brief summary of prescribing information.

ONCE-DAILY **INDERAL LA**
(PROPRANOLOL HCl) *LONG ACTING CAPSULES*

Ayerst.

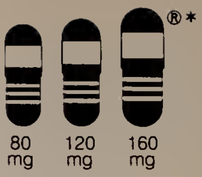
The appearance of INDERAL LA capsules is a registered trademark of Ayerst Laboratories

JUST ONCE EACH DAY FOR SIMPLIFIED CORE THERAPY IN ANGINA

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES



Just once each day...

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*
INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**
DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride. Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules.
CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially.

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

The mechanism of the antihypertensive effect of INDERAL has not been established. Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain. Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use. Effects on plasma volume appear to be minor and somewhat variable. INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients.

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction. Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period. The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity.

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential. The significance of the membrane action in the treatment of arrhythmias is uncertain.

The mechanism of the antimigraine effect of propranolol has not been established. Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient. But there are also situations in which sympathetic stimulation is vital. For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved. In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction. Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

Propranolol is not significantly dialyzable.
INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.
CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL.
WARNINGS. CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e.g., dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA: Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.
PRECAUTIONS. *General:* Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL is not indicated for the treatment of hypertensive emergencies.

Beta adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

Clinical Laboratory Tests: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Carcinogenesis, Mutagenesis, Impairment of Fertility. Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

Pregnancy: Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers: INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.
ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: bradycardia, congestive heart failure; intensification of AV block, hypotension; paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type.

Central Nervous System. lightheadedness, mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances, hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics.

Gastrointestinal. nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: bronchospasm.

Hematologic: agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.
DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily. If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained. INDERAL LA should not be considered a simple mg for mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels. Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval.

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved. The usual maintenance dosage is 120 to 160 mg once daily. In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

ANGINA PECTORIS—*Dosage must be individualized.* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained. Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily. In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established.

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS).

MIGRAINE—*Dosage must be individualized.* The initial oral dose is 80 mg INDERAL LA once daily. The usual effective dose range is 160-240 mg once daily. The dosage may be increased gradually to achieve optimum migraine prophylaxis. If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued. It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily.
PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

*The appearance of INDERAL LA capsules is a registered trademark of Ayerst Laboratories.
8833/384

Ayerst®
AYERST LABORATORIES
New York, N.Y. 10017

Classified Advertising

PHYSICIANS WANTED

DIRECTOR OF HEALTH SERVICES: Ithaca College, a private, coeducational institution of 5,300 students in the Finger Lakes region of New York State, offers academic programs in the liberal arts as well as several professional areas. The College is largely residential with approximately 3,500 students living on campus in a variety of living arrangements. Position requires a full time physician to administer the College Health Service. The Health Center is a primary care facility with laboratory and inpatient beds. In addition to the Director there is a second physician, three PA's, and additional nursing and support staff. Formal duties are during the two major academic semesters. A college maintained house is provided as the expected residence. Liberal fringe benefits. Malpractice insurance. Salary competitive. Position begins August 15, 1985. Applications will be accepted until the position is filled. Applications received by April 15 will be given first consideration. Please submit applications with resumes to Richard J. Correnti, Vice President for Student affairs, Ithaca College, Ithaca, New York 14850.

PHYSICIAN WANTED to share office space with an older general practitioner with intention of sale on retirement. Pleasant, rural community in Upstate New York. Office next to hospital. Terms negotiable. Call (315) 536-4456 or nights (315) 536-8084 or write H.A. Mikk, M.D., Med. Arts Bldg., Penn Yan, New York 14527.

ACTIVE GENERAL INTERNIST seeks board cert. or eligible internist. Modern, luxurious office equipped with Holter Monitor, automative lab partially computerized. Very desirable area Eastern L.I. 1½ hours from N.Y.C. Excellent boating, sailing, fishing, serenity. Salary, Malpractice, early partnership. Send CV to Dept. 379 c/o NYSJM.

MEDICAL CARE CLINIC in Sun City, Arizona, multi-speciality clinic seeks Neurologist, Gynecologist, Ophthalmologist, Internal Medicine, Gastroenterologist and Dermatologist. Area served has approximately 100,000 population, and three full-service hospitals. Retirement community surrounded by rapidly growing communities. Terms negotiable. Contact Barry Horst, 10448 W. Coggins Drive, Sun City, Arizona 85351, (602) 933-5457.

INTERNAL MEDICINE: Full-time opportunities available for board eligible or board certified physicians for busy inpatient internal medicine department of south Florida hospital. Clinic positions also available in Tampa, Palm Beach and Miami. Send C.V. to Fern Blum, EMSA, 8200 W. Sunrise Blvd., Building C, Plantation, Florida 33322 or call (305) 472-6922.

PHYSICIANS WANTED—CONT'D

PHYSICIANS needed for 75 residential children's camps located in Northeast. July and/or August. Contact: Association of Independent Camps (M), 60 Madison Ave., New York, NY 10010. (212) 679-3230.

BOARD CERTIFIED INTERNIST: Preventive & Occupational Health—a leader in field offers varied opportunities (full-time, half-time or per diem) at our clinics and corporate medical units. Send C.V. to Executive Health Examiner, 777 Third Ave., N.Y., N.Y. 10017, Attn: Personnel Director N. [EOE M/F].

SOUTHERN CALIFORNIA: Prestigious HMO is seeking experienced specialists and general practitioners for our facilities in Los Angeles and Orange Counties. Located in close proximity to major teaching centers, we offer the opportunity for continued professional development and rewarding clinical practice. Excellent compensation and benefits including paid malpractice, life, disability, medical and dental coverage, paid vacations, sick leave, educational leave and retirement plan. Please send C.V. to: Director/Physician Recruitment, CIGNA Healthplans of California, 700 N. Brand Blvd., Suite 500-38, Glendale, CA 91203.

EMERGENCY MEDICINE PHYSICIANS, FAMILY PRACTICE PHYSICIANS. New group located in New York State's Capitol District is seeking board certified/board eligible emergency medicine or family practice physicians. Independent contractor status, competitive salary and malpractice insurance. Excellent ground floor opportunity. Reply Dept. 384 c/o NYSJM.

PHYSICIANS WANTED—CONT'D

ASSISTANT DIRECTOR/M.D., our client a major pharmaceutical company is seeking a board eligible/certified physician with private practice or academia/research experience to test new drugs. Send C.V. to Bruce Rogers Co., Mgmt. Consultants, 20 Main Street, Port Washington, NY 11050, (516) 883-1058.

CERTIFIED OR BOARD ELIGIBLE ANESTHESIOLOGIST to join group of 8 at community hospital on Long Island, New York. Early full partnership. Include C.V. with first response to Dept. 383 c/o NYSJM.

DIRECTOR OF AMBULATORY CARE programs in Obstetrics-Gynecology. The position directs and oversees the policy development, planning process, program development, budgeting, operations, teaching activities and evaluations of staff performance of the ambulatory unit. Applicants must be certified by ABOG or Board eligible; willing and able to teach medical students and residents; clinically competent; interested in participation of direct patient care; and dedicated to the pursuit of scholarship. Academic rank and salary are negotiable. Replies held in confidence. Please address inquiries to: Henry A. Thiede, M.D., Chairman, Department of Obstetrics-Gynecology, Box 668, The University of Rochester, School of Medicine and Dentistry, 601 Elmwood Avenue, Rochester, New York 14642. An Equal Opportunity Employer (M/F).

THINGS WE CAN'T KEEP QUIET ABOUT.

When you're disabled, your world comes to a stop — and so does your income. But it doesn't have to be that way.

The Quiet Company is one of the few insurance companies that offers high quality individual disability income insurance. And no one offers broader coverage for partial disability than Northwestern Mutual Life.

For more information, please call:

STEPHEN G. SULTAN
75-22 196 Place
Fresh Meadows, NY 11366
718/479-8474

Northwestern Mutual Life®
The Quiet Company®
Quiet
Quiet
A tough act to follow

On nitrates, but angina still strikes...

After a nitrate, add ISOPTIN®

(verapamil HCl/Knoll)

To protect your patients, as well as their quality of life, add Isoptin instead of a beta blocker.

First, Isoptin not only reduces myocardial oxygen demand by reducing peripheral resistance, but also increases coronary perfusion by preventing coronary vasospasm and dilating coronary arteries — both normal and stenotic. These are antianginal actions that no beta blocker can provide.

Second, Isoptin spares patients the beta-blocker side effects that may compromise the quality of life.

With Isoptin, fatigue, bradycardia and mental depression are rare. Unlike beta blockers, Isoptin can safely be given to patients with asthma, COPD, diabetes or peripheral vascular disease. Serious adverse reactions with Isoptin are rare at recommended doses; the single most common side effect is constipation (6.3%).

Cardiovascular contraindications to the use of Isoptin are similar to those of beta blockers: severe left ventricular dysfunction, hypotension (systolic pressure <90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no artificial pacemaker is present) and second- or third-degree AV block.

So, the next time a nitrate is not enough, add Isoptin...for more comprehensive antianginal protection without side effects which may cramp an active life style.

ISOPTIN. Added antianginal protection without beta-blocker side effects.

Please see brief summary on following page.

ISOPTIN® TABLETS
(verapamil HCl/Knoll)
80 mg and 120 mg

Contraindications: Severe left ventricular dysfunction (see *Warnings*), hypotension (systolic pressure <90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block. **Warnings:** ISOPTIN should be avoided in patients with severe left ventricular dysfunction (e.g., ejection fraction <30%) or moderate to severe symptoms of cardiac failure. Control milder heart failure with optimum digitalization and/or diuretics before ISOPTIN is used. ISOPTIN may occasionally produce hypotension (usually asymptomatic, orthostatic, mild, and controlled by decrease in ISOPTIN dose). Occasional elevations of liver enzymes have been reported; patients receiving ISOPTIN should have liver enzymes monitored periodically. Patients with atrial flutter/fibrillation and an accessory AV pathway (e.g., W-P-W or L-G-L syndromes) may develop a very rapid ventricular response after receiving ISOPTIN (or digitalis). Treatment is usually D.C.-cardioversion. AV block may occur (3rd degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema, and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with ISOPTIN. **Precautions:** ISOPTIN should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Studies in a small number of patients suggest that concomitant use of ISOPTIN and beta blockers may be beneficial in patients with chronic stable angina. Combined therapy can also have adverse effects on cardiac function. Therefore, until further studies are completed, ISOPTIN should be used alone, if possible. If combined therapy is used, patients should be monitored closely. Combined therapy with ISOPTIN and propranolol should usually be avoided in patients with AV conduction abnormalities and/or depressed left ventricular function or in patients who have also recently received methyldopa. Chronic ISOPTIN treatment increases serum digoxin levels by 50% to 70% during the first week of therapy, which can result in digitalis toxicity. The digoxin dose should be reduced when ISOPTIN is given, and the patient carefully monitored. ISOPTIN may have an additive hypotensive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after ISOPTIN administration. Until further data are obtained, combined ISOPTIN and quinidine therapy in patients with hypertrophic cardiomyopathy should probably be avoided, since significant hypotension may result. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. *Pregnancy Category C:* There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. It is not known whether verapamil is excreted in breast milk; therefore, nursing should be discontinued during ISOPTIN use. **Adverse Reactions:** Hypotension (2.9%), peripheral edema (1.7%), AV block: 3rd degree (0.8%), bradycardia: HR<50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%). The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain: confusion, paresthesia, insomnia, somnolence, equilibrium disorders, blurred vision, syncope, muscle cramps, shakiness, claudication, hair loss, maculae, and spotty menstruation. Overall continuation rate of 94.5% in 1,166 patients. **How Supplied:** ISOPTIN (verapamil HCl) is supplied in 80 mg and 120 mg sugar-coated tablets. July 1982 2068

knoll® **KNOLL PHARMACEUTICAL COMPANY**
30 NORTH JEFFERSON ROAD, WHIPPANY, NEW JERSEY 07981

2195

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meetings and lectures unless the information is submitted at least two months prior to the event.*

MAY 1985

AROUND THE STATE

MANHATTAN

May 2–4. **The Medicolegal Autopsy.** 16 Cat 1 credits. *May 8–10.* **CT of the Spine.** 17 Cat 1 credits. *May 27, 28.* **Review of Basic Sciences in Urology.** 14 Cat 1 credits. Info: NYU Medical Center Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295

May 3. **Muscle Diseases—Update 1985.** *May 10.* **Pediatric Rehabilitation.** Info: Beverley R. Baptiste, Medical Education, Beth Israel Medical Center, 10 Nathan D. Perlman Pl, New York, NY 10003. Tel: (212) 420-2849.

May 9–10. **Medical Diseases of the Retina: Comprehensive Update.** 16 Cat 1 credits. Info: Lenox Hill Hospital, Department of Ophthalmology, 100 E 77th St, New York, NY 10021. Tel: (212) 794-4726.

May 13–17. **American College of Cardiology Consultants Course.** Mount Sinai Medical Center. Info: American College of Cardiology, Heart House, 9111 Old Georgetown Rd, Bethesda, MD 20014.

May 14. **Endocrine Pathology.** 2 Cat 1 credits. Info: Victor Tchertkoff, MD, Department of Pathology, Metropolitan Hospital Center, 1901 First Ave, New York, NY 10029. Tel: (212) 360-6253.

May 18. **Kerato-Refractive Surgical Update.** Info: Martha B. Klapp, Manhattan Eye, Ear & Throat Hospital, 210 E. 64th St, New York, NY 10021. Tel: (212) 605-3762.

May 18. **The Joel Hartley Memorial Symposium: Management of Emergen-**

Medical Society of the State of New York

Annual Meeting of the HOUSE OF DELEGATES

June 6-9, 1985

ANNUAL CME ASSEMBLY

HEALTH CARE TODAY

June 7-9, 1985

The New York Hilton
New York City

HIGHLIGHTS. . . . .
- 30 Sessions • Panel Discussions
- Meet the Professors
- Technical Exhibits
- President´s Dinner Dance

CLIP & MAIL

Director of Front Office Operations
The New York Hilton
1335 Avenue of the Americas
New York, N.Y. 10019

Phone: 212-586-7000

for hotel use only

NAME ______________________ TITLE ____________
FIRM ______________________ PHONE ____________
STREET ______________________
CITY/STATE/ZIP ______________________
REMARKS ______________________
(please print)

NOTE: Reservations must be received no later than May 16, 1985 and will be held only until 6 p.m. on day of arrival unless later hour is specified.

please print

ARRIVAL DATE ______________________
☐ 2-4 p.m. ☐ 4-6 p.m. ☐ 6-8 p.m. ☐ after 8 p.m.
DEPARTURE DATE ______________________
(checkout time is 1 p.m.)

HAVE A CREDIT CARD? You can register at our QUICK CHECK desk. If you wish to take advantage of this credit card PAYMENT GUARANTEED reservation, please complete the information below.

☐ AMERICAN EXPRESS
☐ DINNERS CLUB
☐ CARTE BLANCHE

Expiration Date ____________
____
Credit Card No.

Signature ______________________

MEDICAL SOCIETY OF THE STATE OF NEW YORK
Annual Meeting of the House of Delegates
June 6-9, 1985
Annual CME Assembly
June 7-9, 1985

Circle preferred rate:

Singles: $120 $135 $145
Double/Twin: $145 $160 $170
Executive Tower: $190, Single/Double
Suites: (P&1): $315 $350
Suites: (P&2): $425 $460

Rates subject to 8¼% N.Y. State sales tax and $2.00 per room per night N.Y.C. occupancy tax.
(If room at the rate requested is unavailable, one at the nearest available rate will be reserved.)

1985 ANNUAL CME ASSEMBLY

HEALTH CARE TODAY

Medical Society of the State of New York
New York Hilton, New York City

FRIDAY, JUNE 7

8:00 a.m. - 10:00 a.m.	Meet the Professor Breakfast Meetings (5)
10:00 a.m. - 1:00 p.m.	Cardiovascular Diseases, *Beekman Parlor* Pediatrics/Dermatology, *Sutton Parlor North* Obstetrics/Gynecology, *Sutton Parlor South* Physical Medicine & Rehabilitation, *Sutton Parlor Center* Neurosurgery, *Regent Parlor*
2:00 p.m. - 5:00 p.m.	Neurology, *Sutton Parlor Center* Medical Legal & Workers' Compensation/Occupational Medicine, *Beekman Parlor* Cancer Update, *Sutton Parlor North* Chest Diseases, *Sutton Parlor South* Computers in Medicine, *Regent Parlor*

SATURDAY, JUNE 8

10:00 a.m. - 1:00 p.m.	Ophthalmology (All day), *Sutton Parlor North* Urology/Pathology, *Sutton Parlor Center* Internal Medicine (All day), *Beekman Parlor* General Surgery/Orthopedic Surgery/Hand Surgery/Gastroenterology Anesthesiology/Radiology, *Sutton Parlor South* Psychiatry/Alcoholism (All day), *Regent Parlor*
2:00 p.m. - 5:00 p.m.	Ophthalmology, *Sutton Parlor North* Otolaryngology—Head and Neck Surgery, *Sutton Parlor Center* Emergency Medicine, *Sutton Parlor South* Internal Medicine, *Beekman Parlor* Psychiatry/Alcoholism, *Regent Parlor*

SUNDAY, JUNE 9

8:00 a.m. - 10:00 a.m.	Meet the Professor Breakfast Meetings (5)
10:00 a.m. - 1:00 p.m.	Allergy & Immunology, *Beekman Parlor* Women's Medical Society of New York State/Family Practice, *Sutton Parlor Center* The Physician as a Witness, *Sutton Parlor North*

* * *

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis.

* * *

Technical Exhibits

General Information

The Medical Society of the State of New York's 1985 Annual CME Assembly sessions and exhibits are open to all physicians, their guests, and the allied professions. The two and one-half days of meetings will provide the individual physician with a possible 18 credits out of a total of 93 Category 1, CME credit hours being offered. Over 135 outstanding speakers, including a number from out-of-state, will present papers at 30 sessions.

On Friday, June 7th and Sunday, June 9th, five sessions of Meet the Professor will be held simultaneously. These two-hour sessions will include a continental breakfast.

The theme of the CME sessions will be *"Health Care Today"*. Some highlights of this year's sessions are, the Developments in the Relationship between Diet and Cancer; Update on Pacemakers; Computer Applications in Clinical Medicine; the Physician as a Witness; and today's concepts of Chest Diseases diagnosis and management. Our program will offer the clinician new approaches in diagnosis and treatment of diseases in a wide range of medical specialties. Don't miss this opportunity to gain Category 1 credits over a weekend when you are free from office hours. This year there is no registration fee for member physicians which makes it an economical way to brush up on your skills. Professional representatives from more than 70 companies will be in the Rhinelander Gallery Exhibit Hall to discuss their products and services. Technical Exhibits will include drug products, clinical laboratories, office equipment, computer services, educational and financial services.

COURTESY N.Y CONVENTION & VISITORS BUREAU

PULITZER FOUNTAIN AT GRAND ARMY PLAZA

For the first time since 1979, the CME Assembly will be combined with the Annual Meeting of the House of Delegates, and the Auxiliary to the Medical Society.

SPECIAL EVENTS

Thursday, June 6: The Annual Dinner Dance will be held in the Trianon Ballroom of the New York Hilton, in honor of Allison B. Landolt, M.D., President, preceded by a reception in the Mercury Ballroom at 7:00 p.m. All attendees are welcome. Tickets should be ordered in advance from the Medical Society.

Friday, June 7 and Saturday June 8: A reception for physicians, the allied professions, and exhibitors will be held on both days in the Exhibit Hall, between 5:00 p.m. and 6:00 p.m., compliments of the Medical Society of the State of New York.

HOTEL RESERVATIONS

Physicians must make their own reservations with the New York Hilton no later than May 16, 1985. If making reservations by telephone, please mention that you are attending the Medical Society Convention for billing at the special rate.

REGISTRATION

CME Registration will take place in the Rhinelander Gallery Exhibit Hall, located on the second floor of the hotel and accessible by escalators from the lobby. Registration will take place at the following hours:

Friday, June 7th–
7:30 a.m.-5:00 p.m.
Saturday, June 8th–
8:00 a.m.-5:00 p.m.
Sunday, June 9th–
7:30 a.m.-12:30 p.m.

House of Delegates Registration will take place on the third floor, West Promenade, at the following hours: Wednesday, June 5 from 12 noon-7:30 p.m.; Thursday, June 6 from 8:30 a.m.-5:30 p.m.; Friday, June 7 from 8:30 a.m.-5:30 p.m.; Saturday, June 8 from 8:30 a.m.-5:30 p.m.; and Sunday, June 9 from 8:30 a.m.-12 noon.

CME MEETING HOURS

Friday, June 7th–
  Meet the Professor–
    8:00 a.m.-10:00 a.m.
  All other sessions–
    10:00 a.m.-1:00 p.m.
    2:00 p.m.-5:00 p.m.

Saturday, June 8th–
  All sessions–
    10:00 a.m.-1:00 p.m.
    2:00 p.m.-5:00 p.m.

Sunday, June 9th–
  Meet the Professor–
    8:00 a.m.-10:00 a.m.
  All other sessions–
    10:00 a.m.-1:00 p.m.

Advance Registration Form

Complete and return this form to the Medical Society address as indicated below.

1985 Annual CME Assembly, June 7–9

The New York Hilton, New York City

PLEASE PRINT

NAME ______________________________
Last *First*

Address ______________________________

City/State/Zip ______________________________

Medical Specialty ______________________________

REGISTRATION FEE

Please check category

☐ Non-Member Physician	$50.00
☐ Allied Profession	$10.00

Please check category

☐ Nurse
☐ Physician Assistant
☐ Dentist
☐ Lab Technician
☐ PT/OT
☐ Other (specify) ______________

NO FEE

☐ MSSNY Active Member
☐ MSSNY Life Member
☐ Resident/Intern/Fellow
☐ Medical Student
☐ Medical Assistant
☐ Speaker
☐ Spouse/Family
☐ News Media/Medical Writer

MEET THE PROFESSOR—Continental Breakfast Meetings.

Fee: $35.00 per session. Time: 8:00 a.m.-10:00 a.m. Please indicate session(s) attending. Each session is limited to 30 persons.

Friday, June 7
☐ Charles Hollander, M.D.
☐ Harold Itskovitz, M.D.
☐ Harold Lebovitz, M.D.
☐ Donald Louria, M.D.
☐ Carroll Leevy, M.D.

Sunday, June 9
☐ Arnold Aberman, M.D.
☐ Michael Grieco, M.D.
☐ Thomas Kantor, M.D.
☐ Simon Pollack, M.D.
☐ Anthony Rebuck, M.D.

(see this issue for subjects to be presented by each Professor)

REMITTANCE

Registration Fee	$ ______________
Meet the Professor session(s)	$ ______________
TOTAL	$ ______________

Please make check payable to:
Medical Society of the State of New York, and return this form with remittance to: Division of Meeting Services, MSSNY, 420 Lakeville RD., P.O. Box 5404, Lake Success, N.Y. 11042

Important: Your ID Badge will be held at the MSSNY Registration Desk located in the Exhibit Hall, on the second floor of the New York Hilton.

Meet the Professor – June 7th and 9th

at the Medical Society of the State of New York's Annual CME Assembly, June 7-9, 1985, the New York Hilton, New York City. Five continental breakfast meetings, 2 hours each, will be offered on Friday and Sunday mornings from 8:00 a.m.-10:00 a.m. Sessions are limited to 30 attendees each, and there is a $35.00 fee. Please see the advance registration form in this section.

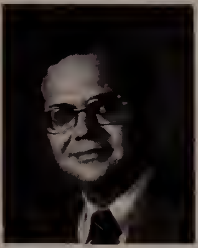
Hollander

Itskovitz

Lebovitz

Louria

Leevy


Aberman

FRIDAY, JUNE 7

Charles Hollander, M.D.

Professor of Medicine & Chief of Endocrinology, New York University School of Medicine, New York City. To provide a very brief review of our understanding of the physiology and pathophysiology of thyroid, pituitary and hypothalamic disorders.

Harold D. Itskovitz, M.D.

Professor of Medicine & Pharmacology, New York Medical College, Valhalla, New York. To familiarize the physician with new drugs used to treat hypertension; to stress changing concepts concerned with etiology, work-up and management of hypertension.

Harold E. Lebovitz, M.D.

Chief, Endrocrinology & Metabolism, SUNY, Downstate Medical Center, Brooklyn, New York. To provide an understanding of the pathophysiology of the two major types of diabetes, and to develop a rational set of goals for the treatment of patients with diabetes.

Donald Louria, M.D.

Professor & Chairman, Department of Preventive Medicine & Community Health, New Jersey Medical School, College of Medicine & Dentistry of New Jersey, Newark, New Jersey. To develop a programatic approach to the seriously ill patient with a putative infection.

Carroll Leevy, M.D.

Professor & Chairman, Department of Medicine, New Jersey Medical School, College of Medicine & Dentistry of New Jersey, Newark, New Jersey. Immunologic abnormalities are key factors in pathogenesis of hepatitus due to infections, alcoholism, and drug induced toxicity. Prevention, diagnosis and treatment have been greatly facilitated by recent advances in genetics, availabilty of monoclonal antibodies and the use of recombinant D.N.D. (Deoxyribonucleic Acid) technology.

SUNDAY, JUNE 9

Arnold Aberman, M.D.

Chief of Medicine, Mt. Sinai Hospital, University of Toronto Faculty of Medicine, Toronto, Canada. To approach the fluid and electrolyte management in the hospitalized patient with particular emphasis on hypo and hypernatremia.

Continued on next page

Meet the Professor - continued

Michael H. Grieco, M.D.

Director, R.A. Cooke Institute of Allergy, St. Luke's-Roosevelt Hospital Center, New York City. A review of current concepts concerning the immunologic and nonimmunologic mechanisms involved on pathogenesis of bronchial asthma, and review the management of ambulatory asthmatics.

Grieco

Kantor

Thomas Kantor, M.D.

Professor of Medicine; Chief, Division of Rheumatology, New York University School of Medicine, New York City. This session will demonstrate the all-purpose analgetic activity of the nonsteroidal, anti-inflammatory drugs in addition to their antiarthritic activity.

Simon Pollack, M.D.

Professor of Medicine, Albert Einstein College of Medicine, Bronx, New York. The objective of this presentation will be to explain current concepts of iron absorption and utilization, and the diagnosis and treatment of iron deficiency and iron overload.

Pollack

Rebuck

Anthony Rebuck, M.D.

Professor of Medicine, University of Toronto Faculty of Medicine, Toronto, Canada. Physicians attending this session will be made familiar with the proper use of oxygen in the every day care of caridac and pulmonary patients.

NEW YORK STATE
JOURNAL OF MEDICINE

April 1985 • Volume 85, Number 4

Racial equality in the American health care system: An unfulfilled ideal

In 1967, a black medical student at the University of Pennsylvania wrote an article in the rogue school newspaper, *Itis*, entitled, "The bedside manner—or how to pretend that you like the niggers on the ward." I was that student.

I do not recall being fearful of the physical and professional reprisals that my activism invariably precipitated. However, I always harbored the dread that I would turn out to be like one of the people in positions of authority with whom I so differed. Very simply, I wanted to be a person who neither condoned wrongdoing nor denied responsibility for solving problems.

My youthful idealism has not dimmed, but it has been tempered by the experience of trying to force, guide, or encourage change in a reluctant system. If anything, the years have increased my impatience with the slow pace of progress. This issue of the *Journal* has provided an opportunity for numerous authors and myself to reflect on aspects of health and minority populations in the United States.

Would that the news were good. Since the gains of the 1960s and early 1970s—many of which were more perceived than real—minority concerns have moved from the "back of the bus" to the "back burner" of America's conscience. During the past decade, national attention has been diverted to economic issues such as inflation, the price of oil, interest rates, and the budget deficit. Nonetheless, there are people who are still committed, by birth or personal choice, to redressing the existing racial inequity that belies our stated ideals. This issue of the *Journal* is one means of keeping faith with that commitment. Some of the persistent problems and needs of minorities in medicine are touched upon by contributors, who are neither complacent nor cynical about the potential for change.

Throughout history, black Americans in all fields, including health care, have borne the brunt of racist behavior and its consequences. Other minorities have shared the misery visited upon blacks, but the most serious societal ascription has always been related to color. Although this issue focuses primarily on blacks, much of it applies to other racial minority groups.

The issue is the result of more than a year of work and discussions with Alan Blum, MD, the *Journal*'s editor, and various colleagues across the country, who are too numerous to list. I do, however, thank them. I would also like to thank David Axelrod, MD, New York State Commissioner of Health, for his support of the undertaking while I was on staff there. Midway between the planning and final work on the issue I left the New York State Department of Health to assume my present position in the District of Columbia. During this transition, Ms Audrey Harvey, who accompanied me from Albany to Washington, DC, gave me invaluable assistance in the development of this issue. I was encouraged to continue the work on the *Journal* by Mayor Marion Barry, who maintains an enlightened policy on professional involvement. The professional *Journal* staff that worked diligently to bring it together also deserves special recognition.

In January, I was saddened to learn of the death of William Chase, MD. At the time of his death, Dr Chase was working on revisions to an earlier submission, in which he expressed the concern that there may not be a future for blacks in medicine if institutional indifference and racial bias are allowed to continue.

Finally, if medicine is viewed as a patient, the results of an examination as well as a diagnosis are offered in the pages of this issue. Included are progress notes, but no simple prescription to ameliorate the symptoms or ease the pain of racism in medicine and its consequences in health care delivery. With this issue, the authors are making a specialty referral to some of the highest-trained people in Western civilization. Surely, people who are trained to simplify the complexities of multiple organic systems can use their art and science to bring about the life-affirming changes that are so desperately needed and passionately sought. Shall we try?

ANDREW D. McBRIDE, MD
Commissioner
Department of Public Health
of the District of Columbia
1875 Connecticut Ave, NW
Suite 825
Washington, DC 20009

Legal and administrative barriers to health care

BETH J. LIEF, ESQ

Despite the Supreme Court's recognition a decade ago that "medical care is . . . 'a basic necessity life,' "[1] two observations must be made: first, the health care system in this country is neither designed to address nor operated to provide equal access to minorities and the poor; and second, there is no constitutional, statutory, or regulatory "right" to health care. Intentional barriers to health care for minorities have largely disappeared, but practices remain that effectively restrict access for minorities and the poor—although they are not intended for this purpose. Yet minorities and the poor are not without protection. Racially discriminatory barriers to health care are illegal, and laws exist that seek to assure, albeit to a limited extent, that the poor receive health services. However, those charged with enforcing civil rights laws have failed to do so. The Reagan administration has not only virtually ceased enforcing laws that guarantee minorities equal access to health services, but has consistently worked to cut funding for health care. Despite concern for cost containment, health care costs continue to skyrocket. Unless these trends are reversed, minorities and the poor may see whatever "rights" to health care they now have diminished.

Many admissions policies of health care institutions have the intended or unintended effect of denying minorities health care. Hospitals that restrict admission to patients who have a private doctor on staff, and the failure of hospitals to assure that doctors with staff privileges accept Medicaid patients often create insurmountable barriers to access. Some hospitals refuse to treat emergency patients who lack third-party insurance, either "dumping" them at the doors of public hospitals or simply turning them away. When private for-profit companies acquire formerly public or voluntary hospitals, these practices often increase. When hospitals close in minority neighborhoods or relocate to other areas, an important source of primary care for the poor is lost, and even fewer health professionals remain to serve the community. Increased travel time and expense make it likely that poor black families defer obtaining medical care until the need is extreme. Finally, restrictive admissions policies of nursing homes have resulted in segregated and unequal care for the minority aged. A recent study of New York City nursing homes,[2] for example, found that minorities are vastly overrepresented in public, lower quality nursing homes. Nearly 50% of the city's minority nursing home residents live in 10.5% of the city's 153 nursing homes.

Theoretically, these and other restrictive practices are vulnerable to attack. Title VI of the Civil Rights Act of 1964[3] prohibits racial discrimination by recipients of federal funds. Because of their heavy reliance on Medicaid and Medicare, most health facilities are covered by this statute. The Supreme Court recently upheld Title VI regulations that prohibit policies and practices that result in discrimination against minorities.[4]

After World War II, in response to the lack of adequate numbers of hospital beds in parts of the country, Congress passed the Hill-Burton Act to finance hospital construction. Funds for construction and modernization of hospitals were allocated under the act (and subsequent legislation) from 1949 until the mid 1970s. Those facilities that received federal funds under the Hill-Burton Act are required to provide a reasonable volume of services to persons unable to pay ("uncompensated service" obligation); and to make services available to all persons residing in the facility's area ("community service" obligation). Although the uncompensated care obligation is limited in duration, the community service obligation is not. Community service regulations, effective since 1979, offer great promise for access previously denied to minorities. For example, Hill-Burton facilities must participate in Medicare and Medicaid programs, if eligible, and cannot deny emergency services to any person who resides in the facility's service area because the person is unable to pay. The rules also cover the practices of excluding persons without private physicians or because of advance deposit requirements.

Title VI has largely succeeded in ending blatant discrimination, although vestiges of such racism remain. The laws have not been used to any significant extent, however, to remove the subtle barriers to access that still exist. As far as these practices are concerned, private lawsuits under the Hill-Burton statute have achieved some success, but litigation under Title VI in the health area has been scant. Only one lawsuit has been filed to challenge nursing home discrimination, and only a few to attack discriminatory admissions practices. Where such litigation has been filed to challenge hospital relocations and closures the results have been mixed. Although advocates have been able to demonstrate that relocations and closures can have a disproportionate and adverse effect on access to health care for poor minorities, the courts have been convinced by the hospitals' defenses of budgetary constraints and modernization needs. Nevertheless, these cases have secured some gains—occasionally, major ones. Although many advocates believe that greater use of Title VI and the Hill-Burton assurances would remove or lower barriers to access and, as a result, relieve some of the burdens on public hospitals and nursing homes that do keep their doors open, litigation is slow and expensive. Unless the government begins to fulfill its obligation to investigate health care facilities on a national scale and to prosecute

---

Address correspondence to Ms Lief, Executive Director, Mayor's Commission on Special Education, 250 Broadway, Rm 1412, New York, NY 10007.

Ms Lief is a former assistant counsel for the NAACP Legal Defense and Education Fund. The opinions expressed in this article are the private ones of the author.

violations of the law, the rights secured by civil rights laws will remain unrealized.

The civil rights guarantees of Title VI have viability in the health planning area as well. The National Health Planning and Health Resources Development Act of 1974 empowers local and state health planning agencies to review applications for capital expenditures and new services provided by health facilities, and to review proposed federally funded projects. Title VI requires health planning agencies to consider the nature and extent of any barriers to equal access to health care in these reviews. However, although the federal government has recognized the civil rights obligations of these agencies, it has yet to issue specific guidelines. As a result, these agencies often approve applications with little consideration of how the projects will affect access for minorities and the poor.

Admittedly, barriers to care are not just the result of discrimination; economics and the lack of health professionals who serve minorities and the poor contribute substantially to the problem. Some states, such as New York, have sought to tackle this problem by instituting hospital reimbursement formulas that pay hospitals a portion of the cost of providing "uncompensated care." This trend is encouraging. Yet, at the same time, major cutbacks in Medicaid coverage have exacerbated the difficulty of securing health care for the poor. Geographical differentials in Medicaid, as well as other third-party reimbursement, draw physicians away from disadvantaged areas. Finding a legal basis to challenge such differentials, however, is difficult, and federal efforts to grapple with the nonavailability of physicians through loan programs such as those under the National Health Services Corps are insufficient. Those efforts that are made are ineffective, in part because the government has not enforced the obligations of persons who receive loans under these programs to practice in regions with health manpower shortages.

Without major changes in the law or revisions in the health care delivery system, minorities and the poor will find it more difficult to obtain health services than their more affluent, white counterparts. Yet, even without the statutory creation of more "rights" to health care, a more equitable distribution of fiscal and personnel resources, combined with the elimination of unnecessary barriers to hospital and other facilities, would go far toward insuring access to care for minorities and the poor.

REFERENCES

1. *Memorial Hospital v Maricopa County*, 415 US 250, 259 (1974).
2. "Racial Discrimination in New York City's Residential Health Care Facilities." New York, Friends and Relatives of Institutionalized Aged, Inc, January 1984.
3. 42 USC §2000d *et seq.*
4. *Guardians Assoc. v New York City Civil Service Commission*, 77 L.Ed.2d 866 (1983).

CIVIL RIGHTS GROUPS ACT ON INFANT MORTALITY

Frustrated by the absence of concentrated efforts to reduce high infant mortality rates among the poor, civil rights groups and women's, children's, and health organizations* are challenging the Reagan Administration to demonstrate leadership in addressing this critical problem. Appallingly high infant mortality rates in black and other low-income communities are not a recent phenomenon. Yet, despite the existence of a known workable solution—making comprehensive prenatal care available to high-risk women—neither government nor the medical community has put its full influence behind ensuring access to care.

To fill this void, civil rights and other organizations, represented by Public Advocates, a public interest law firm based in San Francisco, filed an Administrative Petition with the United States Department of Health and Human Services in June 1983. The petition attempted to focus national attention on high infant mortality rates among blacks and the poor and proposed solutions, which could be administered through the existing Medicaid program, to reduce the incidence of low birth weight (infants weighing less than 5½ pounds at birth) and resultant infant mortality. Specifically, petitioners proposed that the Department of Health and Human Services establish a national standard for comprehensive prenatal care which would help reduce the inequities in care provided in different areas of the country; coordinate existing federal programs aimed at maternal and infant care; develop and fund an educational campaign that would make the need for prenatal care as well known as the phrases, "Only you can prevent forest fires" and "Be all that you can be"—both campaigns launched by the federal government to advance issues that it deems important. Additionally, the petition sought leadership from the Department of Health and Human Services in encouraging states to expand their Title V and Medicaid programs so that comprehensive prenatal care is provided to all low-income families.

The 150 page petition also included an Infant Mortality Survey, conducted in conjunction with the University of California School of Public Health at Berkeley . . . . This survey was necessary because in early 1983 the National Center for Health Statistics did not have infant mortality data by race available after 1979. This absence of information concerning such a vital indicator of the nation's health was particularly disturbing to petitioners given this country's commitment to keeping its finger on the pulse of major economic indicators by providing weekly reports of even minor fluctuations in prime interest rates or housing starts.

Not surprisingly, the Infant Mortality Survey revealed that the mortality rate for black infants before their first birthdays was twice that of white infants, and that gap is widening.

ANGELA GLOVER BLACKWELL, JD
Public Advocates, Inc
1535 Mission St
San Francisco, CA 94103

Maternal and child health care: a deepening crisis

MARIAN WRIGHT EDELMAN

For the past several years (indeed, since the enactment of Medicare and Medicaid in 1965), federal and state policymakers have viewed health system overspending as the major priority in shaping federal health policy. In fact, since 1965, except for enacting cost cutting measures, Congress has left essentially untouched the patchwork of federal health financing programs created as part of the Great Society. Moreover, since 1980, the Reagan Administration has intensified the drive to reduce public health expenditures for the elderly, the disabled, and the poor.

Between 1978 and 1982, per capita federal aid to states and localities fell by 25%. Between 1980 and 1982 alone, federal aid to states was reduced by 20%.[1] The effects of these cutbacks were felt in discretionary health programs (such as the Community and Migrant Health programs and Title V Maternal and Child Health Block Grant program), which were reduced by nearly 25%. Medicaid, the largest public health program for children, was cut by $5.5 billion between 1981 and 1983. Nearly 700,000 children lost Medicaid eligibility outright as a result of the cuts, and hundreds of thousands more have been prevented from gaining eligibility because of new restrictive program criteria.

In the midst of the debate over reducing health expenditures—which can be expected to intensify, as concern over the solvency of the Medicare trust fund and the structural deficit continues to mount—a critical health problem involving underfunding and underutilization still remains. In America today, poor mothers and children are experiencing a severe lack of access to basic health services. One in every 20 pregnant women, and one in every 11 non-white pregnant women, receives little or no prenatal care.[3] Twenty-five percent of children living in poverty, a disproportionate percentage of whom are black, have not seen a physician within the past year.[3] Forty percent of poor children have never seen a dentist.[3]

Access to health services largely depends on possessing health insurance or sufficient amounts of disposable cash. For that reason, millions of poor children and women are unable to make use of pediatric and maternity care.[4] This problem is now approaching crisis proportions because of the unprecedented increase in the number of poor children and because of structural changes in both public and private health care financing programs that have reduced the availability of charity care services. This crisis will only deepen as further efforts to curtail health care spending are undertaken without simultaneously reforming the inequitable features of the American health care system.

RICH/POOR HEALTH GAP WIDENS

Since 1979, three million children (3,000 a day) have fallen into poverty.[5] This 31% increase in poverty among children represents the sharpest increase in child poverty since poverty statistics were first collected.[5] Nearly 40% of the poor in America are children, and children are almost 70% more likely to be poor than adults.[5] Poverty has struck minority children most severely. Nearly 50% of black children in America are poor.[5] Among non-white children living in a home with a female head of household, more than 70% are poor.[5]

Among children, poverty is strongly associated with reduced health status.[6] Poor children have 30% more restricted days of activity and lose 40% more school days because of illness.[6] Three to six times as many poor children are likely to be reported in fair to poor health, and poor children are 40%–50% more likely than non-poor children to be found to have a significant abnormality on physical examination by a physician.[6]

Mortality among poor children is significantly related to poverty.Neonatal mortality is 150% higher,[6] and infant mortality rates are 200% higher among poor babies.[6] Because non-white children are disproportionately poor, these disparities in health status are directly reflected in mortality rates for black infants, which are nearly twice as high as the death rates for white infants.

Mortality rates among poor children continue to remain high after the first year of life. Poor children are one and a half to three times more likely to die in childhood.[6] Moreover, perinatal problems have a greater impact and more sequelae in poor children.[6] Thus, poor children who survive infancy are at greater risk of a lifetime of handicaps and reduced productivity.

There are indications that during the past several years the health risks facing poor children have heightened. For example, babies born to mothers who receive little or no prenatal care are three times more likely to have a low birth weight; and low birth weight increases the risk of death 20 times.[7] Yet, after a 10-year period in which an increasing number of women began prenatal care early in pregnancy, since 1980 there has been a nationwide erosion in the percentage of women receiving early prenatal care and an increase in the percentage of women receiving little or no prenatal care.[5] The trend has been particularly severe among non-white women, whose babies were already at heightened health risk. In a national survey of birthrate statistics, 18 of 23 states reporting data by race showed an increase in the percentage of non-white women receiving little or no prenatal care. Nineteen of 23 states reported a decrease in the percentage of non-white women receiving prenatal care early in pregnancy.[5]

Similar erosions have occurred in the area of preschool immunizations. For example, between 1978 and 1982, there was a decline in the percentage of preschool children adequately immunized against disease. In 1978, 51.7% of

---

Address correspondence to Ms Edelman, President, Children's Defense Fund, 122 C St NW, Washington, DC 20001.

black preschool children were not fully immunized against diphtheria, pertussis, and tetanus. By 1982 that figure had climbed to 66%.[7] In 1978, 60.7% of black preschool children were not adequately immunized against polio. By 1982 the number had risen to 65%.[7]

Even prior to the dramatic federal budget cuts in public health programs that have occurred since 1981, public funding for maternal and child health was seriously inadequate. In 1980, Medicaid, the largest source of health care financing for poor children, reached only about half of all poor children and women of childbearing age.[6] In addition, because private health insurance coverage is closely associated with family income and employment status, thousands of families with children—in which parents worked at marginal jobs that offered no health insurance—were left without any means of financing the most basic forms of health care for their children.[8]

The lack of health insurance for poor children and pregnant women has been exacerbated in recent years by three major trends. First, federal and state budget cuts have eliminated 700,000 children from Medicaid and have kept hundreds of thousands more from ever qualifying for benefits.[2] Indeed the correlation between child poverty and Medicaid eligibility is at its lowest point since the program was first fully implemented in the early 1970s.[5] Second, unemployment caused by the recession led millions of families to lose employment-related health insurance benefits.[9] Third, employer cutbacks in health coverage for workers have led to reductions in family insurance benefits for the spouse and children of employed individuals. Between 1979 and 1982, the number of uninsured children living with working insured individuals has doubled.[10]

EFFECTS ON NEW YORK

The health status of minority children in New York is a reflection of these national statistics, as is the gap between poverty and health insurance coverage. In 1982, black infants born in New York were 1.6 times more likely than white infants to die before reaching their first birthday.[5] Throughout the state, the poorest areas showed significantly higher mortality rates. For example, 12 counties in upstate New York reported infant mortality in some communities that exceeded the county rate by 30% or more.[11] Some of the death rates are astonishing: the 1982 non-white infant death rate per 1,000 live births in some communities was 400% greater than rates in non-black, non-poor communities.[12]

Prenatal care data, particularly for non-white women, are alarming. In 1982, non-white women living in New York experienced a "late-or-no" prenatal care rate (21.3% of all pregnancies) that was the highest in the nation.[5] In central Harlem, the late-or-no care figure reached 35.9% in 1982, the same year that the death rate for non-white infants surpassed 27 per 1,000.[12]

In regard to low birth weight, strong disparities also occur by race. The incidence of low birth weight, the strongest determinant of health outcome, was twice as great for black infants born in New York in 1982 as for white babies.[5] One out of every eight black babies born in New York in 1982 was born at a low birth weight. In central Harlem, that figure jumped to one in six.[12]

Teenage pregnancy among adolescents is as profound a problem in New York as elsewhere. Adolescent pregnancy is significantly associated with heightened health risks to both mother and infant. Yet in 1982, in New York, one sixth of all births to non-white women were born to adolescent mothers.[13] About 90% of these births took place out of wedlock.[13] There were more than 300 births to non-white children under 15 years of age, virtually all out of wedlock.[13] While the 1980 national average for late-or-no prenatal care was 5% among non-white New York teenage mothers in 1982, nearly 39% under 15 years, and 30% between 15 and 19 years, received little or no prenatal care.[13]

Finally, as elsewhere, New York's public programs for the poor fail to help significant numbers of children. In 1980, 34% of poor children in New York were not covered by Medicaid.[14] Because of the poverty in which they live, it is unlikely that these children's parents had jobs that provided them with adequate health insurance for their children.

Children need access to a quality and reliable health care system. Yet two thirds of children living in poverty nationwide are either completely uninsured or else are insured for only a portion of each year.[15] Health insurance status is a strong determinant of access to health care. Data show that uninsured persons are 55% less likely to receive physicians' services and 90% less likely to receive hospital care than the insured.[16] Thus, there should be cause for concern.

The level of concern has intensified in recent years. The increase in the pool of uninsured children and women of childbearing age has coincided with major cutbacks—through revenue limits, rate-setting, and outright budget reductions—in the amount of funding flowing into hospitals and other portions of the health care system. In many areas of the country, the modest amounts of charity care for the uninsured that were once available are now disappearing, as public funding erodes and as institutional cost-shifting to absorb bad debts becomes more difficult. It is not uncommon to find uninsured women routinely travelling for hours to give birth because no nearby hospital will admit them. Even more disturbing, there are indications of a substantial increase in some areas of the country in the incidence of home births among poor women, because they can afford neither doctors nor hospitals.

In light of these trends among poor children and mothers, it is vital that no further efforts to reduce public and private health care expenditures occur unless changes in health care spending patterns are linked to reforms that ensure all individuals access to basic health services. If health care reductions are not linked to equity reforms, the result will be even deeper disparities in health status and health care utilization among poor women and children.

New York State has recognized this need to link equity with cost containment. The state has fashioned a hospital revenue control program that includes pooling provisions for underwriting the cost of hospitalization for uninsured persons. The state has also implemented special initiatives targeted at adolescents, indigent pregnant women, and young children. However, the health status measurements cited above for New York's children, indicate that the state will have to build greatly on this beginning before equity can be achieved.

For example, New York spends about $120 million annually to subsidize medical education. Yet that expenditure has led to an oversupply of physicians in many areas and a simultaneous shortage of minority physicians and physicians who will practice primary care in underserved areas.[17] Similarly, the state is continuing to subsidize what many consider to be the over-purchase of costly medical technology, while at the same time holding Medicaid reimbursement rates for obstetric exams to $12 per visit.[12] This has resulted in an enormous deficiency in the number of obstetricians willing to treat poor pregnant women.

THE ETHICAL CHALLENGE

At the national level, the debate is only beginning to focus again on the equity issue after years of reckless and shortsighted budget reductions that have brought pain to many families and children. In 1984 Congress enacted several modest but crucial Medicaid reforms for mothers and children as part of the Deficit Reduction Act, but a major debate over the future structure of the American health care system lies ahead.

It is within the context of this cost containment debate that the real test of the nation's commitment to child health will occur. Will policymakers fashion reforms that provide essential health services for all Americans, with limitations based on need and medical efficacy, or will we continue to maintain a health system in which a child's receipt of basic health care depends on whether his mother is a housekeeper or a bank executive?

In 1983, in the midst of the severest retrenchment in American domestic policy in 50 years, the President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research concluded that society has an ethical obligation to ensure that all its members have access to basic health services and that the ultimate responsibility for ensuring that the societal obligation is met lies with government. Policymakers at all levels of government must heed these words. No major reshaping of the American system for distributing health services can proceed ethically unless the ultimate goal of such an undertaking is not simply saving money but ensuring a basic equity in health care for all Americans.

REFERENCES

1. Altman D, Morgan D: "The role of state and local government in health." *Health Affairs*, Winter, 1983, p 20.
2. Rosenbaum S, Weitz J: *Children and Federal Health Care Cuts.* Washington, DC, Children's Defense Fund, January, 1983.
3. National Center for Health Statistics, 1980 data.
4. Kleinman J: "Use of ambulatory medical care by the poor: another look at equity." *Medical Care* 1981; 19:1011.
5. *American Children in Poverty.* Washington, DC, Children's Defense Fund, 1984.
6. Budetti P: Testimony Before the Joint Economic Committee, Washington, DC, November 1984.
7. *Healthy People: The Surgeon General's Report on Health Promotion and Disease Prevention.* Washington, DC, Public Health Service, Dept of Health, Education, and Welfare, 1979.
8. *Profile of Health Care Coverage: The Haves and Have-Nots.* Washington, DC, Congressional Budget Office, 1979.
9. Energy and Commerce Committee, US House of Representatives, 1983.
10. Testimony of Katherine Schwartz, Urban Institute, Before the Senate Finance Committee. April 21, 1984.
11. *Prenatal Care in Upstate New York.* Rochester, Statewide Youth Advocacy, Inc, April 4, 1984.
12. *Redistribute America, et al vs David Axelrod, MD.* Rulemaking petition to NYS Health Dept, March 23, 1984.
13. Vital statistics data collected by the Children's Defense Fund.
14. *State Medicaid Report.* Evanston, IL, American Academy of Pediatrics, 1984.
15. Wilensky G, Berk M: The health care of the poor and the role of Medicaid. *Health Affairs*, Fall, 1983.
16. Davis K, Rowland D: "Uninsured and underserved." *Security Access to Health Care.* Washington, DC, President's Commission on Ethical Problems in Medicine and Biomedical and Behavioral Research, 1983.
17. Bernstein B, Ensminger B: "No med school subsidies." *Empire State Report*, April 1984, p 43.

BETTER NUTRITION YIELDS BETTER HEALTH

Since everyone belongs to some minority, a collation of nutritional needs for minorities would be a textbook of nutrition. Books have been written on ethnic and regional differences of food consumption. In glancing through them, one can get the impression that those who consume a well-balanced diet are a minority.

Good nutrition is usually available for all persons and requires little more in effort or finances than is frequently spent on bad nutrition. Some disease states can be helped by eliminating certain foods from the diet; even fewer require expensive supplements. A diet selected from multiple food sources provides all the needed nutrients and avoids the possibility of consuming supplements which may themselves be toxic.

Poverty is a common circumstance of many minorities. Although some of the malnutrition in poor families may be related to lack of knowledge, numerous studies have indicated that increasing the availability of food improves the nutritional value of a poor family's diet.

The Women, Infants, and Children program, not limited to the impoverished but rather to those women, infants, and children at nutritional risk, supports the concept that good nutrition during pregnancy results in improved pregnancy outcome. Similarly, the program has had an apparent beneficial effect on the iron status of young children. Rates of growth in these children were increased.

LEWIS A. BARNESS, MD

A comparison of selected health indicators for black and white children in New York State

LINDA A. RANDOLPH, MD, SYLVIA RIVERS, RN, EDD

In 1981 a report of the Select Panel for the Promotion of Child Health found that "the health status of American children has improved dramatically over the past two decades, but not all groups have shared equally in the progress. Sharp disparities persist in both health status and the use of health services according to family income, ethnic background, parental education, and geographic location," the panelists noted.[1] In 1984, Secretary of Health and Human Services Margaret Heckler stated that, "while the health of all Americans continues to improve radically, a disparity between the majority population and the minorities still plagues us. . .blacks still suffer excessive medical problems that are an affront to our ideals." [2]

There has been a steady increase in the number of homes with female head-of-households in the United States. In 1983, 47% of black households with children were headed by a woman, and this figure continues to rise.[3] This figure has increased from 8% in 1950 and 21% in 1968. On the average, women earn less than men, and a single adult earns less than two adults; therefore, households headed by women are frequently low-income households. According to the United States Bureau of the Census, in 1978, children in families headed by women were six times more likely to be living below the poverty level than children in families headed by men. "Nearly half of all female-headed families with children were below the poverty level in 1980." [3]

Poverty is associated with less access to health care, substandard housing, poor nutrition, and lack of health information. There is a correlation between poverty and excessive rates of sickness and disability. Census data indicate that families headed by single women were twice as likely to live in poverty. Fifty-five percent of black infants were born to unmarried mothers last year, and a large number of these children live with a single parent.[3] According to the report of the Select Panel for the Promotion of Child Health, "factors in the social environment, such as family income, parental education, opportunities for productive work, minority status, child care arrangements, and the availability of community supports for adolescents and parents of young children, all exert a powerful influence on health." [1] McCormick[4] believes that while young maternal age serves as a marker for socioeconomic disadvantage, when the socioeconomic variable is controlled, the outcome for adolescent mothers and other mothers shows no difference. An examination of the relevant statistics concerning health care among black children demonstrates that the health and well-being of this population is in jeopardy. Strategies for dealing with this issue must emphasize preventive measures.

PREGNANCY AMONG ADOLESCENTS

Teenage pregnancy has reached epidemic proportions in New York State and nationwide. There were 27,611 babies born to teenage mothers in New York State in 1982 (Table). The Governor's Teen Pregnancy Initiative is aimed at reinforcing the prevention of initial teen pregnancies through the development of meaningful alternative activities, such as learning opportunities and job training, and assisting pregnant adolescents and adolescent parents to acquire healthy childbearing and childrearing practices. The most significant issues for state action to prevent teenage pregnancies are increased availability of job training and employment for all adolescents, increased concern and attention by the education system for sexually active adolescents, and increased access to confidential and low-cost contraception.

Although adequate prenatal care should be started in the first trimester, only 44.6% of minority women received early prenatal care in 1980 in New York State, compared to 75% of white women.[5] In 1980, 19.8% of minority women received late or no prenatal care compared to 6.5% of white women. During the same year, the situation for the teenager was worse: 28.9% of minority teenagers received late or no prenatal care.[5]

With insufficient prenatal care, the pregnancy and its outcome will suffer. Maternal morbidity and mortality occur at a much higher rate among blacks than among whites. In 1982, the number of discharges from hospitals in New York State of women aged 15 to 19 years (the majority of whom was black) who were being treated for obstetric diseases occurring before and after giving birth was 6,132.[6] This figure does not include women who were not treated in a hospital. There is no distinction made regarding race. With respect to maternal mortality rates, although the absolute numbers are small, maternal mortality occurred at a much higher rate among blacks than among whites. Among blacks, there were 3.6 maternal deaths per 10,000 live births; and, among whites, there were 1.3 maternal deaths per 10,000 live births.[7]

FETAL MORBIDITY AND MORTALITY

In New York State, the fetal mortality rate among black and white women was 13.2/1,000 live births and 9.7/1,000 live births, respectively (Table). Late or no prenatal care has been associated with low birth weight. Low birth weight is strongly linked with neonatal mortality, illnesses during infancy, cogenital malformations, and developmental disabilities. In 1982, in New York State, low birth weight occurred in 12.1% of births to blacks and in

---

From the New York State Department of Health Office of Public Health, Albany, NY.

Address correspondence to Dr Randolph, Director, New York State Department of Health, Office of Public Health, Tower Building, Empire State Plaza, Albany, NY 12237.

Selected Health Status Indicators by Race for New York State & The United States, 1982

	New York State*		United States**	
	Black (%)	White (%)	Black (%)	White (%)
Live births	53,486	185,149	592,641	2,992,054
Low birth weight live births	6,456 (12.1)	10,926 (6.0)	73,360 (5.6)	165,285 (5.6)
Live births with late or no prenatal care	11,500 (21.6)	12,925 (7.0)	209,975 (35.4)	522,258 (17.8)
Teenage live births	10,790 (20.2)	16,821 (9.1)	145,929 (24.7)	362,101 (12.4)
Out of wedlock live births	32,469 (60.8)	29,769 (16.1)	335,927 (56.7)	355,180 (12.1)
Infant deaths†	964	1,096	11,757	30,478
(rate per 1,000 live births)	18.0	10.3	20.0	10.5
Neonatal deaths†	637	1,352	7,900	20,592
(rate per 1,000 live births)	11.9	7.3	13.4	7.1
Post neonatal infant deaths†	327	559	3,857	9,886
(rate per 1,000 live births)	6.1	3.0	6.6	3.4
Spontaneous fetal deaths	707	1,787	6,310	14,352
(rate/1,000 live births)	13.2	9.7	10.7	4.9
Single parent families‡	275,642 (48.8)	593,958 (16.4)	2,619,091 (42.9)	7,003,371 (13.9)
Families with income in 1979 below the poverty level‡	145,357 (25.7)	253,453 (7.0)	1,615,952 (26.5)	3,566,679 (7.1)
Families with income in 1979 below the poverty level, female head-of-household, no husband present, and children under 18 years‡ (percent is of all families below the poverty level)	98,918 (68.1)	98,298 (38.8)	959,085 (59.4)	1,071,886 (30.1)

* Source: New York State Vital Statistics, 1982.
** Source: National Center for Health Statistics.
† Mortality data for areas other than New York State are from 1981.
‡ Source: 1980 Census of the Population.

6.0% of births to whites. This disparity is also reflected in national statistics.

In New York State, the Prevention of Low Birth Weight Project for women of all ages identifies newborns of mothers who reside in areas of the state where infant mortality rates and low birth weight rates exceed statewide averages. This project also attempts to lower the incidence of premature births by providing medical and educational services and to prevent intrauterine growth retardation by promoting early and regular use of prenatal care services. These services are provided through contracts with eligible providers located in areas of New York State where residents exhibit substantial risk of adverse pregnancy outcome.

MEASURES TO PROMOTE INFANT HEALTH

Almost 75% of infant deaths take place during the neonatal period (birth to 28 days). In 1981, the neontal mortality rate in New York State was 75% higher for blacks than for whites (Table). The postnatal mortality rate (the number of infants who die after the first month of life, but before their first birthday) is 6.1/1,000 for non-whites and 3/1,000 for whites in New York State (Table); in 1981, 964 black infants died before age 1. A contributing factor is Sudden Infant Death Syndrome (SIDS), which occurs at a rate of 1.0/1,000 children in New York State and twice as often—2.2/1,000—in blacks.[8]

In New York State, there are several programs that address this problem, such as the Infant Apnea Program, the Sudden Infant Death Syndrome Program, and the Infant Assessment Program. During 1984, 12 Infant Apnea Centers were established statewide. Apnea monitors were made available through Medicaid reimbursement or state aid for infants with a family history of SIDS or apnea. The Sudden Infant Death Program in New York State coordinates educational, reporting, and follow-up activities in partnership with county public health professionals. Included in this program is a uniform autopsy protocol, which is distributed to coroners, medical examiners, and pathologists throughout the state.

In a secondary prevention effort, the Infant Health Assessment Program supports the development and implementation of a model program to ensure that infants and young children at risk for physical and developmental disabilities receive the necessary screening, diagnostic, and treatment services. This program is implemented through contracts with New York's 57 county and city public health units, with an estimated 350 newborns registered monthly for subsequent house visits and follow-up by public health nursing staff. Through the local public health agencies, the program operates to alert providers of public and private health care, social services, and educational services of such children; to determine the nature of the morbidity among this high-risk population; to report on the treatment experience of registered children; and to identify gaps in the provision of services.

A few days after birth, all newborns in New York State are screened for inherited disorders, such as sickle cell anemia, which is prevalent in blacks. The New York State Newborn Screening Program is designed to prevent mental retardation or other disabilities by detecting the presence at birth of genetic and metabolic diseases. The Newborn Screening and Long-Term Tracking Program is designed to ensure that therapeutic measures are provided by determining the status of individuals identified by the screening program, reviewing problems with dropouts, encouraging such steps as may be necessary to minimize dropouts and lack of adherence to treatment regimens. Again, follow-up of the affected population is important. While several thousand affected individuals have been identified in the screening program since 1965, no long-term data on their present status are available. Sickle cell disease is the most common disorder diagnosed. There is a continuing need for education and support in communities and individuals at risk for sickle cell disease and trait. In addition, as the survival of patients with sickle cell disease advances into adolescence and adulthood, counselling of the patient and family is important.

HAZARDS OF GROWING UP

The black toddler is more likely to live in old buildings with peeling lead-based paint. Although lead-based paint is no longer used on interior surfaces, the amount of lead in ambient air and soil from the use of leaded gasoline in automobiles presents a risk to all children. There is evidence that black children may be more susceptible to lead exposure from any source.[9] Toxic levels of blood lead are found in 12% of pre-school black children nationwide, but only 0.4% of whites. For low-income black children, this percentage increases to 18%. Anemia contributes to excessive lead absorption, and minority children experience high levels of anemia as a result of inadequate nutrition.

The New York State Childhood Lead Poisoning Prevention Program provides screening and follow-up services including educational home visits and environmental investigations. Lead can poison adults, but children are especially susceptible because their bodies absorb 40% of ingested lead and their central nervous system is still developing, making it more prone to damage. The Lead Poisoning Prevention Program screened 164,000 children in 1983, finding positive results in over 3% of those tested. Screening is carried out through health department clinics; Women, Infants, and Children (WIC) sites; neighborhood health centers; headstart day care centers; and door-to-door efforts in high risk areas. The program ensures that appropriate medical care is provided and routinely monitors the affected child's blood lead level.

The Women, Infants, and Children Program, set up to reach low-income women, serves only 35% of those in need. It supports the improved nutritional status of women, infants, and children by providing both direct nutrition supplements and education to high risk mothers regarding the importance of nutrition in having healthy babies and in aiding the growth and development of young children. Schwartz et al[10] state that "our results indicate that participation in WIC is associated with enhanced pregnancy outcomes. . .the larger reduction in the incidence of low birth weight, prematurity and neonatal mortality suggests that nutrition intervention programs may be more effective in impacting on proper outcomes of pregnancy."

The Emergency Nutrition Aid Program was established by the New York State Department of Health to supplement the federally-funded Food Program for Women, Infants and Children. It provides nutritional assistance and education to pregnant women and adolescents who have been found to be at nutritional risk.

In addition, the New York State Department of Health's Nutrition Surveillance Project was developed to identify the various nutritional problems of populations at risk and to achieve better understanding of the extent, distribution, and causes of the problem in order to develop effective strategies. This is part of the Health Department's effort to investigate the current status of hunger and nutrition-related health problems, with a focus on target groups at higher risk such as women and children. The waiting list of eligible individuals is increasing monthly, while federal proposals suggest reductions in appropriation levels. Support for increased funding is needed at both the federal and state levels.

When the New York State statistics are compared with national data, the rates of several health indicators appear to be lower in New York State. Low birth weight, infant and neonatal mortality rates, and the incidence of late or no prenatal care have rates in New York State that are lower than those for the United States as a whole. However, when the rates for blacks are compared with those of whites, for both the United States and New York State, the rates are higher for blacks. This supports the premise that the black population still suffers excessive health problems as compared to the white population, and this is a national problem. The development and implementation of aggressive, innovative national initiatives are needed if there is to be improvement in the health status of black children.

DENTAL HEALTH CARE

There is a large backlog of unmet dental needs in the black community. About half of the general population visits the dentist at least once in a given year; that does not happen among minorities. The new mass media techniques developed for the promotion of dental care have not resulted in an increase in demand for dental care in the minority community. This apparent lack of interest can be attributed to three factors: few dentists practice in these areas; there are few blacks and Hispanics being trained as dentists; and, little, if any, progress has been made to overcome the financial barriers to dental care faced by the elderly and the indigent.

Recognizing this deficiency, the State Department of Health has several new initiatives that provide dental programs for underserved populations. There are now dental care demonstration projects in 55 schools and day care centers in nine high-risk communities; and the Health Department is funding 16 preventive dentistry initiatives in low-income neighborhoods. Eleven of the 16 are in black communities. The Department of Health is seeking to encourage public awareness of preventive dental techniques and the need for professional dental care. Funds have also been used to support preventive dentistry for high-risk and underserved maternal and child populations. This program provides screening referrals and low-cost preventive dental services to children residing in communities with a high degree of poverty and an excessive occurrence of dental disease. These activities are carried out by community-based dental hygienists and are linked whenever possible to other health promotion services targeted to children (such as WIC programs, school health programs, and child health clinics).

TEENAGERS’ PROBLEMS

Preliminary data drawn from vital statistics and the 1982 census for New York State give rates for accidental deaths for non-white children aged 1 to 14 years of 17.8/1,000 and for whites of 12.4/1,000.[7] Teenagers are particularly at risk for serious accidents; accidental deaths for those 15 to 19 years are 15.5/100,000 for non-whites, and 39.6/100,000 for whites. Note that this rate is lower for black teens, probably related to the use of cars, which are more accessible to white teens. Suicide rates among teens 15 to 19 years are 6.5/100,000 for whites and 3.3/100,000 for blacks. However, when it comes to homicide rates for teens 15 to 19 years, the rate is 40.9/100,000 for blacks and 9.4/100,000 for whites. This homicide statistic does not lend itself directly to solution by health intervention. However, it is further evidence of the need for innovative social programs that can lessen the negative impact of joblessness, diminishing educational opportunity, and the lack of control over one’s life experienced by minority youth.

STRATEGIES FOR THE FUTURE

Children are a captive audience, whether in the hospital at birth, in pre-school programs and day care, or in the school system. These are strategic contact points for preventive intervention and treatment, and programs are needed to capitalize on this situation and to identify children at risk for health problems.

While federal funds for WIC programs and other maternal and child health services are facing drastic cuts, state initiatives aimed at school health, prenatal care, adolescent pregnancy, and nutrition services become even more essential.

It remains incumbent upon the nation and its health care professionals to develop a health care policy that dictates the direction of all new programs and the expansion of existing ones targeted at high-risk population. In this way, progress can be made toward eliminating the barriers that create unequal chances for black children to grow, to develop, and to succeed.

REFERENCES

1. *Report of the Select Panel for the Promotion of Child Health to the United States Congress and the Secretary of Health and Human Services. Vol. I—Better health for our children: national strategy.* Washington, DC, 1981, p 3.
2. Schwartz H: “Narrowing the black-white health gap.” *Wall Street Journal,* February 7, 1984.
3. “Trends.” New York, New York State Council on Children and Families, vol 1, January 1983.
4. McCormick M, Shapiro S, Starfield B: High-risk young mothers: infant mortality in four areas in the United States, 1973–78. *Am J Public Health* 1984; 74:18–23.
5. New York State vital statistics, 1981. New York, New York State Department of Health.
6. Statewide Planning and Research Cooperative System (SPARCS) annual report series, 1982. New York, New York State Department of Health.
7. New York State vital statistics, 1982. New York, New York State Department of Health.
8. New York State vital statistics, SIDS Program, 1982. New York, New York State Department of Health.
9. “Trends.” New York, New York State Council on Children and Families, vol 1, April 1983.
10. Schwartz B, Anderka T, Finison S: WIC participation and pregnancy outcomes: Massachusetts Statewide Evaluation Project. *Am J Public Health* 1981; 174:1086.

THE “WELLNESS MOVEMENT” AND THE INNER CITY

Stevens Point, Wisconsin—Every morning, soon after dawn, this small college town in the middle of Wisconsin’s farm land comes alive with the activities of disciples of the “Wellness” movement.

They run, they jog, they hike, through misty woods, around small lakes, or they run around the track of the University of Wisconsin’s 9,000-student campus.

They are some of the 700 or so participants in a workshop on how to spread the gospel of wellness to the people back home.

But one speaker took them away from the bucolic charms of mid-Wisconsin to the inner city of Newark, N.J.

There, said Dr. Robert Johnson, director of adolescent medicine at New Jersey Medical School, the strategy for getting people to take responsibility for their own wellbeing—the essence of the wellness movement—has to be different.

“Look at jogging. Let me tell you that if a kid in the inner city gets up in Newark and goes jogging at 5:30 in the morning, he is going to end up shot or in Youth House—a detention center—because someone is going to think he is going to commit a crime.”...

Johnson is concerned that too much health care money is spent on handling the problems of teenage pregnancy, drugs and sexually transmitted disease and not enough on preventive education. The problems may affect 15 percent of the youngsters in the inner cities, he said.

“We are spending very little on the 85 percent who are not supposed to have problems. But you should spend money on them to prevent them from jumping over into the 15 percent.”

The funds should be used, he said, to make young city dwellers self-sufficient and involved in looking after themselves. When they are well, they think more clearly, make better decisions and are less likely to have either social or health problems, said Johnson.

ENA NAUGHTON
The Miami Herald
July 25, 1981

The restraint of progress

Declining participation of blacks and other minorities in medical schools

LEON JOHNSON, JR, DED

Before the mid-1960s, medicine was predominantly a career opportunity for white men. As late as 1966, several medical schools had color barriers.[1] The initiative to increase the number of black and other minority physicians began in the late 1960s as a response to the civil rights movement, the assassination of Dr Martin Luther King Jr, and the recognition by government and private concerns of the serious problems caused by the underrepresentation of minorities in medicine, as detailed in the report, "Minorities, manpower and medicine."[2]

In the fall of 1968, 266 black students, 20 Mexican Americans, three American Indians, and three Puerto Ricans entered their first year of medical school. They represented barely 3% of the entire first-year class of medical schools in the United States, according to data of the Association of American Medical Colleges (AAMC). The 1968 entering class was significant because, for the first time, first-year black enrollment in the predominantly white medical schools had risen to 50% of the total first-year black enrollment in the predominantly black medical schools—Howard University College of Medicine, Washington, DC and Meharry School of Medicine, Nashville. In the next three years, the predominantly white schools' share of black first-year students increased to almost 80% of the total first-year black enrollment.[3]

In 1970, the AAMC issued a landmark report that defined the long-term goal of achieving "equality of opportunity by reducing or eliminating inequitable barriers and constraints to access" to the medical profession.[4] The task force recommended as a short-term goal that "U.S. medical schools increase the representation of minorities in the M.D. degree program from 2.8 percent in 1970–71 to 12 percent in 1975–76."[4] The task force also noted the importance of financial assistance to increase opportunities for minorities in medicine. The report was endorsed by the American Medical Association, the American Hospital Association, and the National Medical Association.

Measured in terms of first-year enrollment, minority student representation appeared to make dramatic gains toward the goal of 12% minority representation by 1975–1976. Minority student representation climbed from 2.96% of the first-year class in 1968–1969 to nearly 10% in 1974–1975. This gain in the enrollment of minorities occurred mostly as a result of the increased admission of minorities into predominantly white mainstream medical schools. In 1974–1975, 43%, or 1,049, of the 2,423 black medical school applicants were accepted; 48%, or 64, of the 134 American Indian applicants were accepted; 49%, or 217, of the 440 Mexican American applicants were accepted; and 43%, or 76, of the 177 Puerto Rican applicants were accepted. Overall, 44%, or 1,406, of the 3,174 underrepresented minority applicants were accepted in United States medical schools in 1974–1975.[5]

Since 1974–1975, the nation's effort to increase diversity among its health care providers has been subdued. The promising increases in minority enrollments disappeared after 1974, becoming a national disappointment and creating a quiet health crisis. This reversal was a renouncement of a national commitment to insure good health care for all Americans.

By 1978–1979, the acceptance rate for blacks had dropped to 38%, from the 1974–1975 level of 43%. Overall, the acceptance rate for all underrepresented minorities dropped from 44% in 1974–1975 to 41% in 1978–1979. The decline in the acceptance rate of black students took place even with an increase of 141, or 5.8%, in black applicants over the 1974–1975 figure. In contrast, the acceptance rate for nonminority applicants rose significantly from the 1974–1975 level of 35% to 45% in 1978–1979, despite the fact that there were 6,135, or 15.5%, fewer nonminority applicants.

By 1982–1983, 39% of black applicants were accepted, 48 fewer black students than had been accepted eight years earlier in 1974–1975, notwithstanding a 7.3% increase in the number of black applicants. Overall, with an 8.8% increase in the number of underrepresented minority students applying to medical school, only 45 more underrepresented minority applicants were accepted in 1982–1983 than in 1974–1975. In the same period, the acceptance rate for nonminorities grew from 34.6% to 49% despite 7,173, or 18.2%, fewer nonminority applicants. The decline in the acceptance rate of black students and the decline in the proportion of blacks in the entering classes took place in spite of the fact that the number of minority applicants was relatively stable, the black applicant pool increased significantly, the mean Medical College Admissions Test scores of black applicants improved, and the number of places in American medical schools increased significantly.

Total medical school enrollment increased by a cumulative 24.6%, or 13,194 students, during the eight years from 1974–1975 to 1982–1983. Blacks comprised only 3.9% of this increased total medical school enrollment. Blacks, Puerto Ricans, Mexican Americans, and American Indians, the underrepresented minorities in medicine, comprised 9.25% of the expanded total medical school enrollment. In other words, between 1974–1975 and 1982–1983, nonminorities captured 90.75% of the more than 13,000 new medical school seats, and maintained their dominance of the existing positions in medical schools.

Despite the significant expansion of the medical education system, nearly 25% of the 1,196 black students in the

---

Address correspondence to Dr Johnson, President, National Medical Fellowships, Inc, 250 W 57th St, New York, NY 10107.

1981–1982 first-year class were enrolled in the predominantly black medical schools—Howard, Meharry, Morehouse School of Medicine, Atlanta, and Drew Postgraduate Medical School, Los Angeles. Furthermore, in 1981–1982, six of the nation's 127 medical schools had no black students, and 75 medical schools had a black student enrollment of less than 5%. In 1981–1982, there were only 90 more black medical students in the entering class nationwide than in the first-year class of 1974–1975. It is significant that during this same period, Howard, Meharry, and Morehouse increased their black first-year enrollment by 89 students.

The decline of black and other underrepresented minorities in medicine is in stark contrast to expert opinion and national policy recommendations that support equitable health care for all people. In 1980, the Graduate Medical Education National Advisory Committee made national headlines with its prediction of a physician oversupply by 1990. However, the report also recognized the continuing underrepresentation of minorities in medicine and recommended that efforts be taken to achieve "a balance between supply and requirements of physicians in the 1990's while assuring that programs to increase the representation of minority groups in medicine are advanced."[6]

Louis Sullivan, MD,[7] President and Dean, Morehouse School of Medicine, noted that all medical schools should expand and reinforce their commitment to recruiting, educating, and graduating more black students, especially in light of the fact that 30 fewer black students graduated from medical school in 1982 than in 1978, despite an 11% gain in the number of all medical school graduates. In 1978, there were only 793 black students among 14,393 medical school graduates (5.5%), but by 1982, this number had decreased to 763 blacks (4.8%) among 15,985 medical school graduates. If equitable health care is to be achieved, American medical schools should be producing 2,200 black graduates annually, rather than the 700 to 800 they now graduate each year.

Economist Bernard E. Anderson, PhD, has suggested that the position of blacks and other minorities in the economy is like that of the caboose on a train.[8] When the train speeds up, the caboose moves faster; when the train slows down, so does the caboose. No matter how fast the train goes, the caboose will never catch up with the engine unless special arrangements are made to change its position.

The progress achieved by minorities in medical school has been similar to the position of blacks and other minorities in the economy as characterized by Anderson. However, in the case of medical school minority representation since 1974–1975, a unique and cruel twist has been added to the analogy. Since 1974–1975, regardless of whether the train sped up or slowed down, more train cars were added. Greater distance has been placed between the caboose and the engine.

Since 1974–1975, there has been a decline in the relative participation of minorities in medical school and a substantial increase in the participation of nonminorities. This restraint of minority participation has affected both the availability and delivery of health care and the distribution and practice patterns of physicians. Most critical in this situation is the fact that restraint of minority participation has retarded efforts to bring the level of health care for minorities up to the standard of care for nonminorities.

Multiple factors such as the cost of medical education, lack of role models, lessening civil rights role of the federal government, lack of minority medical school faculty and staff, racial prejudice and discrimination in medical schools and hospitals, and inconsistent medical school commitment to affirmative action as well as a lack of effort to create a congenial supportive environment for minorities, affect the number of minorities applying and enrolling in medical school. Furthermore, the problems of the public education system have led to poorer academic preparation and less well-developed study skills and habits, which have been cited as the reason for the higher academic failure rate of minorities. This increased academic failure rate for minorities has become a keystone statistic that has been said to discourage medical schools from admitting more minority students and also discourages more minority students from applying. This is an unfortunate circumstance, because the problem focuses on the percentages of minorities that do not succeed and not on the fact that more individual nonminority students experience academic failure than minorities. In this regard, review and retention programs should be developed to improve the performance of all medical students. It is in the nation's best interest to assure that the investment students make in preparation for medical school and the investment medical schools and society make in students is maximized for success.

It has been demonstrated that blacks and other underrepresented minorities in medicine will not reach a full participation level through the growth and expansion of an unregulated medical education system. Affirmative action policy initiatives and other special measures are necessary if the medical community is to achieve its objective of good humanistic health care for all people. Without such definitive action, the caboose—good health care for low-income groups, urban blacks, and people of Hispanic heritage—may be cut loose from the train.

REFERENCES

1. Morais HM: *The History of the Negro in Medicine.* New York, Publishers Company, Inc, 1967, pp 9–10.
2. Hutchins EB, Reitman JB, Klaub D: Minorities, manpower, and medicine. *J Med Educ* 1967; 42:809–827.
3. Odegaard CE: *Minorities in Medicine From Receptive Passivity to Positive Action 1966–76.* New York, The Josiah Macy Jr Foundation, 1977, p 41.
4. Nelson BW: *Report of the Association of American Medical Colleges Task Force to the Inter-Association Committee on Expanding Educational Opportunities in Medicine for Blacks and Other Minority Students.* Washington DC, Association of American Medical Colleges, 1970.
5. Association of American Medical Colleges, Office of Minority Affairs: *Minority Students in Medical Education: Facts and Figures.* Washington DC, Association of American Medical Colleges, 1983.
6. Graduate Medical Education National Advisory Committee: *Summary Report of the Graduate Medical Education National Advisory Committee to the Secretary, Department of Health and Human Services.* Washington DC, US Government Printing Office, 1980, DHHS publication no. (HRA)81-651, p ii.
7. Sullivan LW: The status of blacks in medicine. *N Engl J Med* 1983; 309:807–808.
8. Anderson BE: An economic defense of affirmative action. *Black Enterprise* 1982; 12:40.

Black-Jewish relationships

Access to health services and health careers

SAMUEL WOLFE, MD, DRPH

Why discuss black-Jewish relationships and access to health services and health careers? Why not black-Irish relationships? Why not black-Protestant relationships? Why not black-Catholic relationships? Why not black-white relationships? Why juxtapose society's two most identifiable scapegoats?

Jewish philanthropy was conspicuous in fostering university education for blacks, in support of institutions such as Howard, Fisk, Atlanta, and Dillard. The Rosenwald Fund in particular put emphasis on business principles in philanthropy and, like most northern white philanthropies, accepted the Booker T. Washington philosophy of concentration on elementary and vocational schools rather than broad-based educational efforts.

Jewish philanthropy also attempted to raise the level of health care for blacks. For example, between 1920 and 1942, the Julius Rosenwald Fund gave $1.7 million to health projects for blacks.[1] Rosenwald believed that "colored" doctors and nurses needed white exemplars of counsel and service, as well as white funds, to achieve high standards.

Late in 1930 the National Association for the Advancement of Colored People (NAACP) and an association of New York black physicians condemned Rosenwald for his "policy of financing and advocating separate institutions for American citizens of Negro descent." They maintained that "Jim Crow" setups produced servility, suppressed inspiration, and created artificial and dishonest standards. The doctors rejected the analogy of imitating Jewish hospitals, which were created to circumvent anti-Semitic discrimination in both state and private institutions; religious and cultural factors were associated with the creation of Jewish hospitals. Black groups noted that Jewish students, too, faced limitations in medical school admissions and pointedly asked why Rosenwald had not established a Jewish medical school.[2] Jews were also prominent in support of nursing and social work programs for blacks.

In 1915, the number of institutions and prominent Americans advocating racial equality could be counted on one's fingers, and Jews were atypical in support of civil rights for blacks; 20 years later blacks became increasingly confident in their own self-expression. The Jewish-black alliance had emerged from the almost total weakness of one party. Jews, among others, had wanted to make American political and social institutions sensitive to the needs of blacks, and strove to create a black leadership class through education. With these two missions launched, the need for middlemen—for white pleaders, Jews, and non-Jews—diminished.

The most critical event in the history of world Jewry took place in the 1930s, with the shift of the center of world Jewry to the United States; the holocaust was the single most profound event in 2,000 years for the Jewish people. All other events pale by comparison. From then on, the theme became "Never Again." Jews turned inward and became more blatant and open about the survival of their own groups. This trend became more marked with the creation of Israel and the struggle of the new state to survive.

According to Diner, in the days before World War II, Jews sided with blacks partly out of reticence to tackle anti-Semitism. After the holocaust there was a strident and aggressive search to expose and ferret out anti-Semitism, no matter how minor. At this time, Jewish self-interest became paramount: how would participation in black causes help Jews? Responding to attacks by the Congress of Racial Equality, the Student National Coordinating Committee, and even the NAACP, American Jews reacted by backing off and by looking elsewhere for security and survival.[3]

The most intense confrontation between Jews and blacks in the United States occurred in the New York City school crisis of 1968 and its aftermath. There was prolonged warfare, as Hentoff called it, between the predominantly Jewish United Federation of Teachers and a black population wanting community control of the schools, which had for so long failed to meet the aspirations of black people.[4] Latent racial overtones came to the fore on both sides. Jews accused blacks of anti-Semitism, and blacks accused Jews of unbridled racism. Again, it was an unequal struggle. The Jews were part of the establishment, and they effectively barred the doors against black entry into the school's power system.

Cooler voices were heard amid the conflict. At a meeting in Harlem of rabbis, white ministers and priests, and black clergy, Rabbi Albert Vorspan deplored the recriminations between blacks and Jews and emphasized the coinciding interests of two minorities seeking meaningful and peaceful coexistence. He spoke of the deep psychological insecurity of Jews, culminating in the Nazi holocaust and Arab hostility toward Israel. He spoke of anxiety bordering on hysteria among New York Jews. Reverend Wyatt Walker, a black minister, replied, "It was heartening for me to hear of your paranoia, because it allows me to admit for the black community our schizophrenia." [5]

One outgrowth of the school crisis was the birth of the

Dr Wolfe was former director of health programs at Meharry Medical College, Nashville, TN.

Address correspondence to Dr Wolfe, Professor, School of Public Health, Columbia University School of Public Health, 600 W 168th St, New York, NY 10032.

Jewish Defense League headed by Rabbi Meir Kahane, who mobilized the apprehensions of working lower middle class Jews. Like the George Wallace phenomenon, which also had a strong racial component, it was based on working class grievances and fears. People were threatened by competition for jobs by blacks, rising crime rates, deterioration of neighborhoods, and integration of schools. Coupled with this was the myth that the established elite, in an effort to placate blacks, was sacrificing the interests of whites.

It was also during this period that black nationalist denunciation of Israel reached a peak—not to be paralleled until the Reverend Louis Farrakhan's outbursts of 1984. It is, of course, incongruous for blacks to espouse the Arab cause based on history. The Arabs were the most important slave traders in East Africa, and black slaves were a major commodity that served the economic well-being of Arabs along the Indian Ocean.

The implication is that black-Jewish relationships used to be good, and now they have turned sour. Actually, they never were that good. Jews did a lot for black people which was kind and benevolent, but also colonial. Jews looked at blacks as children in terms of power relations.[6] But is it fair to expect more of Jews than of other whites? As Hentoff points out, the answer depends on whether Jews define themselves in the Prophetic tradition, or in the tradition of South Africa's troubled Jewish community.[4,5]

In the health sector, especially in places like New York, Jews are prominent and predominant in all spheres: policy, planning, administration, financing, research, and evaluation. Blacks are at the bottom of the barrel. Of 281 hospitals in New York State with an operating certificate in 1983, 266 (95%) were either proprietary or voluntary. Only 2, or less than 1%, had minority chief executive officers.[7] If New York City's Health and Hospitals Corporation is included, the number of minority chief executive officers goes up to six, or 2%. Nearly 12% of Americans are black, and yet only 3% of physicians are black. Blacks and Hispanics, by and large, are the menials of the health care workplace.

In terms of access to health services, blacks (as well as Hispanic Americans) lag behind the white community. For example, in 1982, New York State had the dubious distinction of being first in the country in percentage of non-white women having no or inadequate prenatal care (21.3%).[8] This, in turn, is reflected in increasing infant mortality rates in some areas, notably in New York City's South Bronx and Harlem. Black men have about a 6½-year shorter life span than do white men. These, and similar, data show that while Medicaid and Medicare have clearly increased access to health services for the disadvantaged, there is still a long way to go.

Jews are not responsible for these deplorable statistics. Jews also, based on their own long experience, deplore racial quotas. But they are the foremost defenders of good affirmative action programs. As has been stated by The Synagogue Council of America, "As Jews we are committed by our faith to work for racial justice in an integrated society. Any Jew who fails to join in this struggle demeans his faith." It has been true of Jews as it has been true of Americans as a whole, that when all is said and done, more has been said than done.

REFERENCES

1. Diner HR: *In The Almost Promised Land. American Jews and Blacks, 1915-1935.* Westport, Greenwood, 1977.
2. Ibid., pp 176-179.
3. Ibid., pp 236-243.
4. Hentoff N (ed): *Black Anti-Semitism and Jewish Racism.* New York, Schocken Books, 1970.
5. Weisbord RG, Stein S: *Bittersweet Encounter. The Afro-American and the American Jew.* New York, Schocken Books, 1972.
6. Dr Nathan Wright Jr, quoted in the *New York Times,* February 7, 1969.
7. A Preliminary Status Report of Minority Management Recruitment and Employment in the Health Care Industry. Albany, New York State Department of Health, April 1984.
8. *American Children in Poverty.* Children's Defense Fund, Washington, DC, 1984, p 2.

NEW YORK POST FINAL

Celebrate July 4 with our great Food section

Bakke wins on reverse bias

CALIFORNIA MEDICAL SCHOOL TOLD: LET HIM IN

OTB boss cracks down on plums

Bounce Munson for 3 days

TIME

What Bakke Means

Race: Yes

Quotas: No

FINAL DAILY NEWS

'Reverse Bias' Case:

SUPREME COURT UPHOLDS BAKKE

Vote Strike On Bridges And Tunnels

Stop Beefing!

Be a Kitchen Cut-Up and Save Money

His Bride and Joy.

Bakke decision: divisive aftermath

Inequality in the availability of black physicians

DAVID MCBRIDE, PHD

A recent report by the Graduate Medical Education National Advisory Council stressed that there is a severe shortage of black and minority health professionals. Moreover, this shortage is thought to be an important causal factor behind the discrepancy in health status and life expectancy of blacks in comparison with white Americans.[1] It is the contention of this study that the substantial shortage of black medical professionals during the last two decades has its historical roots in developments in medical education and health employment that occurred during the early decades of the 20th century. During this period, short-sighted educational policies curtailed the role of black medical schools and, consequently, there were long-term effects on the availability of black physicians. Although the focus of this article will be on the number of black physicians in Philadelphia—a city traditionally considered a leading center for American medical education—some important parallels with New York City will be drawn.

EFFECTS OF THE FLEXNER REPORT

During the last decade of the 19th century through World War I, the nation was divided along racial lines in all major aspects of socioeconomic and political life. By World War I, the gains of civil and economic rights that blacks had attained during the Civil War and Reconstruction had been reversed, and segregation, or so-called "Jim Crow" conditions, predominated in public facilities, higher education, and social and work life. A direct outgrowth of the severe poverty gripping the black American community during this period was widespread ill health and increased mortality. In 1900, for instance, the life expectancy of nonwhites was estimated to be between 32 and 35 years, some 16 years lower than that of the average white American.[2,3]

Within such a social and health context, widespread segregation prevailed in American medical education. In 1905, virtually all of the nation's 152 white medical schools (about one third more than the number of medical schools in the nation today) barred blacks from attendance, as did the nation's white dental and nursing schools.[4] As a result of the efforts of black self-help groups and white philanthropic organizations, Howard Medical School in Washington, DC and Meharry Medical College in Nashville emerged as the two main centers for the education of black physicians. Five other black medical schools were also established during the post-Reconstruction decades, the most productive of these being the Leonard Medical School of Shaw University in Raleigh, NC. This school produced many distinguished black doctors and, overall, about 500 graduates from all sections of the country during its operation from 1882 to 1915.[5] An additional 500 black physicians graduated from the other four black medical colleges, which functioned in Memphis, Louisville, Knoxville, and New Orleans from 1895 to 1923.[6]

The demise of black medical schools, with the exception of Howard and Meharry, came largely as a result of the findings and impact of the Flexner Report on Medical Education (1910).[7] Not only did this study lead to the closing of the majority of black medical schools located in areas heavily populated by blacks, but it also shaped the condescending and stereotypical view that the medical profession maintained throughout the years between World Wars I and II regarding the functions and "place" of black physicians.[8–10] In its relatively minute section on "The medical education of the Negro," the report stated that "the practice of the Negro doctor will be limited to his own race." While it recommended the "upbuilding" of Howard and Meharry, the report described the other five schools as "feeble, ill-equipped institutions" that were "wasting small sums annually and sending out undisciplined men, whose lack of medical training is covered up by the imposing M.D. degree." Worse still, according to the report, was the victimization of the black public by these black doctors, because blacks were "more easily 'taken in' than the white." The report argued that given the black race's tendency to avoid personal cleanliness and community sanitation, black physician aspirants should have "received a substantial education in which hygiene rather than surgery. . .is strongly accentuated."[7]

The report and its author's influence on the Council on Medical Education and on key medical and charitable philanthropic bodies like the Rockefeller, Carnegie, and Julius Rosenwald Foundations perpetuated a dual system for the training of black and white physicians. This approach stressed the elimination of inferior, or "low-standard," black medical colleges (and their white counterparts). It also emphasized greater aid to specific institutions, and projects to generate a black medical elite for service solely in the nation's black communities.[11,12]

BLACK PHYSICIANS IN NORTHEAST CITIES

While it is obvious that the termination of these educationally and financially weak black medical schools led to a decline in the gross number of black physicians nationwide, it is important to note the impact these closings had on northern cities such as Philadelphia and New York—urban areas that were experiencing phenomenal increases in black population during World War II. Three questions in particular should be addressed: What was the medical school background of black physicians in the urban North? Since all of the black medical schools were in the South, what role did predominantly white medical schools in the northern cities play in producing black physicians to

---

Address correspondence to Dr McBride, Assistant Professor, Departments of History and Afro-American Studies, SUNY-Binghamton, Binghamton, NY 13901.

serve northern communities? How did the supply of black physicians compare with that of white physicians? To answer these questions, information on the black and white physicians listed in the *American Medical Directory* for the years 1925, 1929, and 1938 was surveyed. (These years were chosen because they are characteristic of a period in the history of American medical education that has not been explored in depth by scholars. Moreover, during this period the ground work for contemporary race relations was laid as great masses of the country's southern-agrarian based black population migrated to the northern industrial centers.) In these three years, respectively, for Philadelphia there were 72, 98, and 108 black physicians and 3,430, 3,714 and 4,112 white physicians recorded in the directory.[13]

The data revealed that most of the black doctors in Philadelphia were graduates of black medical schools—60% in the 1925 group, 57.7% in 1929, and 64% in 1938. This preponderance of black medical school graduates among Philadelphia's black doctors was similar to overall patterns found in other, earlier surveys. For example, Lewis'[14] nationwide survey done in 1932 concluded that more than 69% of America's black physicians were black medical school graduates; Woodson's[15] 1934 survey (of a limited sample) placed this proportion at 59.9%; and Adams[16] established a figure of 83.3% in 1936.

However, when the years of graduation as well as the types of medical schools are considered simultaneously for Philadelphia's pre-World War II black doctors, other outgrowths of medical school segregation can be observed. For example, the decrease in the proportion of Philadelphia's 1929 black physician group who graduated from black as opposed to white medical schools can be explained on the basis of the years in which these black physicians graduated from medical school (Table I). In 1925, there was a total of 43 black physicians in Philadelphia who had graduated from black medical schools. Of these 43 physicians, 7 had graduated from 1882 to 1900, and 19 from 1901 to 1910. The peak in the number of blacks graduating from white medical schools during this time occurred from 1911 to 1920. Of all 29 black physicians in Philadelphia during 1925 who had graduated from white schools, most (13) had done so from 1911 to 1929. Similarly, the majority of the city's black physicians practicing in 1929 and 1938 who were graduates of white schools also graduated during the decade of 1911–1920.

TABLE I. Distribution of Black Physicians in Philadelphia in 1925, 1929, and 1938 According to Year of Graduation and Type of Medical School

Year of Graduation	No. of Black Physicians in Philadelphia from Predominantly Black Medical Schools				No. of Black Physicians in Philadelphia from Predominantly White Medical Schools			
	1925	1929	1938	Total	1925	1929	1938	Total
1882–1900	7	6	7	20	4	5	2	11
1901–1910	19	19	12	50	8	9	8	25
1911–1920	11	12	11	34	13	14	16	43
1921–1930	6	19	21	46	4	14	9	27
1931–1937	—	—	18	18	—	—	4	4
Total	43	56	69	168	29	42	39	110

Of the 110 black physicians in Philadelphia between 1925 and 1938 who graduated from white medical schools, most (43) had attended those schools during the World War I decade. On the other hand, the number of graduates of black medical schools dropped for all three groups of physicians from 1911 to 1920. Of all 178 black physicians in Philadelphia from 1925 to 1938 who were black medical school graduates, a majority (96) completed study from 1901 to 1910 and from 1931 to 1937 and fewer from 1911 to 1920.

The relative rise in the number of black physicians in Philadelphia who had attended white medical schools from 1911 to 1920 and the concomitant lower number of black medical school graduates during this same period has a twofold explanation. First, there was the comparative liberalization on the part of predominantly white medical schools in admitting blacks during World War I.[13] This more liberal attitude apparently stemmed from the increased sympathy among certain American politicians and educators toward the concept of improved professional training opportunities for blacks—due to the enthusiastic service of blacks in World War I. In addition, the abrupt growth in the concentration of impoverished blacks during World War I throughout the nation's larger cities—where many medical schools were located—seemed to motivate these schools to make a greater effort to educate some black physicians. Although the predominantly white schools admitted only one or two black students per entering class, it was a gain over strict segregation. Another factor behind the dip in the number of black medical school graduates during 1911 to 1920 relates to the closing of black medical schools. The extinction of these schools reduced the availability of training outlets for black physicians, with a "delayed" impact in Philadelphia after the World War I decade. The effects of black medical school closings, as well as other important trends, are reflected in the institutional sources for the medical education of black physicians in the large cities during the decades between World Wars I and II.

About one in five of Philadelphia's black doctors during 1925 who had attended black medical schools had gone to Leonard Medical College or the University of West Tennessee Medical Department, Memphis. In the 1938 black physician group, only one in 10 of the city's black medical school graduates were alumni of Leonard or West Tennessee.[17] In fact, by 1938 two larger trends were evident that indicate a deepening segregation in medical education and an overall decline in the number of the city's black physicians. First, the number of black physicians in Philadelphia who had graduated from the city's predominantly white medical schools between 1929 and 1938 was declining. Second, the supply of black physicians in Philadelphia was becoming increasingly, indeed overwhelmingly dependent on the only two surviving black medical schools, that is, Howard and Meharry. In 1929, nearly 49% of the black medical practitioners in Philadelphia were graduates of these schools, and in 1938 that number rose to 57% (Table II).

TABLE II. Medical Schools of Black Physicians in Philadelphia in 1925, 1929, and 1938

		1925		1929		1938	
	Medical School	No.	%	No.	%	No.	%
Predominantly black	Howard U. Medical School	32	44.4	41	41.8	55	50.1
	Meharry Medical College	3	4.2	7	7.1	8	7.4
	Leonard Medical School	7	9.7	7	7.1	5	4.6
	West Tennessee Medical College	1	1.4	1	1.0	1	0.9
Predominantly white in Philadelphia	U. of Pennsylvania	10	13.9	11	11.2	10	9.2
	Temple U.	9	12.5	13	13.2	15	13.8
	Thomas Jefferson U.	3	4.2	3	3.0	2	1.9
	Medico-Churgical College*	3	4.2	3	3.0	2	1.9
	Hahnemann U.	1	1.4	2	2.0	1	0.9
	Woman's Medical College	1	1.4	4	4.0	—	—
Predominantly white outside Philadelphia	College of Physicians and Surgeons (MA)	1	1.4	1	1.0	1	0.9
	American Medical College (MI)	1	1.4	1	1.0	1	0.9
	U. of Michigan	—	—	2	2.0	—	—
	U. of Toronto (Canada)	—	—	1	1.0	1	0.9
	Queen U. (Canada)	—	—	1	1.0	—	—
	U. of Illinois	—	—	—	—	1	0.9
	Harvard U.	—	—	—	—	1	0.9
	College of Physicians and Surgeons (NC)	—	—	—	—	1	0.9
	Total	72	100%	98	99%	105	96%

* Operated only through 1918.

The decline in black student enrollment in Philadelphia's medical schools during the late 1920s and 1930s was part of a nationwide trend. During the nine years between 1927–1928 and 1935–1936 there was a drop in the number of black medical students in the United States from 497 to 369.[18] This decrease occurred despite the fact that American medical schools were enrolling increasing numbers of native and foreign-born white students.[19–21]

An examination of New York and other cities throughout the nation demonstrates that by the mid-1930s there was a strong connection between the availability of black-controlled medical education for blacks in certain locales and the number of black physicians found practicing in these locales. A case in point is the survey conducted by Lewis[14] in 1935 on the number and geographic distribution of the nation's black physicians. Lewis found that Washington, DC had almost twice as many black physicians (271) as did New York City (151), although the latter city had a black population three times larger than the former. He attributed this to the location of Howard Medical School in Washington, DC. Likewise, the city of Nashville, in which Meharry Medical College was founded, had 77 black physicians among its black population of about 40,000.[14]

The failure of the medical schools in cities like Philadelphia to educate a substantial number of blacks during the late 1920s and 1930s had far-reaching negative effects. Coupled with the economic barriers in medical education confronting blacks, the de facto segregation of the medical schools in the northern industrial states contributed to making Pennsylvania and New York the two worst states in the North in terms of the ratio of black physicians to black population.[22] Lewis' survey included findings on the black population, black doctors, and the population per physician ratio for nine northern states. These states were chosen because they had black populations of 30,000 or more, according to the 1930 census. He found Pennsylvania's ratio in 1932 to be 2,552 black residents to one black doctor; in New York, the ratio 2,281:1 was not much better. This placed these two states last behind seven other states: Illinois (991:1), California (1,081:1), Massachusetts (1,114:1), Indiana (1,417:1), Michigan (1,448:1), New Jersey (1,498:1), and Kansas (1,896:1).[14]

The decline in availability and quality of medical education for blacks during this period is a legacy from which the nation's black health care community has not yet recovered. As the black population was spiraling upwards as World War II approached, the pool of black physicians was diminishing. In 1920, when the black population was roughly 10.5 million, there were 3,855 black physicians; in

1942, although the black population had increased to about 12.9 million, the number of black physicians had dropped to 3,810; and in 1948 it dropped even lower—to 3,753.[23] On the other hand, the number of white physicians had increased from about 156,000 in 1931 to 176,000 in 1942 and 193,000 in 1948.[23,24]

The patterns in black physician to black population ratio observed in the 1930s were strengthened throughout the 1940s and 1950s, as New York City and other northern urban centers replaced the South as the primary residence for black Americans. In 1942, New York City and other Northeast cities ranked far behind Washington, DC and St Louis in black physician availability. Cornely[24] attributed this development to the strong local impact of "two important Negro medical centers. . .Howard Medical School, which graduates close to 50% of the Negro physicians in this country. . .while in St. Louis [there is] the second largest of all Negro hospitals, H.G. Phillips, a municipal institution with over 700 beds." In New York City during 1954, the number of black physicians stood at 305 (2% of all physicians in the city), which amounted to only a 1:2,754 ratio compared with the 1:480 figure for whites. This placed the city ninth among the 14 cities with the largest black populations. By contrast, that same year Nashville and Washington, DC ranked first and second in black physician:black population ratios: 1:1,235 and 1:1,674, respectively.[25]

RENEWED COMMITMENT

In conclusion, it must be stressed that before American medical institutions underwent their initial nationwide integration during the 1950s and early 1960s, the training and supply of black medical manpower was decreasing in quantity. In light of such a tremendous shortage in black medical personnel that had been half a century in the making, the changes predominantly white medical schools underwent during the civil rights decades—the 1950s and 1960s—turned out to be no more than cosmetic. As medical historian Sorenson[26] has pointed out, "during the period 1951–52 to 1961–62, when ostensibly many. . .racial barriers [in medical education] were being dropped, the number of black students enrolled at predominantly white medical schools grew by a mere two students, from 162 to 164." When black population growth is taken into account, Sorenson emphasized, "the burgeoning of the ranks of the medical profession by blacks was greater from 1900 to 1930 than from 1930 to 1967."[26]

It was only during 1968 to 1972, at the height of the government, corporate, and public preoccupation with black civil unrest and the assassination of Martin Luther King, Jr, that the nation's predominantly white medical schools departed from their traditional path of de facto segregation. For the first time, these institutions undertook dramatic policy and program changes geared toward increasing substantially black enrollment as well as black faculty and administrators. By 1972, virtually all of America's medical schools had some type of program for increasing black student enrollment.[27]

The gains for blacks in American medical schools during the 1970s were significant. However, the longer view presented in this analysis as well as recent trends should suggest cause for concern. Developments stemming from the Bakke decision (in which the Supreme Court ruled to prohibit quotas based solely on race in the selection of students for admission to universities) and the weakening of governmental policies to enforce integration in the education and employment of minority health professionals are turning back the clock of progress. This essay should not encourage a hearkening back to the segregated medical education characteristic of the pre-1950s. However, this study recommends a closer look at the efforts it took to educate even a moderate number of black physicians as well as the merits of traditionally black medical schools. Lastly, this overview should reveal the greater obligation all medical education institutions must assume in the decades ahead to establish racial parity in the medical profession.

REFERENCES

1. Sullivan LW: Preface, in Hanft RS, Fishman LF, Evans WJ: *Blacks and the Health Professions in the 80's: A National Crisis and a Time for Action.* Association of Minority Health Professions Schools, 1983, p vi.
2. Pettigrew TF: *A Profile of the Negro American.* Princeton, Van Nostrand, 1964, p 99.
3. Lerner M, Anderson OW: *Health Progress in the United States.* Chicago, University of Chicago Press, 1963, p 11.
4. McShane MG: *Medical Schools in the United States—A Descriptive Summary.* Washington, DC, Association of American Medical Colleges, 1977, p 9.
5. Morais HM: *The History of the Negro in Medicine.* New York, Publishers Co, 1968, p 62.
6. Ibid., pp 60–66.
7. Flexner A: *The Flexner Report on Medical Education in the United States and Canada, 1910—A Report to the Carnegie Foundation for the Advancement of Teaching,* bulletin no. 4. New York, Carnegie Foundation, 1910, pp 180–181.
8. Morris, op. cit., pp 62, 66, 89–90.
9. Daley W: AAMC milestones in raising the standard of medical education. *J Med Educ* 1965; 40:321–328.
10. Hudson RP: Abraham Flexner in perspective: American medical education 1865–1910. *Bull Hist Med* 1972; 56:545–561.
11. Hine DC: The pursuit of professional equality: Meharry Medical College, 1921–1938, a case study, in Franklin VP, Anderson JD (eds): *New Perspectives on Black Educational History.* Boston, GK Hall, 1978, pp 173–192.
12. Summerville J: *Educating Black Physicians: A History of Meharry Medical College.* University, AL, The University of Alabama Press, 1983, pp 51, 53–54.
13. McBride D: *Black Health Care Labor and the Philadelphia Establishment: 1910–1965,* dissertation. New York, Columbia University, 1980.
14. Lewis JH: Number and geographic location of Negro physicians in the United States. *JAMA* 1935; 104:1272–1273.
15. Woodson CG: *The Negro Professional Man and the Community.* Washington, DC, ASALH, 1934, p 85.
16. Adams NPG: Sources of supply of Negro health personnel: physicians. *J Negro Educ* 1937; 6:468–476.
17. Morris, op. cit., pp 89–90.
18. Adams, op. cit., pp 470–471.
19. Ibid., p 471.
20. Steward CT, Siddayao CM: *Increasing the Supply of Medical Personnel.* Washington, DC, American Enterprise Institute for Public Policy Research, 1973, pp 16–17.
21. Richmond JB: *Currents in American Medicine: A Developmental View of Medical Care and Education.* Cambridge, MA, Harvard University Press, 1969, p 6.
22. Adams, op. cit., p 470.
23. Johnson JL: The supply of Negro health personnel—physicians. *J Negro Educ* 1949; 43:346–356.
24. Cornely PB: Distribution of Negro physicians in the United States in 1942. *JAMA* 1944; 124:826–830.
25. Reitzes DC: *Negroes and Medicine.* Cambridge, MA, Harvard University Press, 1958, pp 386, 389.
26. Sorenson AA: Black Americans and the medical profession. *J Negro Educ* 1972; 41:337–342.
27. Wellington JS, Montero P: Equal opportunity programs in American medical schools. *J Med Educ* 1978; 53:633–639.

Eugene Percy Roberts: black physician and leader

DENNIS C. DICKERSON, PHD

Most black physicians in the early 1900s, while actively engaged in the practice of their profession, assumed additional responsibilities as black leaders. The limited availability of educated and financially independent spokesmen to advocate racial advancement and to support organizations that aimed to improve the social and economic condition of blacks made these physicians prime candidates for leadership roles. Some of them became outspoken critics who believe that protest and militant action can eliminate racial segregation and discrimination. Others believed that their patient and inconspicuous participation in important groups and institutions devoted to the betterment of the nation's largest racial minority would free blacks from the constraints of second-class citizenship.

One such black physician was Eugene Percy Roberts, who practiced medicine in New York City for nearly 60 years. A distinguished leader in religious and educational affairs, Roberts was a supporter of Booker T. Washington, an influential black leader between 1895 and 1915. Roberts, who helped to establish the National Urban League, also belonged to the New York City school board and supported numerous efforts to improve the health of black New Yorkers.

Roberts was born in Louisburg, NC, on Oct 5, 1868. The Civil War had freed his parents, Weston and Olivia Yarborough Roberts, from slavery only three years before his birth. One of his eight siblings, Charles H. Roberts, became a New York City dentist, alderman, and a Republican nominee for Congress in 1924. The aspiring physician enrolled in Lincoln University in Pennsylvania in 1887. The school was established originally as Ashmun Institute in 1854 to train black missionaries for Africa; the white Presbyterian sponsors who renamed it in 1866 wished to educate black teachers, preachers, and physicians to serve southern blacks. At this strict Calvinist institution, Roberts imbibed the fervent Presbyterianism of his professors and responded to their exhortations to help less-fortunate blacks.

When he earned his bachelor of arts degree in 1891, he did not return to the South to attend a black medical school. Instead, he studied at the predominantly white New York Homeopathic Medical College and Flower Hospital in New York City. He graduated in 1894 with a medical degree. Although his professors at Lincoln University preferred that he and other graduates pursue their careers in the South, Roberts remained a part of New York City's burgeoning black population.[1]

Roberts' professional services were much needed in New York, a state that in 1910 had 134,191 black people but fewer than 100 black physicians. A specialist in heart and lung diseases, Roberts extended his medical activities to include work with various clinics and health agencies and in important medical investigations. He was a medical inspector for the New York City Board of Health from 1898 to 1912, and in later years he gave radio talks on tuberculosis for the agency. He lectured on infant care for the city's public schools, conducted studies on children's diseases, and served as chief physician at the St Cyrian's Babies Clinic. In 1913, he conducted an inquiry for the National League on Urban Conditions, a forerunner to the National Urban League, on the treatment of blacks in city convalescent homes; he wished to determine whether a black facility was needed.[2,3]

EUGENE PERCY ROBERTS, MD

Mrs Sophy H. Cornwell, Special Collections Librarian, Langston Hughes Memorial Library, Lincoln University, assisted in locating a photograph of Dr Roberts, and Dr John Reichert, Dean of the Faculty, Williams College, assisted in reproducing it.

Roberts' affiliation with black medical organizations led to other professional opportunities. Although he, like other black physicians, belonged to the predominantly white New York County Medical Society, Roberts devot-

---

Address correspondence to Dr Dickerson, Associate Professor, Department of History, Williams College, Williamstown, MA 01267.

ed most of his energies to the National Medical Association (NMA), a group of black doctors, dentists, and pharmacists organized in 1895 in response to racial discrimination in the American Medical Association and its affiliates. He served the NMA as New York State vice president and as an officer in Manhattan's Dunham Club. Membership in the NMA provided Roberts with a forum for the presentation of papers and the publication of articles related to his medical practice. Influenced by the growing emphasis on scholarship and research among American physicians, Roberts expressed pride in the Dunham Club and its "very good scientific meetings." In 1911, for example, in his role as president he stressed that "it is simply useless to practice dentistry or medicine in the ordinary way. We must discover something. We must discover some cure for some particular disease. In other words, we must do some research work, if we want to make history." His studies on the causes and prevention of tuberculosis, infant gastroenteritis, and cardiac arrhythmias appeared in the *Journal of the National Medical Association*.[4-9]

Roberts also participated in numerous other activities. He belonged to Booker T. Washington's National Negro Business League. League members believed that when the economic interests of blacks in business and the professions were advanced, the entire race benefitted. Additionally, Roberts helped to found the National Urban League in 1911. The organization grew to include numerous affiliates, mostly in the North. Its purpose was to find jobs and to provide a range of social services to urban blacks. Roberts, a financial contributor and executive board member, chaired the industrial committee of the New York affiliate.[6,10-12]

An active churchman, Roberts helped to found St James Presbyterian Church in 1895. St James became one of the largest black churches in New York City. He also spoke frequently to religious groups. In 1919, he gave the commencement address to the graduating class of the Lincoln University Theological Seminary. In 1928, he spoke at the New York Annual Conference of the African Methodist Episcopal Zion Church, and in 1932 he lectured in Harlem to the Interdenominational Preacher's Union on the relation of the Church to health.[13-17]

Roberts believed that politics, like the church, offered opportunities for racial advancement. At a time when most blacks voted consistently for the Republican Party, Roberts, a political maverick, left the GOP for the Democratic Party. Roberts joined the United Colored Democracy, a black Democratic group organized in 1898. He also started an affiliate organization called the Democratic Club of Negro College Men in New York City. These political activities paid off in 1917, when Democratic Mayor John Purroy Mitchel named him to the school board. Roberts' party connections, together with recommendations from the National Urban League and from officials and benefactors of the Tuskegee Institute—who were familiar with his ties to Booker T. Washington—helped him win the position. His term expired in 1922.[6,18]

Roberts' involvement with the school board prepared him to assume the presidency of the Lincoln University Alumni Association in 1925. When the trustees elected a white Presbyterian minister with alleged Ku Klux Klan connections to the Lincoln presidency, Roberts led the alumni in protest and compelled the new president to withdraw. During this controversy, the alumni reminded the Presbyterians who controlled the university that no blacks belonged to the Board of Trustees. In 1926, the board capitulated and invited Roberts to become Lincoln's first black trustee. While Roberts served as chairman, Horace Mann Bond became, in 1946, Lincoln's first black president.[19-21]

Roberts, who died in 1953, typified black physicians who accepted leadership roles. Cognizant of the many needs of New York's largest racial minority, doctors like Roberts participated in organizations and activities devoted to black advancement. For Roberts and many of his professional colleagues, a medical degree was a symbol of professional achievement and an invitation to racial leadership.

REFERENCES

1. "Dr. Roberts' rites plain, impressive." *New York Age*, January 17, 1953, p 3.
2. "Eugene P. Roberts." *National Cyclopedia of the Colored Race*. Montgomery, AL, National Publishing Company, 1919, p 225.
3. Eugene Kinckle Jones to Booker T. Washington, February 19, 1913, in Harlan LR, Smock RW (eds): *The Booker T. Washington Papers*, vol 12, 1912–14, Urbana, IL, University of Illinois Press, 1982, pp 118–119.
4. "Doctors go to Boston." *New York Age*, September 3, 1908, p 10.
5. "Doctors meet in 'Hub' city." *New York Age*, August 26, 1909, pp 1, 4.
6. "Roberts named board member." *New York Age*, January 11, 1917, pp 1–2.
7. Roberts EP: Causes and prevention of tuberculosis. *J Nat Med Assoc* 1909; 1:81–84.
8. Roberts EP: Infantile gastro-enteritis. *J Nat Med Assoc* 1912; 4:16–19.
9. Roberts EP: Cardiac arrhythmias. *J Nat Med Assoc* 1932; 24:13–18.
10. "Tributes from New Yorkers to dead leader." *New York Age*, November 18, 1915, p 7.
11. "Extending National Urban League work." *New York Age*, October 5, 1916, p 8.
12. "Dr. E. P. Roberts pays fine tribute to Dr. Washington." *New York Age*, February 5, 1927, p 3.
13. "St. James Presbyterian Church," Sunday, June 26th, 1983, 88th anniversary Sunday, 1895–1983. (Courtesy of Reverend Lenton Gunn).
14. *Lincoln University Herald*, 1919; 23:6.
15. *The Star of Zion*, June 19, 1928.
16. "Dr. E. P. Roberts speaks to ministers' association." *New York Age*, May 7, 1932, p 7.
17. "Interdenominational group has reception for Dr. E. P. Roberts." *NJ Afro-American*, June 24, 1950, p. 10.
18. "Negro to get place on education board." *New York Times*, January 7, 1917, part I, p 10.
19. "Chairman Laird of Lincoln trustees defends himself in letter to alumni president against charges of prejudice." *New York Age*, July 31, 1926, p 102.
20. "Lincoln seeking new president." *NJ Afro-American*, October 4, 1941, pp 1-2.
21. Branson HR: "Introduction," in Bond HM: *Education for Freedom: A History of Lincoln University, Pennsylvania*. Princeton, Princeton University Press, 1976, pp xiii–xv.

The black woman physician

NORMA J. GOODWIN, MD

The assumption by some individuals that any discussion of women in the professions will be political in nature undermines the opportunity for a serious hearing; and a serious hearing is very much in order given the rapidly changing gender mix of the medical profession in America. Consideration of the emergence and contributions of black women in medicine also necessitates acknowledgment of the fact that they have been subjected, since the outset, to dual obstacles—as members of a profession which has been and continues to be white and male dominated. In accepting this reality, one can better appreciate the challenge to black women physicians, and the nature and scope of their contributions to the profession and society. The insights thus gained provide a framework for understanding the greater contributions that can be made as a result of further enlightenment and reform in both the health professions and society at large.

Although there are scant data available on black women physicians per se, it is known that as late as 1920 only 65 black women physicians had been documented in the United States.[1] One half-century later, in 1970, there were 1,051,[2] and as recently as 1980, the number had increased to 3,153.[3] Available data on blacks in medicine and women in medicine, however, provide a telling story of their own about the milieu in which black women in medicine have emerged.

In 1980, of the 357,842 physicians in the United States, 13,243, or less than 4%, were black, although blacks make up approximately 12% of the American population. In 1981, less than 5% of all physicians-in-training in the United States were black.[4] According to the Association of American Medical Colleges (AAMC), for the academic year 1983–1984, the total number of black students in the 127 medical schools in the United States was 3,892, out of a total of 56,167, or 7%.[5] The problem of underrepresentation of blacks in medical schools does not stop at the student level. In 1981, all ethnic minorities combined comprised 2% of the 31,000 full-time faculty members in American medical schools.[5] The figures for women (regardless of race) also indicate substantial underrepresentation, especially in leadership roles. In 1981, women comprised only 16.4% of faculty with the rank of professor. In the same year, 28% of women faculty members of at least 47 years of age had attained the rank of professor, as compared to 61% of men.[5] Women are few and far between in the higher echelons of medicine, although they comprise 70% of the health workforce.[6]

The preceding statistical data, as well as the following biographical sketches of five pioneering black women physicians are not merely of demographic and historic interest. They put into perspective the struggles and levels of achievement of black women physicians. They also illustrate the obstacles black women physicians confront as they advance in their profession and contribute to society. These women participate with others of their race, and with women of all races, in the ongoing struggle for equal opportunity. Many also have the additional responsibilities that are shared by professional women who are also wives and mothers.

HISTORICAL PERSPECTIVE

The history of black women in medicine is as old as civilization, having its tap roots in ancient Africa. The pharmacological and surgical knowledge and skills that existed in Memphis, Benin, and Kano,[7] survived a devastating forced transfer to a land where they were both exploited and denigrated.[8]

The image of the handkerchief-headed "granny" midwife still lingers. It is a symbol thought by many to best represent the hundreds of black women who practiced as lay midwives and nurses throughout the South during the Civil War, caring for the sick and wounded of both sides. One marvels at the tenacity, restraint, and humanity of such women.

To appreciate the struggle of black women physicians, one need only consider examples of the barriers they have surmounted, and the numerous achievements they have made. Dr Rebecca Lee, reported to be the first black woman physician in the United States, received her medical degree from New England Female Medical College, Boston, in 1864, one year before the end of slavery.[9] Dr Rebecca J. Cole soon followed Dr Lee, receiving her medical degree from Woman's Medical College of Pennsylvania, Philadelphia, in 1867.[9] These women became bona fide physicians at a time when blacks were considered chattel, and women hardly better; indeed, only a few white males could hope to enter the profession.

Dr Susan Smith McKinney Steward, the third black woman physician in America and the first in New York State, graduated from the New York Medical College and Hospital for Women in 1870.[10] In 1888, when she completed a postgraduate course at Long Island Medical College Hospital, she was the only woman in the school. Her acceptance at a time when postgraduate work was almost impossible to attain by black physicians was based in large part on the quality and impact of her prior accomplishments in the profession. In 1881, she co-founded the Women's Hospital and Dispensary at Myrtle and Grand Avenues in Brooklyn.

Although Dr May E. Chinn's early hopes for a medical career were discounted by her father, a former slave, she was able to attend college with the help of her mother. She went on to become the first black woman graduate of the

---

Dr Goodwin served as Speaker of the House of Delegates of the National Medical Association from 1977 to 1979.

Address correspondence to Dr Goodwin, President, AMRON Management Consultants, Inc, 3020 Glenwood Rd, Brooklyn, NY 11210.

New York University Bellevue Medical Center. In 1926, she became the first black woman physician to intern at Harlem Hospital, which was a predominantly white institution. When Dr Chinn was not able to obtain hospital privileges, because of her race, she set up practice with several black male physicians in a brownstone in Harlem. Dr Chinn later recalled of her partners that they were divided into three groups: "those who acted as if I wasn't there; another that took the attitude 'what does she think she can do that I can't do?'; and the group that called themselves supporting me by sending me their night calls after midnight." [11] Dr Chinn let neither these nor other slights and obstacles deter her from responding to the needs of her patients, or from later pursuing her interest in cancer research and treatment. Based on her belief that her patients were victims of society as much as of disease, she studied for and obtained a Masters degree in public health from Columbia University. Hospital doors in New York, however, remained closed to her to pursue her interest in the early detection of cancer. Through persistence, she received an invitation to join the staff of the Strang Clinic. Although she worked in cancer research at the clinic for 26 years, her name was never included on the roster.

MAY E. CHINN, MD

LENA F. EDWARDS, MD

Dr Lena F. Edwards, now in her 80s and retired from active practice, is considered a living legend by many black women in medicine. She is still active in the kind of humanitarian pursuits for which she was awarded the Presidential Freedom Medal in 1964. Dr Edwards began her career in the 1920s in Jersey City, NJ, where she remained for more than 30 years, serving inner city residents and working on the staff of Margaret Hague Maternity Hospital. Her early experiences there mirrored those of many other black physicians and women physicians at the time. Upon being told by the chief of staff of the difficulty she was initially felt to be causing the hospital because of two "handicaps" ("first, you're a woman, and secondly, you're a Negro"), she retorted, "That is quite true, but it so happens that God gave me both of those characteristics, and I don't think that there's a human being who has the right to judge me for what He did. You'll find that I do my work properly, so you'll have to find a better excuse for not wanting me." [12] When Dr Edwards finally left her position at the hospital, she had established a reputation for outstanding obstetrical work, including instituting innovative practices such as the use of minimal or no anesthesia during deliveries. She subsequently became an associate professor of obstetrics and gynecology at Howard University, in Washington, DC, where she had received her medical degree. At the age of 60, she resigned her teaching post to establish Our Lady of Guadalupe Clinic, a 25-bed hospital and health center, in Hereford, Texas, which eventually served a population of 5,000 migrant workers.[12]

The achievements of Dr Edwards and her forerunners represent only a glimpse at the accomplishments of black women physicians. It is imperative that funding be obtained to record the history of the many outstanding black women physicians of the twentieth century. This would be of great historical significance and would be an inspiration to young women and men of all races.

TODAY'S LEADERS

During the past two decades, more women have assumed important leadership roles in the National Medical Association (NMA)—which has represented the interests and concerns of black physicians and their patients for more than 80 years. Although the NMA, as well as the American Medical Association (AMA), continues to be dominated by men, five women have served on the NMA's Board of Trustees, including two on the Executive Committee of the Board. Vivian Pinn-Wiggins, MD, (Chairperson of the Department of Pathology, Howard University) is expected to become the second woman elected as Speaker of the House of Delegates of the NMA in 1985, and Edith Irby Jones, MD, was installed as the first woman president of the NMA.

Black women physicians have been organizing women's groups to exchange information and address issues of mutual concern. The first major group, the Susan Smith McKinney Steward Medical Society, founded by Dr Muriel Petioni in the mid-1970s, has approximately 150 members in New York, New Jersey, and Connecticut. In 1984, it was approved as a chapter of the NMA. Organized groups of black women physicians also exist in Los Angeles and Oakland, California, and in Atlanta. Because support for affiliating with established organized medical groups varies among these groups, it is important that the NMA and AMA develop programs for addressing the special concerns of women physicians. Indeed, the NMA has established a Council on Concerns of Women Physicians.

HEALTH POLICY CONCERNS

Black women physicians, as well as their black male colleagues, are especially concerned about a number of evolving health policies, including those related to Medicaid and Medicare, Diagnosis Related Groups (DRGs), and medical school and post-graduate admission and retention.

A disproportionate percentage of patients of black physicians who practice in or near inner city areas are assisted by Medicaid or Medicare. Many other community residents lack health insurance coverage and are unable to pay the full cost for their care. Thus, consistently low Medicaid reimbursement rates and projected decreases in Medicare benefits are issues of special concern to these physicians. The escalating cost of malpractice insurance presents an additional financial burden because of the physician's inability to pass the full cost to the patient through higher fees. Health policies related to these issues threaten the financial viability of the private practices of many black physicians.

DRGs and similar regulations may also have an unduly negative impact on those black physicians who practice in high risk areas, for their patients less frequently seek preventive care, and often defer too long seeking care for illness. As a result, some of these patients may require hospital stays beyond reimbursable periods according to their DRGs, making them "less desirable patients." As a secondary effect, their physicians may encounter greater difficulties in obtaining hospital privileges. This possibility requires close monitoring in regard to the assurance of access for high risk patients and hospital privileges for black physicians.

Another special concern of black women physicians is that recent increases in the number of women admitted to medical schools (nearly 30% of medical students are now women) appear to have come, at least in part, at the expense of overall minority admissions. The latter percentage has either levelled off or decreased in many schools. Given the inadequate number of black male and female physicians, the production of both in increased numbers requires continued pursuit. Finally, as a greater percentage of black physicians are women, there may be an even greater scarcity of black physicians in those specialties which continue to be overwhelmingly male dominated, such as surgery and the surgical specialties. While the scarcity of women in some specialties undoubtedly relates to both lack of access and women's perception of lack of access, the reality of their small numbers portends a special problem for the black population which must be addressed, given the fact that in the academic year 1983–1984, 44.9% of black medical students were women (as compared to 29.4% among white students).

CHALLENGES FOR THE FUTURE

Black women physicians must be afforded increased opportunities to realize the potential which they have amply demonstrated—as practitioners, academicians, researchers, and administrators; their numbers must increase, but not at the expense of the also sorely needed increased number of black male and other minority physicians; and they must be recognized as full, contributing members of the medical profession. This inadequate recognition remains a problem in most, if not all, predominantly black as well as predominantly white health agencies and institutions. Finally, it is hoped that the response to these challenges will be speedy and forthright, as will support for health policies which assist black physicians who remain committed to providing private, personalized care to the residents of disadvantaged, predominantly black communities. As these challenges are met, the long submerged potential of black women physicians for even greater contributions to the profession, and to society, will be realized.

Acknowledgment. The Schomburg Center for Research in Black Culture, the largest archive of black cultural and historical information in the United States, served as a valuable research resource in the preparation of this article.

REFERENCES

1. Brown SW: Colored women physicians. *Southern Workmen.* Hampton, VA, Hampton Institute, 1923.
2. 1970 Census of the Population. Detail of the occupations of employed persons by race and sex. Washington, DC, US Census Bureau.
3. 1980 Census of the Population. Detail of civilian labor force by sex, race, and Spanish origin. Washington, DC, US Census Bureau.
4. Pinn VW: The underrepresented in graduate medical education and medical research. *Public Health Reports* 1984; 99:53–58.
5. *1983–1984 Directory of Residency Training Programs.* Chicago, American Medical Association, 1983, pp 75–85.
6. Rodriguez-Trias H: The women's health movement, in Sidel VW, Sidel R (eds): *Reforming Medicine.* New York, Pantheon Books, 1984, p 113.
7. David R: *The Making of the Past: The Egyptian Kingdom.* New York, EP Dutton & Co, Inc, 1961, pp 114–116.
8. Rogers JA: *Africa's Gift to America.* New York, Helga M Rogers, 1961, pp 222–225.
9. Davis MW (ed): *Contributions of Black Women to America,* vol II. Columbia, SC, Kenday Press, 1982.
10. *Bulletin of the Medico-chirurgical Society of the District of Columbia, Inc.* 1949; 6:3.
11. Hunter-Gault C: "Black women M.D.'s: spirit and endurance." *New York Times,* November 16, 1977.
12. Scally, Sr. MA: *Medicine, Motherhood and Mercy: the Story of a Black Woman Doctor.* The Associated Publishers, Inc, Library of Congress Cat. No. 79-55656, 1979.

Minorities and New York medical schools

Past failures and the prospect for future successes

ROBERT K. WATTS, MSW, JAMES J. MCCORMACK, PHD

Between 1940 and 1970 the number of black physicians in New York State increased from approximately 300 to 1,069. However, the black population increased from 571,221 to 2,168,949. Thus, the ratio of black physicians to black people decreased from 1:1,904 to 1:2,029,[1] compared with the overall state physician-to-population ratio in 1970 of 1:477. This pattern, which continued into the 1980s, resulted in a much lower physician-to-population ratio in many minority* communities.

It is in minority communities where physicians are most needed. National and state data consistently show the health status indicators of nonwhites to be significantly poorer than those of the white population. In New York State, infant mortality is 62% higher among nonwhites than among whites, and the nonwhite, age-adjusted death rate exceeds the white age-adjusted death rate by 29%, according to the New York State Department of Health's Office of Biostatistics.

A 1980 New York State Education Department survey found that physician distribution in the state mirrors the racial and ethnic distribution of the state population.[3] A 1982 Association of American Medical Colleges (AAMC) survey indicated that 75% of minority graduates would consider practicing in a socioeconomically deprived area, while only 49% of nonminority graduates would consider such a practice.[4] Other studies also support the hypothesis that minority physicians are more likely to practice in a medically underserved area or to choose primary care specialties. Expanding the supply of minority physicians is consistent with state priorities to make primary care physicians more available and to improve care in medically underserved areas.

In 1970, the AAMC established the objective that by 1975–1976, 12% of the first-year medical school students would be black.[5] Minority first-year enrollment was 9.1% nationwide for 1975–1976. Black first-year enrollment was 6.8%, far short of the goal. New York State's first-year minority enrollment peaked at 8.3% in 1974–1975, then declined to 6.6%, virtually eliminating the gains made since 1960. In 1983–1984, minority enrollment rose slightly to 8.3% and was 8.9% in 1984–1985.

Between 1973–1974 and 1982–1983, first-year minority enrollment in allopathic medical schools in the state averaged 6%, considerably below the national average.[6] There were no minorities enrolled in New York's single osteopathic medical school as of October 1981.[7] Not until 1982–1983 did New York's first-year minority enrollment (9.3%) approach the 9.4% national average.[6] Nevertheless, this is considerably below the 17% of the state population that minorities represent. Clearly, both national and state efforts to approach parity for minorities have failed.

The Governor's Special Advisory Commission on Minority Enrollment in Medical Schools noted, in its 1982 report, that there are many reasons for minority underrepresentation in medical schools. The report characterized the problem as "complex" and "chronic," stating that "no single agent can be isolated as *the* cause of the problem."

This conclusion was accompanied by the Commission's observation that the problem has recently become exacerbated by a long-standing lack of leadership and focus on this matter.[7] In our view, the principal elements of the problem include the low level of institutional commitment in some medical schools, a limited pool of qualified minority applicants, and diminishing moral and political leadership. There is also the residual of societal racism.

Institutional commitment is the most important single factor in the recruitment and retention of minorities. Simply stated, institutional commitment is the firm resolve to recruit and retain minorities. This resolve is reflected in the policies and actions of an institution, from the boardroom to first-line supervisors. Institutional commitment may be manifested in many ways: minority representation on faculty and staff, sensitivity to minority preferences in the institution's facilities and programs, and minorities on the governing board of the institution. When there is strong institutional commitment, adequate resources are made available for an effective affirmative action program, and creative use is made of management and administrative tools to achieve results. Medical schools with a consistently high proportion of minorities tend to show a high level of commitment not present in schools with consistently low proportions of minorities.

The pool of qualified minority applicants to medical school must be enlarged to correct minority underrepresentation. Approximately 285 new minority first-year entrants are needed to approach parity, and the present applicant pool may be too small to produce this number. The total number of minority applicants is declining, while at the same time medical schools in the state are accepting proportionately fewer minority applicants.[6] In 1983–

Mr Watts served as staff coordinator for the Special Advisory Commission on Minority Enrollment in Medical Schools and editor of its report. Dr McCormack, who served as chairman of the consultant panel to the Commission, is the Executive Director, New York State Health Planning Commission.

Address correspondence to Mr Watts, Chief Health Planner, New York State Department of Health, Bureau of Health Resources Development, Corning Tower, Empire State Plaza, Albany, NY 12237.

* Minorities as used in this article means black Americans, mainland Puerto Ricans, native Americans and Mexican-Americans. We have adopted the concept developed by the Association of American Medical Colleges (AAMC) in 1969. In that year, an AAMC Task Force recognized that these groups were substantially underrepresented in medical schools and the medical profession, compared to their proportion of the population. Asians and other minorities were not included because historically they have not been underrepresented.[2]

There are limited data available on underrepresented minorities, particularly before 1970. However, blacks have historically made up more than 90% of the nonwhite, or minority, population of the United States. Although this picture is now changing, we believe the use of data on blacks, especially pre-1970 data, is an appropriate surrogate for other underrepresented minorities.

1984, 1,291 of the nationwide total of 3,440 minority applicants applied to medical schools in New York State; about one third were state residents. In that year, medical schools in the state accepted 218, or 16.8%, of minority applicants. First-year minority enrollment was less than 160 students.[6]

The minority applicant pool is influenced by factors such as family socioeconomic status and the quality of education and counseling in high school and college. Many capable minority students are channeled into other careers, because some counselors perceive them to be more accessible or appropriate for minorities.

Although a larger pool is needed, New York State medical schools are not enrolling their share of the existing pool. New York produces more than its share of physicians and specialists, and its medical schools receive 37.5% of minority applications nationwide.[6] The state also has one of the largest black populations in the country. Despite these numbers, New York medical schools have not been able to reach a 10% minority enrollment, and they graduate fewer than 1% of the nation's minority physicians.[4]

Medical schools are not solely responsible for this problem. There is a dearth of leadership and concern at every level, from the federal government to organized medicine. At the federal level, it is reflected in declining support for scholarship aid and withdrawal from affirmative action policies. At the state level, it is reflected in the failure of public officials to take action in response to the findings and recommendations of several public policy study groups and advisory bodies during the past few years. Since the Bakke decision (in which the Supreme Court ruled that quotas imposed strictly on the basis of race are unlawful), a backlash has set in, crippling moral and political leadership in correcting the effects of past discrimination. In such a negative climate, few medical schools are willing to be in the vanguard of affirmative action.

Organized medicine should follow the example set by the engineering profession and provide the leadership to develop programs and resources to expand the applicant pool and increase the proportion of minorities in medicine. The National Medical Association, a predominantly black organization, has stood virtually alone in its tangible and consistent efforts to increase the number of minority physicians.

Finally, there are the residual effects of institutional racism, which is endemic to our society. By its nature, institutional racism is covert and subtle. It is often unintentional and invisible, and frequently involves a failure to act or correct adverse actions, rather than explicit practices or acts. It can be as subtle as not accommodating ethnic food preferences in the cafeteria or as blatant as overt discrimination in the recruitment and promotion of staff. Medical schools, like other institutions, are not immune to the many forms of racism or the past effects of racism. Strelnick and Younge[8] observed that "medicine has not earned the exemplary professional status it often claims in regard to its own history of discrimination." Tracing institutional racism in medicine from 1948 to 1981, they conclude by quoting a 1981 Institute of Medicine study: "While *de jure* discrimination has ended, individual and institutional *de facto* discrimination persists." [8]

The Special Advisory Commission report reviewed several US Supreme Court and New York Court of Appeals decisions and offered conclusions regarding the legal context of affirmative action programs. The commission observed that race may be a factor, though not the sole factor, in determining admission to a program. Furthermore, if race is used as a factor, it must be done to serve a compelling or substantial interest. Finally, it appears that there are no violations of majority rights against which legal action can be taken as long as substantial state interests can be demonstrated in support of race-conscious policies to benefit minorities. If a statement of purpose were written for medical school subsidies and student assistance programs that indicated a compelling or substantial state interest in achieving greater minority medical school enrollment, the context for successful affirmative action efforts would certainly improve.

Conducting an affirmative action program requires commitment. This means a formally stated policy, dedicated financial and professional resources, stubborn persistence, and a strong competitive spirit. The Special Advisory Commission Report contains a positive picture for the future of a collaborative program designed and operated by medical schools and the state government. Recently, a thorough analysis by Manard and Lewin[9] answered the question of whether affirmative action intervention is necessary to increase the number of black physicians: "Research . . . strongly indicates that in the absence of a deliberate effort, the number of Blacks who apply, are accepted, and graduate from U.S. medical schools will not increase substantially."

The discrepancy between minority representation in the general population and in the state's medical schools should not exist in a society that proclaims that equal opportunity is a right of all its citizens and that moves its public policy leadership to correct the effects of past discrimination.

Desired public policy on this topic is clearly stated, including the goal of increasing the proportion of minorities receiving professional and higher-education degrees.[10] Gubernatorial messages on health and human services policy in 1981, 1982, and 1983 all cited the need for a program to increase minority student enrollment in New York medical schools. The medical schools express a similar goal. However, the decline in minority enrollment suggests the need for new public policies to reverse this trend. Bold new programs and objectives should be initiated. The following are some ways that lasting improvement in minority enrollment levels might be assured:

- Selected minorities in residency training should be groomed for faculty positions in medical schools. Minority faculty and staff role models are necessary to recruit and to retain minority students.
- Counseling and intervention programs should be designed jointly by medical schools and colleges to assist students in achieving greater success with the Medical College Admissions Test.
- Admissions criteria and processes in the schools must reflect a willingness to examine criteria beyond grades and academic success.
- Medical schools must ensure that a supportive environment with appropriate programs is in place. This includes faculty and staff who are sensitive to the experience of minorities.
- Schools expecting to attract and retain minority students must continue to offer financial aid.

The long-range prospects for change demand action of a sustained nature to enlarge the pool of qualified minority applicants to medical schools. The following recommendations are offered:

- A statewide program for identification, guidance, and recruitment of minorities for medical schools should be undertaken. This program would focus on identifying students in high school or early college years and would provide a track to draw larger numbers toward premedical programs and eventual application to medical school. Such a program would involve consortia of high schools, colleges, and medical schools in various regions of the state, perhaps funded through a combination of state and private-sector dollars.

  A proposal was made by the Governor in 1983 for such a program. It was passed by the Assembly in June 1984, but was not acted on by the Senate. Essentially the same proposal has been made by the Governor in 1985 and is under consideration by the legislature.
- In exchange for continued state financial support, medical schools should be required to submit performance goals for minority enrollment and graduation to the appropriate authorities.

Recruiting and retaining minorities in medical schools is not without problems or risk. Until fundamental defects in the society and educational system are corrected, minorities as a group will continue to be more poorly prepared for medical school than nonminorities. Data from the AAMC show that minority students have lower scores on the Medical College Admissions Test and are more likely to repeat classes and drop out before graduation.[4] However, the risks can be minimized by good tutorial programs and effective support systems. The societal rewards of more minority practitioners far outweigh the risks involved.

Underrepresentation of minorities in medical schools and the serious shortage of minorities in the health professions is a problem that must be addressed jointly by the public and private sectors. There is a great need for more health practitioners in minority communities. National and state data show health status indicators for minorities to be significantly poorer than for nonminorities. Studies show that minority physicians are more likely to practice in an economically deprived area than are nonminorities. Complex, interwoven factors have created the problem of minority underrepresentation in medical schools, and, as a whole, since 1975, the medical schools in the state have failed to significantly increase the proportion of minority enrollment.

Organized medicine should not remain on the sidelines of this issue. There is a crucial need to increase the role of minorities in every sector of the health care system. The limited success of medical schools in recruiting and graduating minorities reflects, to a large degree, the anemic affirmative action efforts by organized medicine.

The situation in New York State is critical but not yet terminal. A transfusion of strong commitment will reverse the situation. Organized medicine should grasp the hands of both academic medicine and government and lead both toward actions necessary to make equal opportunity in medicine a reality rather than an unfulfilled dream.

REFERENCES

1. Hall JY: *A Historical Study and Analysis of the Distribution of Black Physicians in New York State 1890–1970,* thesis. Rochester, University of Rochester, 1980.
2. *Report of the Association of American Medical Colleges Task Force on Minority Student Opportunities in Medicine.* Washington, DC, Association of American Medical Colleges, 1970.
3. *Physician Manpower Survey 1980–81.* New York, New York State Education Department, Office of Postsecondary Research, Information Systems, and Institutional Aid, 1982.
4. *Minority Students in Medical Education: Facts and Figures.* Washington, DC, Association of American Medical Colleges, 1983.
5. *Report of the AAMC Task Force on Minority Student Opportunities in Medicine.* Washington, DC, Association of American Medical Colleges, 1978.
6. Data files supplied to the New York State Health Planning Commission by the Association of American Medical Colleges, Washington, DC.
7. Watts RK (ed): *Report of the Special Advisory Commission on Minority Enrollment in Medical Schools.* Albany, New York State Health Planning Commission, 1982.
8. Strelnick H, Younge R: Affirmative action in medicine: money becomes the admissions criteria of the 1980's, in Siedel V, Siedel R (eds): *Reforming Medicine: Lessons of the Last Quarter Century.* New York, Pantheon, 1984.
9. Manard BB, Lewin LS: *Physician Supply and Distribution: Issues and Options for State Policymakers.* Washington, DC, National Center for Health Services Research, US Dept of Health and Human Services, 1983.
10. *1980 Regents Statewide Plan for the Development of Postsecondary Education.* New York, New York State Education Department, 1980.

FROM THE LIBRARY

According to the 1950 United States Census of Population, the total population of New York State as reported is 14,830,192. The Negro population of New York State as reported is 918,191. The total number of physicians in New York State is 29,750, and the total number of Negro physicians in New York State is 326. Thus, Negroes constitute approximately 6 percent of the total population of New York State, while the Negro medical profession constitutes slightly over 1 percent of the total number of physicians.

PETER MARSHALL MURRAY, MD
"The status of the Negro physician in New York State"
(*NY State J Med* 1955; 55:2980)

A glittering opportunity for colored physicians is seen in the fact that for each 4,000 Negroes, there is only one Negro doctor—which gives young men of the race a wide field for leadership and service.

That advice was given at the fifth annual Capital District Vocational Guidance Conference in Booker T. Washington Center in Albany in April by Dr. W. Adrian Freeman, secretary of the Manhattan Medical Society and staff member of Harlem Hospital.

(*NY State J Med* 1937; 37:1157)

Black doctors on television

J. FRED MACDONALD, PHD

He is the invisible doctor—the physician who is seldom seen and almost never practices medicine. As the central character in prime time television programming, the black doctor is a practical nonentity. Relegated to video oblivion, his treatment on television goes to the heart of American racial prejudice and the stereotypes that have helped it survive.

The predicament of the black American physician on television is summarized by the juxtaposition of two recent video offerings. In April 1984, "A Doctor's Story" was a made-for-TV film featuring Howard Rollins, Jr as a caring physician whose patients (many of whom were white) included a man who had a heart attack and a woman who tried to commit suicide. Although acclaimed by numerous television critics, the film generated neither a sequel nor a series.

Ironically, the hit program of the 1984–1985 television season has been "The Cosby Show"—a situation comedy focusing on the trials and tribulations of a black family, headed by an obstetrician. In earlier television series Cosby enjoyed great popularity in roles as a spy for the Central Intelligence Agency and as a high school coach. His current character, Dr Cliff Huxtable, has become the most successful black physician in the history of television. Yet he seldom is seen at the hospital, and rarely seems to practice medicine. Like a black Ozzie Nelson, Dr Huxtable really has no job except to be the father of a comedic family.

BILL COSBY IS BACK!
He's a family man whose main hope is that his children run away before he does.
THE Cosby SHOW
8PM 4

Advertisement for "The Cosby Show," which stars Bill Cosby as an obstetrician.

Black heroes have been scarce in television, and the black doctor as a leading character has had little exposure in the almost four decades since the medium became a popular instrument of mass entertainment and information.

Certainly, television has created a veritable pantheon of medical stalwarts—solid urban types such as Drs Kildare, Welby, Gannon, Quincy, and Casey—as well as benign eccentrics like "Doc" Adams of "Gunsmoke" and most of the cast of "M*A*S*H." In "Julie Farr, M.D." (aka "Having Babies") industry moguls even spotlighted a woman doctor. Yet all these medical heroes have been portrayed by white actors.

In its quest to amuse mass audiences and to gather the highest possible ratings and profits, commercial television has never supported a practicing black doctor in prime time drama. Until the civil rights movement reminded the television industry and white Americans that minorities comprise a significant proportion of the national population, the black doctor never existed in the medium. Cast as a neurosurgeon in the last season (1968–1969) of "Peyton Place," Percy Rodrigues was the first reoccurring black physician in evening television. Hal Frederick followed as one of five doctors-in-training in "The Interns" (1970–1971).

But television has always preferred blacks as buffoons rather than as serious dramatic characters. Thus, Cleavon Little jived and gambled his way through two seasons and three different formats on the situation comedy "Temperatures Rising" (1972–1974). In contrast, Louis Gossett, Jr lasted only six episodes as a cardiologist and central character of "The Lazarus Syndrome" (1979). And black female physicians have yet to be featured in either situation comedies or dramatic series.

Although more than 12% of the prime time audience are black, and 3% of the doctors in the United States are black, the percentage of black video physicians does not match these figures. In fact, if a minority doctor is portrayed in evening television, he has usually been the focus of racial tension or disparagement. When Archie Bunker was treated by a black physician in the mid-1970s, it created enough serio-comedy to fill an episode of "All in the Family." In an installment of "Sanford and Son," Fred Sanford, a black man, felt he would receive inferior medical care from a dentist of his own race. Even today on "Trapper John, M.D." and "St. Elsewhere" relatively little time and attention is given to the medical professionals portrayed by black actors.

Despite the legacy of cultural exclusion and racial prejudice encountered in prime time television, the medium has not been unmitigatingly hostile to the dramatic black physician—he has survived for more than a decade on daytime video, woven into the melodramatic story lines of some of the nation's favorite soap operas.

Often in the vanguard of mature representation and the probing of sensitive issues, network soaps have been hospitable to the black doctors. In the early 1970s James Earl

---

Dr MacDonald is the author of *Blacks and White TV* (Chicago, Nelson-Hall, 1983).

Address correspondence to Mr MacDonald, Professor, Department of History, Northeastern Illinois University, 5500 N St. Louis Ave, Chicago, IL 60625.

One of the first black actors to portray a doctor in prime time, Hal Frederick was Dr Cal Barrin on the short-lived series, "The Interns."

Louis Gossett, Jr lasted only six telecasts as cardiologist Dr MacArthur St. Clair, the central character of "The Lazarus Syndrome."

Early in his career, James Earl Jones portrayed Dr Jim Frazier on "The Guiding Light" and Dr Jerry Turner on "As the World Turns."

Jones played physicians on "The Guiding Light" and "As the World Turns." John Danelle portrayed a doctor on "All My Children" for many years. During the 1970s Palmer Deane was Dr Hank Iverson on "The Doctors." And one of the newer serialized dramas, "Loving," features Willie Carpenter as psychiatrist Ron Turner.

This is not to suggest, however, that soap operas have been as well integrated as could be expected. Until recently many ethnic groups were invisible in the genre. Moreover, given the fact that black women constitute as much as one quarter of the daytime audience, minority representation has been inadequate. Nonetheless, a sprinkling of black performers appears regularly in the soaps, and the medical profession has become a familiar role for such actors.

Still, it is prime time television that attracts most viewers. It is evening fare that most affects Americans with its influential mix of entertainment and information—forever projecting role models, forging attitudes, and shaping values and self-concepts. Warm and caring Marcus Welby, who gave viewers an indelible image of the concerned, even saintly physician, was but one in a long history of trustworthy pop culture doctors dating to earliest television, and even to feature films and network radio in the 1930s. At the same time, however, these medical heroes offered audiences a counterimage: that warm and caring doctors were not black—or Asian, Hispanic, Native American, Arab, or of any other non-Caucasian racial lineage.

For a black doctor to survive in prime time television—be he a comedic or dramatic character—would be nothing less than a cultural transformation demanding the abandonment of popular prejudices and the rejection of racial stereotypes. The last person to face the challenge directly was Lou Gossett six years ago. Bill Cosby has realized unprecedented success, but his Dr Cliff Huxtable practices medicine off camera. And the experience of Howard Rollins suggests that the television industry maintains its old restrictive opinion about black doctors and white audiences. It is likely that the next black doctor in his or her own series will not appear until the next decade—and possibly the next century.

A scene from the series "Marcus Welby, MD," starring Robert Young: the warm and caring doctor of prime time television has always been white—and almost always a man.

The status of Asian Americans in the health care delivery system in New York

SETSUKO MATSUNAGA NISHI, PHD, CHARLES PEI WANG, MSW

The number of Asian Americans* in New York has increased significantly since the 1965 immigration reforms, and this population's role in filling critical gaps in the state's health care delivery system is particularly noteworthy. Ironically, however, this role obscures the recognition and resolution of difficult problems. The distribution of Asian Americans in New York's medical care system delineates issues of concern, not only to this minority population, but with respect to the status of the system itself.

ASIAN PRESENCE IN NEW YORK

Asian Americans constitute the fastest-growing racial or ethnic group in New York, with a 167% increase in population (330,972 in 1980)[1] since the last decennial census.[2] Three quarters were foreign-born[1] and three quarters, or 245,759, lived in New York City, where they comprised 3½% of the city's total population.[3] Language and residential patterns, among other factors, have contributed to what community leaders believe to be a significant under-enumeration.

New York State, with 8.9% of all of the country's Asian and Pacific Islanders, ranks below only California, with 35.8% and Hawaii with 16.7%.[2] By far the largest group within the Asian and Pacific Islander population in New York are the Chinese, numbering 148,105 in 1980. Eighty-four percent live in New York City,[4] where the Chinese population now exceeds even that of San Francisco. As recently as 1960, the sex-ratio was 165 males per 100 females,[4] a vestige of the near-genocidal effects of the Chinese Exclusion Act of 1882 and subsequent restrictive policies.

Asian Indians, with 60,505 people, are the second largest Asian group in the state. The 34,157 Koreans and 33,956 Filipinos rank next. A little over two thirds of their respective populations live in New York City.[4] Virtually nonexistent in New York until the post-Sputnik "brain-drain" of Asian intellectuals, the concentration of Asian Indians, Koreans, and Filipinos in highly skilled professions, particularly in health care, reflects the 1965 Immigration Act's preference provisions to fill shortages in the United States' scientific and technical personnel.

Of the 24,524 people of Japanese descent in New York, only 56% or 13,730 live in New York City,[4] indicating greater residential dispersion than other Asian groups. According to the Japanese Chamber of Commerce in New York, which takes a biennial census, about two thirds of the 23,512 Japanese[4] in the New York metropolitan area are employees (and their families) of overseas Japanese corporations who are in the United States on rotating three to five-year assignments. The remaining third are Japanese Americans, many of whose families came from the West coast following their evacuation and incarceration during World War II.

The 6,644 Vietnamese counted in the 1980 census represent only the beginnings of the arrival of this refugee population in New York, and the separately reported Pacific Islander groups—1,566 Hawaiians, 773 Guamanians, and 296 Samoans[4]—exhibit yet another facet of the census category of the Asian and Pacific Islander population.

Asian Americans in New York are a diverse population. However, in the health care delivery system, their role can be distinguished from that of both the white majority and other minorities.

UNEVEN DISTRIBUTION IN HEALTH CARE

The 1980 census figures[1] show that of all employed Asian Americans in New York, 15.1% work in health services. This is a much larger proportion than the 8.9% of all employees in the state. In some Asian groups, the concentration in health services is remarkable: Filipinos, 37.4%; Asian Indians, 26.1%; and Koreans, 18.7%. In contrast, only 5.7% of the Chinese and 4.2% of the Japanese are so employed.

Asians found entry into those sectors of the American medical system when and where there have been the most severe personnel shortages. The Immigration and Naturalization Act of 1965 removed the barriers against immigration from Asian countries. The Act also allowed medical professionals to be exempt from the usual visa requirements of an employment offer and labor certification that there was insufficient manpower in the United States to perform the work and that the exemption would not adversely affect American workers. In the growing health industry, the personnel shortages were on the professional levels of physicians, nurses, and technicians. This deficit was especially acute in government service: in urban hospitals, in state psychiatric hospitals, and in the Veterans Administration.

Asian Americans' concentration in the professional and technical levels of the health care system was evident in their profile in specific health-related occupations (Table I). While Asian Americans constituted only 2.1% of the state's employed population, they made up 16.6% of New York's physicians, 7.9% of clinical laboratory technicians, 7.8% of dieticians, 7.3% of pharmacists, and 5.5% of regis-

---

From the Department of Sociology, Brooklyn College, and the Graduate Center of The City University of New York (Dr Nishi), and the Chinatown Planning Council (Mr Wang), New York.

Address correspondence to Dr Nishi, Professor, Department of Sociology, Brooklyn College, Brooklyn, NY 11210.

* The term "Asian Americans" refers to the racial/ethnic category termed "Asian and Pacific Islander" by the US Census Bureau.

TABLE I. Health Occupations of All and Asian American Employed Persons in New York by Sex in 1980[7]

		Asian Americans		
			% of Total	
Occupation	Total	No.	M	F
Employed persons 16 years and over	7,440,768	155,479	1.2	0.9
Health occupations	464,795	21,193	2.8	4.6
Managers, medicine and health	8,792	149	0.9	0.8
Health diagnosing occupations	66,242	8,295	8.4	4.1
Physicians	47,273	7,856	11.2	5.4
Dentists	13,027	333	1.7	0.8
Health diagnosing occupations, not elsewhere classified	5,942	105	1.5	0.3
Health assessment and treating occupations	155,073	8,063	0.7	4.5
Registered nurses	118,708	6,521	0.3	5.2
Pharmacists	10,869	794	5.0	2.3
Dieticians	5,944	460	0.4	7.4
Therapists	17,608	234	0.6	0.7
Physicians assistants	1,944	54	1.6	1.1
Medical science teachers	738	34	4.6	—
Health technologists and technicians	71,300	2,627	1.3	2.5
Clinical laboratory technologists and technicians	18,957	1,498	2.9	5.0
Dental hygienists	4,293	2	—	—
Health record technologists and technicians	1,048	61	1.0	4.9
Radiologic technicians	6,710	141	1.5	0.6
Licensed practical nurses	29,582	644	0.1	2.1
Health technologists and technicians, not elsewhere classified	10,710	281	1.7	0.9
Health service occupations	162,650	2,025	0.3	0.9
Dental assistants	12,289	92	0.2	0.5
Health aides, except nursing	28,911	238	0.3	0.5
Nursing aides, orderlies, and attendants	121,450	1,695	0.3	1.1

tered nurses. This skewed distribution was reflected further in the fact that three quarters of the Asian Americans in health occupations were accounted for in just three categories: physicians, nurses, and clinical laboratory technologists and technicians (Table II). The corresponding distribution of all medical workers in these three occupations combined equals 39.8%.

Only 8% of Asian American health workers were nursing aides, orderlies, or attendants. According to Kim's[5] study of Korean-American medical professionals in New York and testimony presented at a conference convened by the New York State Advisory Committee to the United States Commission on Civil Rights,[6] many of these were foreign-trained professionals who had not passed their licensure examinations. Indeed, census data[7] indicate that 20.9% of the Asians, compared to 3.9% of all those employed in this occupational category, had four or more years of college.

A second feature of the distinctive distribution of Asian Americans in health services is their concentration in inner cities. In New York, 53.8% of Asian Americans in the health diagnosing occupations (physicians, dentists, and others) are in the central cities, as opposed to 47.2% of all those in this field in the state. Nurses and technicians are even more concentrated in urban areas: 78.7% of Asian Americans as opposed to 44.7% of all employees in the health assessment and treating occupations (mainly nurses), and 72.8% of Asian Americans, compared with 44.5% of all health technologists and technicians.[1]

Another notable characteristic is that Asian Americans

TABLE II. Percent Distribution in Health Occupations of All and Asian American Employed Persons in New York in 1980[7]

Occupation	% Total* (No. = 464,794)	% Asian Americans† (No. = 21,193)
Health occupations	100.0	100.0
Managers, medicine and health	1.9	0.7
Health diagnosing occupations	14.3	39.1
Physicians	10.2	37.1
Dentists	2.8	1.6
Health diagnosing occupations, not elsewhere classified	1.3	0.5
Health assessment and treating occupations	33.4	38.0
Registered nurses	25.5	30.8
Pharmacists	2.3	3.7
Dieticians	1.3	2.2
Therapists	3.8	1.1
Physicians assistants	0.4	0.3
Medical science teachers	0.2	0.2
Health technologists and technicians	15.3	12.4
Clinical laboratory technologists and technicians	4.1	7.1
Dental hygienists	0.9	—
Health record technologists and technicians	0.2	0.3
Radiologic technicians	1.4	0.7
Licensed practical nurses	6.4	3.0
Health technologists and technicians, not elsewhere classified	2.3	1.3
Health services occupations	35.0	9.5
Dental assistants	2.6	0.4
Health aides, except nursing	6.2	1.1
Nursing aides, orderlies and attendants	26.1	8.0

in the diagnosing occupations are found disproportionately in government employment: 22.6% of men and 33.8% of women, compared with 10.5% of white men and 21% of white women. Proportionately far fewer Asian men (20.9%) and women (12%) are in practice for themselves, compared with white men (45.8%), and white women (23.2%). Nearly a quarter of the government employees in diagnosing occupations in New York are Asian Americans (16.5% of the federal, 29.7% of the state, and 21.4% of the local government employees).[7]

Thus, Asian American physicians are concentrated in government employment. It was the public sector that faced the most extreme shortages, and foreign medical graduates were pulled into this vacuum.

Asian health professionals comprise 30% of the total state employees in health diagnosing occupations.[8] About three quarters or 1,514, of the 2,029 total Asian American state employees in 1982 were professionals, according to Civil Service records that did not specify occupation.[8] However, 71.3% are in just three health-related agencies: 42.6% are in the Office of Mental Health, 21.5% are in the Office of Mental Retardation and Developmental Disabilities, and 7.2% are in the Department of Health.

The most recent figures available from the New York City Health and Hospitals Corporation,[9] indicate that as of June 1983, 16.3% or 2,096, of the corporation's professionals are Asian Americans. Of all the Asian Americans employed by the corporation, 77.2% are professionals and 8.7% are technicians. Yet compared with other full-time professionals of the corporation, Asian American men are underrepresented in the higher salary brackets compared with white men, and Asian American women are proportionately fewer in the upper income categories than all other female groups: 29.1% of Asian American men earn $25,000 or more, compared with 40.6% of white men, 23.7% of black men, and 22.6% of Hispanic men. For women in that earning range the percentages are as follows: 7% of Asian Americans, compared with 21.2% of whites, 15.7% of blacks, and 10.1% of Hispanics.

HOSPITAL-BASED SERVICE

The fourth distinctive aspect of Asian Americans in the New York medical system is their relatively large continued presence in hospital-based care, despite the sharp curtailment of foreign medical graduates (FMGs) as residents in 1976.

Unfortunately for this analysis, the published physician profile data collected by the American Medical Association[10] on FMGs have not been broken down by country of origin. For the United States, however, data are available on contributing countries. Asian countries have been the source of 38.2% or 37,717, of the total 97,726 FMGs in the United States in 1980. The countries contributing the largest numbers were India, 15,032; the Philippines, 12,613; and Korea, 4,280. Together these countries contributed 32.7% of the total FMGs from all countries excluding Canada.

In New York State, an astounding 39.2% or 19,115, of the total 48,713, nonfederal physicians were medical graduates of countries outside the United States and Canada.

In the New York metropolitan area, the 12,771 FMGs constitute 43.3% of the total 29,490 physicians.[11] This is more than double the 20.9%[10] proportion of FMGs to physicians in the United States.

Inner-city and state psychiatric hospitals were major points of entry for FMGs into the American medical system. The Exchange-Visitor Program, developed to provide advanced training opportunities, was a significant means of filling hospital staff shortages. More than 16% of FMGs in the United States are full-time physician staff of hospitals, compared with 9.1% of all physicians. In 1970, nearly half (47.6%) of FMGs were engaged in hospital-based practice, including 29.1% as residents. By 1980, the hospital-based proportion had declined to 27.8%—with only 11.7% as residents. However, the proportion of FMGs who are full-time physician staff members has changed very little, despite a dip in 1976.[10]

In New York State, even more FMGs are hospital based; 36.9% are in hospitals, compared with 24.8% of non-FMG physicians in 1979.[11] FMGs make up 58.3% of the full-time physician staff of New York's hospitals, and despite the restrictions imposed at the end of 1976, 43.4% of hospital residents are FMGs.

The decline in FMGs entering residency training programs in the United States is largely a consequence of the 1976 Health Professions Educational Assistance Act (PL94-484), which instituted special requirements, such as the Visa Qualifying Examination, and rescinded the visa exemptions formerly granted physicians. In some sectors of the health care delivery system, this change precipitated shortages of crisis proportions, according to Goodman and Wunderman.[12]

As noted earlier, New York State and New York City had by far the largest number of FMGs and Exchange Visitor Foreign Medical Graduates (EVFMGs) as medical residents in the country. The 3,830 FMGs who were medical residents in New York represented 29.3% of all FMG residents in the United States in 1979, and the 555 EVFMGs were 28.3% of all such residents. New York City alone had 3,056 FMG residents.

These 1979 figures reflect a decline of 25.4% in FMGs and 74.4% in EVFMGs in the state since 1975. New York City figures showed a still greater reduction. Even then, New York City had more FMGs than US medical graduates. Goodman and Wunderman[12] observed that these reductions have caused "a substantial disruption of medical services," particularly in New York, where about three quarters of the requests for waivers of certain provisions of PL94-484 have originated. Waiver requests have been increasing annually, contrary to the expectation that US medical graduates would be in sufficient supply. Clearly, the continued dependence on FMGs is in those locations and types of institutions considered less desirable by US medical graduates.

Psychiatric hospitals and Medicaid-financed programs have been especially dependent on FMGs. The study by Goodman and Wunderman[12] attributes the disproportionate presence of FMGs in state mental hospitals to the undesirability of these positions to US medical graduates and to permissive licensure policies that enable these institutions to employ residents with limited licenses or temporary work permits. Stevens, Goodman and Mick[13] have noted that "hospitals with the least university affiliations and the least opportunities for a research oriented curriculum act as residual portals of entry" for FMGs not born in the United States.

FMGs' variable participation in the health care delivery system is evident in their specialty profile.[10] There are relatively fewer FMGs in surgery (13.8%) than the proportion of all physicians in surgery in the United States (18.1%). There are also fewer in general practice—9.4%—than the 12.8% of all physicians. There are relatively more, though, in internal medicine—17.6%, compared with 15.3% of all physicians, anesthesiology—6% of FMGs and 3.4% of all physicians, and psychiatry—7.7% of FMGs and 5.9% of all physicians.[10]

Though the specialty profiles do not indicate major differences for FMGs as compared with all physicians, their relative underparticipation in surgery and overparticipation in psychiatry contributes to income differentials. For example, in 1979, Middle Atlantic-area office-based non federal surgeons had an average net income of $94,400, compared with $61,200 for psychiatrists.[14]

THE PROFESSIONAL UNDERCLASS

Examination of demographic data has delineated a distinctive profile of Asian American participation in New York's health care delivery system. Filipinos, Asian Indians, and Koreans are employed primarily in the health services industry. They are concentrated on the professional and technical level as physicians, nurses, and technicians. They are located disproportionately in cities, in government employment in city hospitals and state mental hospitals, in hospital-based service, and somewhat more in specialties such as psychiatry and anesthesiology and somewhat less in higher-income specialties, compared with all physicians.

With the increasing availability of American-trained health professionals, new entry for Asian Americans into the US health care system is sharply curtailed. The more stringent licensure requirements for foreign-trained health professionals, though ostensibly to assure the maintenance of professional standards, are directly related to the supply of United States-trained personnel. As in the past, however, requirements have been waived to accommodate the personnel needs of less-attractive sectors of the medical system. Thus, New York City hospitals are still paying large commissions for recruiting nurses from the Philippines and applying for waivers to fill their residency programs with FMGs. There is some indication of a lessening concentration of Asian Americans in the least-desired sectors of the health care system, but the gap continues.

It is unclear whether Asian American health professionals can progress with equitable opportunity based on merit. It is possible that many Asian Americans and other Third World-trained health professionals, unable to overcome the disadvantageous circumstances of their entry, will constitute a more or less stable underclass in New York's health care delivery system. The implications of such stratification are regressive for the system and are particularly serious for the medically underserved populations of the state.

REFERENCES

1. General social and economic characteristics, pt. 34, in *1980 Census of Population*, vol 1: *Characteristics of the Population*. Washington, DC, US Bureau of the Census (PC80-1-C34), 1980.
2. Race of the population by states, Suppl report in *1980 Census of Population*, vol 1: *Characteristics of the Population*. Washington, DC, US Bureau of the Census (PC80-S1-30), 1980.
3. Advance estimate of social, economic and housing characteristics, pt. 34, Suppl report in *1980 Census of Population and Housing*. Washington, DC, US Bureau of the Census (PHC80-S2-34), 1980.
4. General population characteristics, pt 34, in *1980 Census of Population*, vol 1: *Characteristics of the Population*. Washington, DC, US Bureau of the Census (PC80-1-B34) 1980.
5. Kim I: *New Urban Immigrants: The Korean Community in New York*. Princeton, Princeton University Press, 1981.
6. *Asian Americans: An Agenda for Action*. Washington, DC, New York State Advisory Committee to the US Commission on Civil Rights, 1981.
7. Detailed characteristics of the population, pt. 34, in *1980 Census of Population*, vol 1: *Characteristics of the Population*. Washington, DC, US Bureau of the Census (PC80-1-D34), 1980.
8. *Ethnic Survey of New York State Agency Work Force, 1982*. Albany, New York State Department of Civil Service, 1983.
9. *Equal Employment Opportunity Commission State and Local Government Information* (form EEO-4). New York, New York City Health and Hospitals Corporation, 1983.
10. *Physician Characteristics and Distribution in the U.S., 1981*. Chicago, American Medical Association, 1982.
11. Jensen LE, Wunderman LE, Goodman LJ: *Characteristics of Physicians: New York, December 31, 1979*, US Dept of Health and Human Services Publication No. (HRA) 82-133, 1982.
12. Goodman LJ, Wunderman LE: Foreign medical graduates and graduate medical education. *JAMA* 1981; 246:854-858.
13. Stevens R, Goodman LW, Mick SS: *The Alien Doctors: Foreign Medical Graduates in American Hospitals*. New York, John Wiley & Sons Inc, 1978.
14. *Statistical Abstract of the United States 1981*. Washington, DC, US Bureau of the Census, 1980.

One of the most important publications to appear in recent years on health care and American ethnic minority groups is *The Western Journal of Medicine* of December, 1983 (Volume 139, Number 6)—a special issue entitled "Cross-Cultural Medicine."

—*Editor*

Gastrointestinal malignancies in black Americans

WILLIAM MCBRIDE, MD, GARY C. RICHTER, MD

In 1982, the last year for which figures are obtainable approximately 81,000 black Americans learned that they had cancer: 27% had cancers of the oral cavity or digestive organs. Studies of the incidence of cancer during the past several decades show that the rates for black Americans have been higher than for white Americans.[1] In reference to gastrointestinal malignancies, death rates have increased for cancers of the oropharynx, esophagus, pancreas, and colorectal region during a 25-year period ending in 1978. Although death rates have declined for gastric carcinoma for both black and white Americans, black Americans still have higher incidence rates for gastric carcinoma. This article will discuss the major gastrointestinal malignancies that occur in black Americans and, when possible, make specific references to environmental or socioeconomic factors as they relate to cancer incidence rates, survival trends, and cancer prevention.

ESOPHAGEAL CARCINOMA

Approximately 9,000 cases of esophageal carcinoma are diagnosed each year in the United States. The incidence rate in white men is 6 cases per 100,000; for black men it is 20 cases per 100,000.[2] In the Washington, DC vicinity, the incidence rate in black men is 28.6 cases per 100,000—such unexplained regional variations are common with esophageal carcinoma.[3] In general terms, cancer of the esophagus is a disease of urbanites,[3] although in some regions urbanization is not a major factor.[4] There appear to be no available data in relation to incidence and etiologic factors for esophageal carcinoma in rural black communities. There is evidence that, in certain regions, esophageal carcinoma may develop at an earlier age in blacks as compared to whites.[5]

There are several major risk factors associated with the development of esophageal carcinoma. Alcohol consumption is considered a major risk factor for development of this disease. There is evidence that a gradient exists, with abuse of straight liquor presenting the highest risk, wine and mixed drinks having an intermediate risk, and beer having the least risk for the development of carcinoma in black men.[3] Cigarette smoking also appears to be a major risk factor in the development of carcinoma in black men.[3] Cigarette smoking also appears to be a major risk factor in the development of esophageal carcinoma and may work concomitantly with alcohol to increase the risk of developing this disease. Nutritional status has been shown to be a risk factor for development of this cancer in urban populations. In a recent study, the risk of esophageal carcinoma developing in black men shows an inverse correlation with several indicators of nutritional status: consumption of dairy products, fruits, and vegetables; number of meals eaten per day; and relative weights.[3] Specific dietary deficiencies have not been related to the development of this disease. In the general population, esophageal carcinoma occurs more frequently in patients with a history of lye stricture, Barrett's esophagus, achalasia, and tylosis, and in women with the Plummer-Vinson syndrome. Whether these factors have any relationship to black patients who develop esophageal carcinoma is not known.

Esophageal carcinoma is difficult to detect in its early stages. Approximately 50% to 75% of the patients already have metastatic disease when the disorder is diagnosed. Some controversy exists concerning the best therapy for esophageal cancer, but most approaches involve surgery for lesions in the lower third of the esophagus and radiation therapy for lesions in the middle and upper third of the esophagus. Five-year survival rates after surgical resection and radiation therapy have been estimated at approximately 5% and 6%, respectively.[6] A recent study of treatment of esophageal cancer in inner-city blacks reported a 14% survival rate with surgery and a 13% survival rate in patients who receive radiation therapy. The cost of surgical treatment is estimated at $33,000, and for radiation therapy, $23,000.[7]

Worldwide, the best results of treatment have been reported from China, where there are regions in which the frequency of esophageal carcinoma is 139 cases per 100,000 people. Early diagnosis has been achieved in these regions with blind abrasive cytology, which can be used to detect esophageal carcinoma in asymptomatic patients and pre-malignant dysplasia. In asymptomatic patients, resectability rates are approximately 100%, and five-year survival rates approach 90%. Thus, the diagnosis of esophageal carcinoma in an asymptomatic state can result in higher cure rates.[6]

The frequency of esophageal carcinoma is increasing among black Americans. Prevention can be enhanced by promoting public health programs against alcohol and smoking and by improving the nutritional status of black Americans. Massive screening of high risk individuals (such as those who abuse alcohol) with blind abrasive cytology or periodic esophagoscopy would be superior to screening with barium swallow radiologic methods. However, such programs may not be cost effective and will be difficult to implement due to the nature of the patient population.

GASTRIC CARCINOMA

There has been a steady decline in the death rates resulting from gastric carcinoma among both black men and women. However, black Americans still have the highest incidence rate, and the disease occurs in both urban and rural settings. In the United States, there is an inverse correlation between the development of gastric carcinoma

---

From the Department of Family Medicine, Morehouse School of Medicine, Atlanta.

Address correspondence to Dr McBride, Consultative Gastroenterology, 730 Peachtree St, NE, Suite 1045, Atlanta, GA 30308.

and socioeconomic status. In an inner-city population in Detroit, incidence rates for black men increased 2.3% during the 1973–1977 period.[8] Surveys from the Dallas-Fort Worth area indicated that twice as many black men and women were afflicted with gastric carcinoma per 100,000 people as compared to their white counterparts.[9] In southern Louisiana, there is a high rate of gastric carcinoma among blacks, both in farming communities and in the areas populated near food processing and machinery manufacturing plants.[10]

The specific cause of gastric carcinoma is not known, thus making prospects for prevention and improving survival unfavorable. To diminish the death rate from gastric carcinoma and to improve survival, improved endoscopic surveillance of high risk populations—such as those with gastric polyps, a history of subtotal gastrectomy, and achlorhydria—may prove useful. Endoscopic recognition of "early gastric carcinoma," in which malignancy is confined to the mucosa, will also be important in reducing the incidence and death rates of gastric carcinoma.[11] Early gastric carcinoma may remain stable for many months prior to reaching a more invasive stage, and thus is more amenable to cure. The five-year survival rate for early gastric carcinoma is approximately 90%, compared to five-year survival rates of 12% in more advanced cancers.

Recognition and surveillance of pre-cancerous conditions or early gastric carcinoma will be especially difficult in low-income black communities. Endoscopy can be expensive, and access to subspecialists such as gastroenterologists can be limited at city and county hospitals. Also, surveys have shown that urban blacks are less knowledgeable about cancer warning signals and less likely to see a doctor when cancer symptoms occur. These surveys also indicate that blacks are less likely to undergo common specific cancer tests such as Papanicolau's smear, which are considerably less costly and complicated than a surveillance endoscopy in a black patient at high risk for gastric carcinoma.[1]

CANCER OF THE PANCREAS

Mortality rates from pancreatic carcinoma in the United States increased between 1920 and 1970 from 2.9 to 9.0 per 100,000 people. During this period, blacks showed the greatest percentage increase in patients under the age of 50 years.[12] In a cohort analysis of pancreatic carcinoma, the mortality rates for blacks were higher than for their white counterparts in patients born between 1900 and 1904 and followed through 1969.[13] The investigators suggested that smoking, occupation, and dietary factors are important environmental stimuli for the development of this disease.

The cause of cancer of the pancreas remains an enigma. Smoking, drinking, diabetes mellitus, chronic pancreatitis, and coffee consumption have all been implicated, but the data are inconclusive or contradictory. Primary prevention of pancreatic carcinoma in blacks will remain elusive until etiological mechanisms are identified.

COLORECTAL CANCER

There are approximately 123,000 cases of colorectal cancer diagnosed each year, and there has been an increase in death rates for blacks over the past 25 years. Although it is well known that early detection of colorectal carcinoma favorably improves outcome, there has been no drastic increase in the survival rates of patients with colorectal carcinoma over the past three decades. As of 1973, the five-year survival rate for whites was 48%, compared to 35% for blacks.[1]

It is thought that many, if not most, colorectal carcinomas develop from benign adenomatous polyps. The minimum time period for development of a cancer from a polyp appears to be about five years, but probably averages 10 to 15 years.[14] There is an extremely high incidence of colon cancer in patients with familial polyposis and in patients with longstanding (greater than seven years) universal ulcerative colitis. Patients with Crohn's colitis have a slightly increased frequency in developing colon cancer. Other high risk groups include patients over the age of 40 years, patients with a personal or family history of colorectal polyps or cancer, and women with a history of genital or breast cancer.

Detection of colorectal cancer in asymptomatic individuals usually results in finding lesions that are localized, resulting in better survival rates. There are three ways to detect colorectal cancer in asymptomatic individuals: digital examination, stool occult blood testing, and sigmoidoscopy.

Only 12% of colorectal carcinomas are palpable on rectal examination. However, annual digital examinations appear to be useful in lowering death rates in large patient populations. In a study of a health maintenance organizations, annual digital examinations of 5,000 patients over an 11-year period resulted in significantly lower death rates for colorectal cancer compared to matched controls.[15] Unfortunately, in a review of practices at a large urban hospital treating mainly black patients, only 13% of the patients at risk for developing colorectal cancer, received rectal examinations for screening purposes.[16] There is a clear need to perform more rectal examinations in urban medical centers as part of routine physical examinations, particularly in middle-aged patients.

Stool hemoccult testing can aid in detecting early lesions, but there are no data indicating that fecal occult blood testing causes a decline in mortality from colorectal cancer.[17] Screening of high risk patients, using stool occult blood testing,was recently done at Cook County Hospital.[16] The study involved approximately 10,000 patients, of which about 95% were black. The patients were given stool hemoccult cards; 37% of the patients returned the cards. Four percent of the patients who returned cards had at least one positive stool hemoccult blood test. Overall, 0.283% of the patients returning their cards had colorectal cancer; the majority of the lesions were localized. Among 117 patients with positive occult blood tests who underwent further testing, 15.4% had pre-malignant polyps; 8.6% had carcinomas; and 15.4% of this group had no lesions and thus were false-positives. Thus, stool occult blood testing appears to be a useful and inexpensive screening procedure for large numbers of patients, but further studies will be necessary to show if such screening decreases mortality from colorectal cancer.

The efficacy of sigmoidoscopy in detecting pre-malignant polyps and early colorectal cancers—resulting in decreased mortality in patients who undergo periodic screening—is well documented. Unfortunately, sigmoidoscopy use is low among practitioners as a screening procedure. In a recent survey by the American Cancer Society, only 17% of low-income blacks had ever heard of a sigmoidoscopy examination.[1] In the Cook County study, less than 1% of the patients at high risk for developing colorectal cancer had undergone sigmoidoscopy. Flexible sigmoidoscopes, with lengths varying from 30 cm to 60 cm, have replaced the rigid sigmoidoscope in many practice situations because of greater patient comfort and greater ability to detect neoplastic lesions.[18] Training general practitioners and internists in the use of this instrument has begun, and preliminary studies reporting the training of non-physicians with the use of the flexible sigmoidoscope have produced favorable results.[19]

With the increase in frequency of colorectal cancer in black Americans, widespread screening procedures for high-risk patients will be necessary to maximize earlier detection of cancer and of pre-malignant polyps. The American Cancer Society has presented the following guidelines for screening asymptomatic individuals:

- annual digital examinations for patients 40 years and over
- annual hemoccult testing in patients 50 years and over
- sigmoidscopy in patients 50 years and over for two consecutive years, and, if both are negative, every three to five years subsequently

Because of monetary considerations, compliance problems, and inferior preventive medical services at many public hospitals and outpatient care centers, widespread application of the American Cancer Society guidelines among public institutions serving low-income blacks will be difficult.

CANCER AND LIVER CIRRHOSIS

Primary cancer of the liver is uncommon in black patients who have not developed cirrhosis. The frequency of liver cancer in black Americans has decreased in the past 25 years, in the period ending in 1978.[1] Earlier surveys support a low incidence of primary hepatic tumors in black Americans compared to some of their African counterparts, in whom hepatic cancer occurs more frequently and has been linked to hepatitis-B infection. The incidence rates for hepatocellular carcinoma in various African countries have ranged from 6.2 to 103 cases per 100,000 people annually.[20]

Black patients with alcoholic cirrhosis are more prone to develop cancers at an earlier age and have three times the frequency of esophageal and hepatic cancers. These patients tend to have less gastric and prostatic cancer when compared to black patients without cirrhosis.[21] Multiple primary sites of cancer occur more frequently among black men with cirrhosis. In a study of 237 black male patients with cirrhosis and cancer, matched to 237 black patients with cancer but not cirrhosis, there was a significant increase in the frequency of mouth and pharyngeal carcinoma and gastrointestinal tumors and significantly fewer lung and genitourinary tumors. Furthermore, the risk of developing multiple primary sites was 3.2 times greater for patients with cirrhosis when compared to the patients without cirrhosis.[22] Among the most common combinations were mouth and pharynx, and mouth, pharynx, and esophagus. The higher cancer rates in patients with cirrhosis may be related to patterns of heavy alcohol consumption and smoking, both of which were common in the cirrhotic group.

CONCLUSIONS

Gastrointestinal malignancies are common among black people. Incidence and mortality rates are less favorable for blacks in reference to cancers of the oropharynx, esophagus, stomach, and pancreas. Black patients with cirrhosis appear to be more prone to liver cancer and multiple primary neoplasms. It is likely that racial differences in food preferences and alcohol ingestion, and differences in detrimental environmental exposure contribute to the high risk of developing carcinoma in black Americans. There is a definite need for further study in blacks alone and in comparison to whites and other ethnic groups to identify the specific risk factors and mechanisms of these gastrointestinal neoplasms.

Specific socioeconomic inequalities such as the higher unemployment rate in black Americans, the fact that the average black family earns two thirds that of the white family, and the fact that blacks represent only 0.5% of all college graduates[2] would tend to stifle progress in preventing gastrointestinal cancer or in early diagnosis of gastrointestinal neoplasms. These socioeconomic factors impair the ability of many black Americans to obtain quality health care and wholesome lifestyles, and may impair their ability to understand and comply with cancer screening and prevention programs.

REFERENCES

1. *Cancer Facts and Figures for Black Americans. 1983.* New York, American Cancer Society, Inc, 1983.

2. Smith EB: Cancer of the esophagus in blacks. *J Natl Med Assoc* 1985; 77:44-48.

3. Pottern LM, Morris LE, Blot WJ, Ziegler RG, Fraumeni Jr JF: Esophageal cancer among black men in Washington, D.C. I. Alcohol, tobacco and other risk factors. *JNCI* 1981; 67:777-783.

4. Rogers EL, Goldkind L, Goldkind SF: Increasing frequency of esophageal cancer among black male veterans. *Cancer* 1982; 49:610-617.

5. Attah EB, Hajdu SI: Benign and malignant tumors of the esophagus at autopsy. *J Thorac Cardiovasc Surg* 1968; 55:396-404.

6. Lee DJ: Cancer of the esophagus: radiaton therapy. In Buyless T (ed): *Current Therapy in Gastroenterology and Liver Disease.* 1984, pp 44-49.

7. Mandal AK, Shaman IN, Bier RK, Oparah SS: Outcome of thoracic esophageal carcinoma in blacks in the inner city. *Cancer* 1984; 54:924-928.

8. Swanson GM, Young JL: Trends in cancer incidence in metropolitan Detroit, 1937-1977: Leads for Prevention. *Prev Med* 1983; 12:403-420.

9. Frenkel EP, Clark B, Percy C: Cancer incidence rates in the Dallas-Fort Worth metropolitan area. *Tex Med* 1975; 71:71-78.

10. Fraumeni, JF: The face of cancer in the United States. *Hosp Pract* 1983; 18:81-96.

11. Blackstone MO: *Endoscope Interpretation.* New York, Raven Press, 1984, pp 137-158.

12. Kovi J, Heshmat MY: Incidence of cancer in negroes in Washington, D.C. and selected African cities. *Am J Epidemiol* 1972; 96:401-413.

13. Benarde MA, Weiss W: A cohort analysis of pancreatic cancer, 1939-1969 *Cancer* 1977; 39:1260-1263.

14. Day DW, Morson BC: The polyp problem, in Hunt RH, Way JD (eds): *Colonoscopy: Techniques Clinical Practice and Colon Atlas.* London, Chapman and Hall, 1981, pp 301-326.

15. Dales LG, Friedman GD, Collen MF: Evaluating periodic multiphasic health checkups: a controlled trial. *J Chronic Dis* 1979; 32:385-404.

16. Hoffman A, Young Q, Bright-Assaire P, et al: Early detection of bowel cancer at an urban public hospital: demonstration project. *CA* 1983; 33:344-358.

17. Winchester DP, Sylvester J, Maher ML: Risks and benefits of mass screening for colorectal neoplasia with the stool guiaiac test. *CA* 1983; 33:333-342.

18. Zucker, GM, Madura MJ, Chemiel JS, Olinger EJ: The advantages of the 30 cm flexible sigmoidscope. *Gastrointest Endosc* 1984; 30:59-64.

19. Shapiro M: Flexible sigmoidoscopy—the long and short of it. *Gastrointest Endosc* 1984; 30:114-115.

20. Wright R, Alberti KGMM, Karran S, Millward-Sadler GH: *Liver and Biliary Disease.* Philadelphia, Saunders, 1979, pp 866-925.

21. Keller AZ: Liver cirrhosis, tobacco, alcohol, and cancer among blacks. *J Natl Med Assoc* 1978; 70:575-580.

22. Keller AZ: Alcohol and tobacco use among cirrhotic and non-cirrhotic males with cancer, in *Cancer Among Black Populations.* New York, Alan R. Liss, 1981, pp 213-223.

Hypertension in blacks

Is it a different disease?

LEONARD G. MEGGS, MD

Despite the notion that hypertension is a major cause of morbidity and mortality in the black population, objective evidence linking hypertension in blacks with increased target organ damage has only recently appeared. Hypertension is now recognized as the principal risk factor in cerebrovascular disease, a complication to which blacks are particularly predisposed.[1] Recent evidence suggests that this sensitivity to vascular injury may extend to the renal circulation as well.[2,3] This review will focus on the potential causes and consequences of hypertensive vascular injury to the kidney, the impact of this complication as a public health concern, and the therapeutic dilemma associated with hypertension.

The relationship between the kidney and blood pressure regulation, depicted in the early studies of Goldblatt,[4] has long been appreciated. More recent observations suggest that the kidney plays a central role in the pathogenesis of primary or essential hypertension—the variant accounting for greater than 90% of the hypertension encountered clinically—and that blacks are predisposed to developing the renal lesion most closely identified with hypertension: nephrosclerosis. Although the basis for this predisposition remains uncertain, several hypotheses have emerged: an alteration in vascular tone, secondary to an imbalance or deficiency in vasoactive hormones or, perhaps, increased vascular sensitivity to a dietary constituent such as sodium[5,6]; increased sympathetic nervous system activity—a manifestation of autonomic dysfunction or a stress response[7]; and the occurrence of hypertension at an earlier age, thereby exposing the kidney for a more prolonged period. In line with this hypothesis is the well documented increased incidence of hypertension in the black adolescent population.[8] Whatever the case, it now appears certain that this predisposition to nephrosclerosis accounts in large part for the disproportionately high percentage of black patients with end-stage renal disease (ESRD).

The number of patients enrolled in the ESRD Program, as well as the current and projected expenditures for the program, have exceeded original estimates.[2] An unexpected finding was the extraordinarily high relative risk for developing ESRD in black patients with hypertension. The demographic data of patients entering ESRD programs indicate that hypertension is the principal cause of renal failure in black patients, approaching 50% in some instances. Beyond the social and economic implications of this finding is the realization by physicians and allied health professionals that hypertension is a chronic illness for which effective therapy is available which can reduce the associated morbidity and mortality. The salutary effects of therapy are greatest in patients with the highest blood pressures.

If treatment of hypertension is so effective, why does this disorder remain a significant cause of ESRD? Can it be assumed that for any given level of blood pressure elevation the consequences will be more severe in the black hypertensive patient? The data imply that the answer to the first question is related to the propensity of untreated or poorly controlled hypertension in blacks, leading to nephrosclerosis and ESRD. The answer to the second question is of importance, particularly in light of the recent controversy as to whether patients with "mild hypertension" (diastolic blood pressure <100 mmHg) should be treated.[9] Apparent differences between blacks and whites in the risk of developing target organ damage raises speculation as to whether a straightforward risk-benefit analysis is appropriate. Nevertheless, there are those who favor withholding therapy for mild hypertension based on a number of well publicized clinical trials that have failed to document a significant impact of pharmacologic therapy.[10,11] One must exercise caution in extrapolating the results of these trials, as the number of black patients enrolled in the more widely cited trials (Multiple Risk Factor Intervention Trial, Australian Therapeutic Trial in Mild Hypertension, United States Public Health Service trial) has been conspicuously small. Conclusions based on the results of these trials may not be applicable to the black hypertensive population. Recent data from the Hypertension Detection Follow-Up program, in which blacks comprised 40% or more of the patient population, would appear to support this contention. In the mild hypertensive group as a whole there was a significant decline in mortality, with almost 80% of the decreased mortality due to improved survival among black patients with hypertension.[12] These data suggest that untreated mild hypertension may indeed have a more ominous prognosis in black patients.

The preceding discussion has been concerned with the propensity of black hypertensive patients to develop ESRD as a result of nephrosclerosis. Relman[13] advanced the hypothesis that black patients with primary renal disease may be more inclined to develop secondary hypertension. Data indicating that black patients with such diverse pathologic entities as interstitial renal disease, chronic glomerulonephritis, and diabetic glomerulopathy are more likely to progress to ESRD than their white counterparts is consistent with this hypothesis.[3] Several lines of evidence suggest that hypertension hastens the course to ESRD once functional renal mass is reduced by a disease process.[14] Traditionally, hypertension associated with a reduction in functional renal mass was thought to be a volume-dependent state that was responsive to diuretic

---

Address correspondence to Dr Meggs, Assistant Professor, Department of Medicine, Division of Nephrology, Renal Center, Westchester County Medical Center, Valhalla, NY 10595.

therapy or the removal of excess salt and water by ultrafiltration. However, neurogenic and hormonal mechanisms may also be operating, and, under some circumstances, they may be the primary underlying factors.[15] Nonetheless, hypertension is a prime factor in the high incidence of cardiovascular disease and its attendant mortality in the patient with ESRD.

Hypertension remains a major public health concern because of its prevalence and potential to shorten and impair the quality of life. Because of its indolent nature, hypertension may go undetected for many years. Recent evidence cited in this review indicates that blacks are predisposed to severe target organ damage at the level of the kidney. Intensified efforts to identify hypertensive patients that are not being treated, coupled with increased awareness that race may be an independent risk factor, are only part of the multifaceted approach required to address this problem. The past decade has witnessed enormous progress in the understanding and treatment of this disease. New classes of pharmacologic agents such as converting enzyme inhibitors, potent vasodilators, and, perhaps in the not too distant future, the application of newly characterized cardiac atriopeptins,[16] have made the goal of effective blood pressure control obtainable in virtually all hypertensive patients. The consequences of untreated or poorly controlled hypertension are indeed a preventable tragedy.

REFERENCES

1. Ostfeld AM, Shekelle RB, Klawans H, Tufo HM: Epidemiology of stroke in an elderly welfare population. *Am J Public Health* 1974; 64:450.
2. Easterling RE: Racial factors in the incidence and causation of end-stage renal disease (ESRD). *Trans Am Soc Artif Intern Organs* 1977; 23:28–32.
3. Rostand SG, Kirk KA, Rutsky EA, Pate BA: Racial differences in the incidence of treatment for end-stage renal disease. *N Engl J Med* 1982; 306:1276–1279.
4. Goldblatt H, Lynch J, Hanzal RF, Sumerville WW: Studies on experimental hypertension. I. The production of persistent elevation of systolic blood pressure by means of renal ischemia. *J Exp Med* 1934; 59:347–379.
5. Warren SE, O'Connor DT: Does a renal vasodilator system mediate racial differences in essential hypertension? *Am J Med* 1980; 69:425–429.
6. Williams RR, Hopkins PN: Salt, hypertension, and genetic-environmental interactions. *Prog Clin Biol Res* 1979; 32:183–194.
7. James SA, Kleinbaum DG: Socioecologic stress and hypertension related mortality rates in North Carolina. *Am J Public Health.* 1976; 66:354–358.
8. Kilcoyne MM: Adolescent hypertension. II. Characteristics and response to treatment. *Circulation* 1974; 50:1014–1019.
9. Fries ED: Should mild hypertension be treated? *N Engl J Med* 1982; 307:306–309.
10. Helgeland A: Treatment of mild hypertension: a five year controlled drug trial. The Oslo study. *Am J Med* 1980; 69:725–732.
11. Multiple Risk Factor Intervention Trial Research Group: Multiple Risk Factor Intervention Trial: risk factor changes and mortality results. *JAMA* 1982; 248:1465–1477.
12. Hypertension Detection and Follow-Up Program Cooperative Group: Five-year findings of the Hypertension Detection and Follow-up Program. I. Reduction in mortality of persons with high blood pressure, including mild hypertension. *JAMA* 1979; 242:2562–2571.
13. Relman AS: Race and end-stage renal disease. *N Engl J Med* 1982; 306:1290–1291.
14. Mogensen CE: Renal function changes in diabetes. *Diabetes* 1976; 25:872–879.
15. Lilley JJ, Golden J, Stone RA: Adrenergic regulation of blood pressure in chronic renal failure. *J Clin Invest* 1976; 57:1190–1200.
16. Currie MG, Geller DM, Cole BR, et al: Purification and sequence analysis of bioactive atrial peptides (atriopeptins). *Science* 1984; 223:67–69.

HEALTH CARE DELIVERY AND THE BLACK PHYSICIAN

The National Medical Association (NMA) as an organization has always supported the progressive idea of excellent, effective, accessible health care for all people. In 1946, the NMA strongly supported Senate Bill 1606, known as the Wagner, Murray, Dingell Bill, which was a form of national health insurance. That bill was opposed by the American Medical Association (AMA). In 1964, the NMA testified in favor of House Resolution 3920, the King-Anderson Bill, which passed and is now known as Medicare. The AMA originally opposed that bill also. The irony of this is that after supporting the Democratic Administration in its passage of HR 3920, members of our organization were not asked to serve on advisory committees for its implementation. Because of the close proximity of its members to problems of delivery of health care to the poor and underprivileged, the NMA has expertise that should not have been excluded.

Although opposing positions may have been taken on various issues by the NMA and the AMA in the past, the two organizations have recently established a formal mechanism for dialogue . . . .

In recent years, legislation at the local, state, and federal levels has forced changes in health care delivery and affected the ability of many black physicians to treat their patients. Most of these changes have been in relationship to cost containment. Black physicians, out of necessity, have traditionally practiced cost containment.

Marked reduction in federal, state, and local budgets has decreased health care services to the socioeconomically deprived. Freedom of choice of medical care has been eliminated or markedly curtailed in a number of locales. Waivers of federal guidelines have facilitated procedures in many instances which have resulted in the elimination of freedom of choice for the federally-funded patient. Because of contractual agreements with local and state governments, patients are being forced into health maintenance organizations, independent practice associations, and overcrowded local governmental institutions. As Diagnosis Related Groups become the law of the land, black physicians will face additional problems because they usually encounter patients with more severe illnesses and greater dependence on some form of medical assistance.

PHILLIP M. SMITH, MD
President
National Medical Association
1012 Tenth St, NW
Washington, DC 20001

Sarcoidosis: focus on the black patient

RONALD P. DANIELE, MD

Sarcoidosis is a multisystemic granulomatous disorder characterized by epithelioid cell granulomas on histologic analysis. Its estimated incidence is about 1/10,000 persons per year, making for about 22,500 cases annually in the United States.[1] Sarcoidosis is many times more frequent in blacks and was thought to be prevalent in southeastern states.[2] The notion that sarcoidosis is more prevalent in southern blacks has been held for at least three decades.[2] However, epidemiologic studies have led investigators to question this view. For example, the greater incidence of sarcoidosis among individuals in the south, as suggested by the original Veterans Administration studies, is not confirmed by case-matched comparisons.[3] In the earlier study, population density statistics were not taken into account, ignoring the fact that the number of blacks in the south at that time outweighed that in the north. Thus, when previous studies have been corrected for the black-white population densities, the incidence of sarcoidosis in the south and southeast is similar to the frequency in other parts of the country.

Another erroneous perception is that sarcoidosis occurs infrequently, if at all, in black Africans. This belief has been challenged by more recent and accurate population surveys.[4]

Studies done at the Mount Sinai Hospital Sarcoidosis Clinic reveal further inconsistencies in the epidemiologic data.[5] The clinic population consisted of 57% black patients and 31% white patients. However, when a matched private office population was analyzed, 81% of the sample was white and 16% was black. Moreover, the same study showed that more women attended the clinic than the private office—casting questions on the overall sexual predilection in sarcoidosis. Although some surveys indicate that sarcoidosis is more common among women,[6] recent evidence suggests a female predominance only among black patients.[3]

Further epidemiologic studies, involving mass roentgenographic screening and, in one case, a search for the disease at autopsy, also provide persuasive evidence that patients in whom sarcoidosis is diagnosed are only the tip of the iceberg. Approximately 90% of patients with sarcoidosis are either asymptomatic or have such trivial symptoms that they do not bother to consult a doctor.[1,7]

These studies emphasize the need for more careful and controlled epidemiologic research. Taken together, however, experience throughout the country indicates that the disease is more frequent in blacks than in whites.

Sarcoidosis is predominantly a disease of young adults; most cases occur between the ages of 20 and 40, and the disease rarely develops before age 10 or after age 60. It may appear in a variety of guises; indeed, it has been called "the great masquerader." Thus, a general physician may initially be confronted with acute rheumatoid arthritis or lymphadenopathy; a physician specializing in diseases of the chest may see a patient with dyspnea or cor pulmonale; the dermatologist may see a case of erythema nodosum; the gastroenterologist may entertain a diagnosis of Crohn's disease.

As these examples suggest, the characteristic granulomatous lesions of sarcoidosis can affect almost any organ system, including the central nervous system. In about 25% of asymptomatic patients, the disease is diagnosed on the basis of a routine chest film.[1,7] Among symptomatic patients, the most common complaints[8] (in about 40% of cases) relate to the respiratory system: shortness of breath, nonproductive cough, nonspecific chest pain, and, occasionally, hemoptysis. More than 40% of patients, whatever their other signs and symptoms, will report such nonspecific symptoms as fever, sweating, anorexia, weight loss, fatigue, and myalgia.[8] Also common are skin lesions, with or without pulmonary symptoms: erythema nodosum (in about 10% of cases) and other lesions (about 15%), such as lupus pernio of the face, skin plaques on the extremities or trunk, and chronic scars. These figures represent overall and approximate prevalence rates for both white and black patients.[8]

Extrapulmonary manifestations of sarcoidosis, however, appear to be more prevalent in blacks, especially when they involve the skin. This is the more serious and chronic form of skin involvement which results in plaques, nodules, and lupus pernio. The other common type of skin involvement is erythema nodosum, which is more prevalent in whites than in blacks, and is usually associated with a good prognosis.

When a physician has reason to suspect sarcoidosis, a chest film should be taken, since virtually all patients, regardless of their symptoms, have some degree of thoracic involvement that will show up on film in at least 90% of cases. About 70% of roentgenograms will show the most distinctive sign of the disease—bilateral hilar adenopathy—either with or without changes in the lung parenchyma[7,8]; unilateral adenopathy is rare. The hilar involvement usually has a peculiar and distinctive appearance, often described as "potato nodes." About 20% of patients with sarcoidosis have pulmonary involvement without adenopathy—most often showing a reticulonodular pattern, alveolar infiltration, or both, on a chest film.[7,8]

The most useful procedure in confirming a diagnosis of sarcoidosis is a biopsy. If the characteristic skin lesions are present, the biopsy will be positive in nearly 90% of cases.[8] (Biopsies of the labial salivary glands or of the conjunctiva are not reliable, since they are negative in more than 40% of cases.) When skin lesions are not present, the procedure of choice is transbronchial biopsy of the lung parenchyma, which has replaced the earlier, invasive procedures of me-

---

Address correspondence to Dr Daniele, Professor, Cardiovascular-Pulmonary Division, Department of Medicine, Hospital of the University of Pennsylvania, 3400 Spruce St, Rm 807 E Gates Bldg, Philadelphia, PA 19104.

diastinoscopy and open lung biopsy.

In general, a reliable indicator of a good prognosis is absence of radiographic evidence of thoracic disease (stage 0): in such patients the rate of spontaneous recovery approaches 100%.[1] Another favorable sign is abrupt onset of the disease, especially if coupled with erythema nodosum (which, however, occurs in only about 10% of patients). Still other favorable indicators, although less absolute, are hilar enlargement with miliary or reticulonodular patterns (stage 2); normal or near-normal pulmonary function tests; and no evidence of involvement of other organ systems.

Because the course of disease is variable, and because 80% of patients have spontaneous remissions,[8] therapy for sarcoidosis has proved difficult to define. In about 20% of patients, however, the disease leads to progressive lung destruction. At present, there is no reliable way to predict which group of patients will have a poor outcome. Patients with hypercalcemia or eye, central nervous system, cardiac, or splenic involvement should receive therapeutic doses of corticosteroids. Patients with rapidly progressive pulmonary disease, as shown by clinical, functional, and radiographic criteria, should also be treated. In patients in whom the rate of deterioration occurs more slowly, the decision of whether to administer steroids is a difficult one. In this situation, one or more of the newer methods of disease assessment will be helpful (for example, bronchoalveolar lavage, gallium scanning, and serum angiotensin-converting enzyme assay).

Prognosis and management are particularly important in the black patient. It is in this group that the disease tends to manifest in extrapulmonary symptoms and tends to be chronic and more severe. Thus, it is important to look for extrapulmonary manifestations of the disease. It has been suggested that steroid therapy be initiated early, when there are indications of moderate disease.

REFERENCES

1. Daniele RP: Sarcoidosis: diagnosis and management. *Hosp Pract* 1983; 18:113-122.
2. Cummings MM, Dunner E, Schmidt H Jr, Barnwell JB: Concepts of epidemiology of sarcoidosis. *Postgrad Med* 1956; 19:437-446.
3. Teirstein AS, Lesser M: Worldwide distribution and epidemiology of sarcoidosis, in Fanburg BL (ed): *Sarcoidosis and Other Granulomatous Diseases of the Lung*. New York, Marcel Dekker, Inc, 1983, pp 101-134.
4. Sartwell PE: The state of the art: epidemiology, in Chretien J, Marsac J, Saltiel JC (eds): *Sarcoidosis and Other Granulomatous Disorders*. Proceedings of the Ninth International Conference. Paris, Pergamon Press, 1983, pp 229-233.
5. Teirstein AS, Siltzbach LE, Gabelman M, Lesser M: Socioeconomic factors influencing epidemiologic data in sarcoidosis among a mixed ethnic urban population, in Chretien J, Marsac J, Saltiel JC (eds): *Sarcoidosis and Other Granulomatous Disorders*. Proceedings of the Ninth International Conference. Paris, Pergamon Press, 1983, pp 234-236.
6. Mayock RL, Bertrand P, Morrison CE, Scott JH: Manifestations of sarcoidosis. Analysis of 145 patients with a review of nine series selected from the literature. *Amer J Med* 1963; 35:67-89.
7. Rossman MD, Dauber JH: Sarcoidosis: assessment of inflammatory activity, in Fishman AP (ed): *Update: Pulmonary Diseases and Disorders*. New York, McGraw-Hill Book Co, 1982, pp 193-204.
8. James PG, Neville E, Siltzbach LE, et al: A worldwide review of sarcoidosis. Proceedings of the Seventh International Conference on Sarcoidosis and Other Granulomatous Disorders. *Ann NY Acad Sci* 1976; 278:321-334.

INCREASING THE NUMBER OF MINORITY HEALTH PROFESSIONALS

Mr. Speaker, the severe underrepresentation of minority physicians, dentists, veterinarians, pharmacists, and other minority health professionals is a critical national problem that can no longer be studied, discussed, and debated. It is a problem that deserves priority national attention and action by Congress . . . .

Moreover, Mr. Speaker, I consider it a national disgrace that there has been little or no improvement in minority representation in the health professions, during a period when there has been a tremendous growth overall in the numbers of students being trained for service in the health industry. I can assure you, Mr. Speaker, that recent projections of a surplus of some types of health professionals in the United States by 1990 is totally unrelated to the status of blacks and other minorities in the health arena . . . .

Mr. Speaker, our country has the mot sophisticated health system in the world. Yet, the average life expectancy for black males is 6 years less than that of white males. The infant mortality rates for black babies is 21.4 per 1,000 live births, twice the rate for white babies. And, this gap is growing.

Our Nation's history has shown that a major factor in improving the health status of blacks and other minorities lies in expanding the supply of qualified health professionals and enhancing the likelihood that these professionals will practice in inner city and rural communities that are underserved. We also know for example, that black physicians also tend to locate in underserved areas and to specialize in primary medicine more so than other physicians. We know that this is true of other black health professionals as well.

Mr. Speaker, because minority health professionals and predominantly minority health professions schools are needed to address the severe unmet health care needs of our poor and minority citizens, we face a philosophical and ethical dillemma: Either we provide the financial and other resources needed to implement our country's creed of equal opportunity, or we abandon a large segment of our population to a high infant mortality rate, a shortened life expectancy, debilitating poverty, a crushing burden of illness and disability

and decreasing disillusionment, frustration and loss of hope. As the most affluent of nations and the leader of the free world, we should not allow this to happen.

THE HONORABLE LOUIS STOKES
(Ohio)
HR 5503, The Health Professions
Minority Training Assistance Act of 1984
Congressional Record
Tuesday, May 1, 1984

Coronary heart disease in black Americans

JAY BROWN, MD

The importance of coronary heart disease (CHD) in blacks in the United States has only recently received appropriate attention.[1-3] Epidemiologic and clinical research in CHD in the black population has been modest, and the data from some studies upon which current medical knowledge is based may not be applicable to the general black population. Epidemiologic studies, such as the Framingham Heart Study, have provided considerable data on CHD incidence and prevalence, coronary risk factors, and mortality for white populations.[4-7] No comparable data exist for blacks. Questions regarding the distribution, clinical expression, and natural history of CHD in black Americans remain.

In September 1983 the National Heart, Lung and Blood Institute convened the Working Conference on Coronary Heart Disease in Black Populations to review current knowledge of the subject and to make recommendations for future research.[8] The outcome of the conference included a recommendation for a large-scale, longitudinal study to determine the prevalence and incidence of CHD in blacks and to examine the relationship between established coronary risk factors and the development of CHD in this population. Another recommendation was to explore the influence of social and sociopsychologic behavior on the prevalence, morbidity, and mortality of CHD in blacks. A natural history study, designed to determine the accuracy of clinical and laboratory variables as predictors of CHD mortality in blacks, and to explore the question of sudden cardiac death in blacks, was also proposed.

These recommendations emanated from the paucity of current data and the conflicting information present in existing reports. The purpose of this report is to summarize some of the important studies on CHD in black Americans and to provide the basis for the recommendations of the working conference.

PREVALENCE AND INCIDENCE

The Evans County Cardiovascular and Cerebrovascular Epidemiologic Study (Evans County Study) was the first longitudinal study designed to determine the distribution of CHD in a population of southern blacks and whites and to assess the relationship between blood pressure, cigarette smoking, and serum lipid levels on the incidence and prevalence of CHD.[9,10] In this study prevalence estimates were derived from baseline examinations in which CHD was defined by the presence of angina pectoris, a history of myocardial infarction, or electrocardiographic evidence of myocardial infarction. Overall age-adjusted prevalence rates for CHD were found to be significantly lower in the black population. The rate for white men exceeded that for black men by nearly a 3 to 1 margin (59/1,000 versus 22/1,000). The rates did not differ significantly for white and black women (18/1,000 versus 14/1,000). The incidence of CHD, defined as all new cases of CHD at the time of a second examination approximately seven years after the initial one, was nearly 3 times higher in white men than in black men. No differences were noted for incidence rates among black and white women.

Another longitudinal population study of CHD in blacks, the Charleston (South Carolina) Heart Study, differed from the Evans County Study by the inclusion of a predominantly urban cohort.[11] Criteria for CHD were similar in both studies as was the method of determining prevalence and incidence rates. The study population included a random sampling of blacks and whites above the age of 35 years. As in the Evans County Study, black and white women had identical prevalence rates. Among the men the prevalence estimates were more than twice as high in whites as in blacks (75.8/1,000 versus 32.7/1,000). At the follow-up period 15 years later, black women and white men had experienced the highest incidence of CHD, 161/1,000 and 188/1,000, respectively. In black men the incidence was 132/1,000. It is of interest that a small cohort of black men with a higher socioeconomic status, followed over a shorter period, had the lowest incidence of all groups, 61/1,000.

These two studies showed higher prevalence rates of CHD among white men than black men and similar rates among black and white women. The findings may lack reliability because of the small numbers of blacks studied and the lack of representative data (the data were derived solely from southern black populations).

Acute myocardial infarction. Comparative incidence rates for acute myocardial infarction (AMI) between blacks and whites have been reported in a number of studies.[12-15] These studies consistently demonstrated higher age-specific AMI rates in white men than black men. Black-white comparisons in women, however, have shown no consistent differences. In the Nashville hospital surveillance study,[14] which focused on AMI in persons 35 to 74 years of age, overall rates in a one-year period were 2.1/1,000 and 5.9/1,000 for black men and white men, respectively. Rates for AMI were slightly higher in white women than in black women.

In a study of AMI based on a survey of 10 hospitals in Newark, NJ, during 1973, Weisse and colleagues[16] determined AMI rates for whites and blacks by age decade. No statistical differences in incidence between blacks and whites were evident.

In a similar study reported by Ahmed et al[17] age-decade specific AMI rates in whites exceeded rates in blacks only

---

Address correspondence to Dr Brown, Chief, Division of Cardiology, Department of Medicine, Harlem Hospital Center, Columbia University, College of Physicians and Surgeons, New York, NY 10037.

in the 60 to 69 year age group.

MORTALITY

The decline in cardiovascular mortality in the United States during the last two decades has been largely a result of a reduction in mortality rates from CHD.[18,19] This remarkable decline in death rates from CHD is demonstrable in all races and sexes. Nonetheless, CHD remains the major cause of deaths in the adult population in this country. Among black Americans, CHD may account for 25% of all deaths.[1]

Using mortality data from the National Center for Health Statistics, Gillum and Liu[19] calculated age-adjusted CHD death rates for whites and non-whites for the period from 1940 to 1979. (Vital statistics were maintained for two groups: whites and all other races, designated non-white. Because blacks constituted more than 85% of this latter category, the non-white rates approximated rates for blacks.) Non-white CHD mortality rates increased from the 1940s to a peak in 1968, when rates for non-white men exceeded the rates for white men. Rates for non-white women have remained higher than those for white women since the late 1940s. A downturn in CHD mortality occurred in all groups in the late 1960s, with a steeper decline appearing in the non-white groups. Death rates for white and non-white men remained similar between 1970 and 1979. For the female groups, rates were higher among non-whites.

This decline in CHD mortality is evident in most of the United States. There are, however, striking differences in mortality rates between regions of the country and between states.[20] Clusters of states in the South, including West Virginia, Kentucky, Delaware, North Carolina, and South Carolina, as well as Illinois and Louisiana, have consistently high CHD mortality rates among blacks.[21] Intrastate differences have also been reported.[22]

Data from the National Health and Nutrition Examination Surveys (1971 to 1975 and 1976 to 1980) suggest that the decline in the prevalence of two coronary risk factors, cigarette smoking and hypertension, explains in part the reduction in CHD mortality in blacks.[22]

Mortality following myocardial infarction. Available data on mortality in blacks following myocardial infarction are limited, which precludes any conclusions regarding the influence of race on prognosis following infarction.[15]

Recent data relevant to this point come from the Beta-Blocker Heart Attack Trial (BHAT).[23,24] Although the study was not designed to evaluate differences between blacks and whites in response to beta-blockers, the black-white mortality differences are interesting. Subgroup analysis of the data reveals a trend toward continuous higher mortality among blacks, regardless of their treatment assignments.[25] Total mortality in blacks treated with propranolol was 11% compared to 6.7% in whites assigned to this treatment group. It is of interest that a difference in mortality between blacks in the treated groups and blacks in the control group did not appear until the 18th month of the trial. Among whites, the mortality differences between treated and control groups were established early and continued throughout the trial.

The reason for the lower survival among blacks is not certain. The clinical variables noted at the start of the study that may have predicted a greater risk of mortality were statistically higher in the black groups. These included hypertension, cardiomegaly, diabetes mellitus, and a history of smoking.

These data must be interpreted with caution because of the small size of the black study group—only 6.8% of the total BHAT study group—and because of the possibility of selection bias.

CORONARY RISK FACTORS

The Evans County Study reported a relationship of hypertension, blood cholesterol ≥260 mg/dL, and cigarette smoking to CHD incidence in black men.[26,27] For black women, increased risk factor levels did not predict an increased incidence of CHD. Gillum and Grant[2] questioned the statistical power of the study because of the small number of CHD cases.

The association between the decline in CHD mortality and the reduction in the prevalence of hypertension and cigarette smoking in blacks suggests a relationship between the development of CHD and coronary risk factors in this population. However, the relative strength of each standard coronary risk factor is unknown in blacks. Higher than observed CHD prevalence rates might be expected because of the high prevalence rates of hypertension and diabetes mellitus in the black population. Perhaps the effect of these risk factors in the pathogenesis of CHD is modulated by other factors, such as serum concentration of high-density lipoprotein cholesterol, which has been shown to be higher in blacks than in whites.[28,29]

The role of social, economic, and environmental factors in the development of CHD and its subsequent morbidity and mortality are unknown. Higher mortality rates from CHD were reported in both black and white men with lower socioeconomic status in the Charleston Heart Study.[11] These findings suggest that socioeconomic conditions, rather than race itself, may influence CHD mortality in the population.

CONCLUSIONS

Epidemiological data on CHD prevalence and incidence in black Americans are limited and are not representative of the black population. Some clinical and population-based studies provide inconsistent and conflicting information and are inadequate to promote understanding about CHD in blacks. The recommendations of the Working Conference on Coronary Heart Disease in Black Populations are timely and appropriate.

REFERENCES

1. Gillum RF: Coronary heart disease in black populations. I. Mortality and morbidity. *Am Heart J* 1982; 104:839–851.
2. Gillum RF, Grant CT: Coronary heart disease in black populations. II. Risk factors. *Am Heart J* 1982; 104:852–864.
3. Proceedings of the symposium on coronary heart disease in black populations. *Am Heart J* 1984; 108:727–862.
4. Kannel WB, Dawber TR, Kagan A, Revotskie N, Stokes J: Factors of risk in the development of coronary heart disease—six-year follow-up experience: The Framingham Study. *Ann Intern Med* 1961; 55:33–50.
5. Doyle JT, Dawber TR, Kannel WB, Kinch SH, Kahn HA: The relationship of cigarette smoking to coronary heart disease. *JAMA* 1964; 190:886–890.
6. Stamler J: Cigarette smoking and atherosclerotic coronary heart disease. *Bull NY Acad Med* 1968; 44:1476–1494.
7. Brand RJ, Rosenman RH, Sholtz RI, Friedman M: Multivariate prediction of coronary heart disease in the Western Collaborative Group study compared

to the findings of the Framingham Study. *Circulation* 1976; 53:348–355.

8. Report of National Heart, Lung and Blood Institute Working Conference on coronary heart disease in black populations. *Am Heart J* 1984; 108:633–726.

9. McDonough JR, Hames CG, Stubb SC, Garrison GE: Coronary heart disease among Negroes and whites in Evans County, Georgia. *J Chronic Dis* 1965; 18:443–455.

10. Cornoni JC, Wailler LE, Cassel JC, Tyroler HA, Hames CG: Evans County Cardiovascular and Cerebrovascular Epidemiologic Study. The incidence study—study design and methods. *Arch Intern Med* 1971; 128:896–900.

11. Keil JE, Loadholt CB, Weinrich MC, Sanifer SH, Boyle E: Incidence of coronary heart disease in blacks in Charleston, South Carolina. *Am Heart J* 1984; 108:779–786.

12. Shapiro S, Weinblatt E, Frank CW, Sager, RV: Social factors in the prognosis of man following first myocardial infarction. *Milbank Mem Fund Q* 1970; 48:37–50.

13. Kuller L, Perper J, Cooper MB: Demographic characteristics and trends in arteriosclerotic heart disease mortality: sudden death and myocardial infarction. *Circulation* 1975; 52:1–15.

14. Hagstrom RM, Federspeiel CF, Ho YC: Incidence of myocardial infarction and sudden death from coronary heart disease in Nashville, Tennessee. *Circulation* 1971; 44:884–890.

15. Goldberg RJ, Kennedy HL: The influence of race on prognosis after acute myocardial infarctions: a community-wide perspective. *J Cardiac Rehab* 1983; 3:195–201.

16. Weisse AB, Abiaso PD, Thind IS: Acute myocardial infarction in Newark, N.J.: a study of racial incidence. *Arch Intern Med* 1977; 137:1402–1405.

17. Ahmed SS, Rozefort R, Brancato R: Incidence of acute myocardial infarction among blacks in an urban community. *J Med Soc NJ* 1977; 74:1058–1060.

18. Cooper R, Stamler J, Dyer A, Garside D: The decline in mortality from coronary heart disease, U.S.A., 1968–1975. *J Chronic Dis* 1978; 31:709–720.

19. Gillum RF, Liu KC: Coronary heart disease mortality in United States blacks, 1940–1978: trends and unanswered questions. *Am Heart J* 1984; 108:728–732.

20. Leaverton PE, Feinleib M, Thom T: Coronary heart disease mortality rates in United States blacks, 1968–1978: interstate variation. *Am Heart J* 1984; 108:732–737.

21. Keil JE, Lackland DT, Hudson MB, et al: Coronary heart disease and stroke mortality in South Carolina: geographical and temporal trends. *J SC Med Assoc* 1983; 79:65–69.

22. Rowland ML, Robinson F: Coronary heart disease risk factor trends in blacks between the first and second National Health and Nutrition Examination Surveys, United States, 1971–1980. *Am Heart J* 1984; 108:771–779.

23. Beta Blocker Heart Attack Study Group: The Beta Blocker Heart Attack Trial: preliminary report. *JAMA* 1981; 246:2073.

24. Beta Blocker Heart Attack Trial Research Group: A randomized trial of propranolol in patients with acute myocardial infarction. I. Mortality rates. *JAMA* 1982; 247:1707–1714.

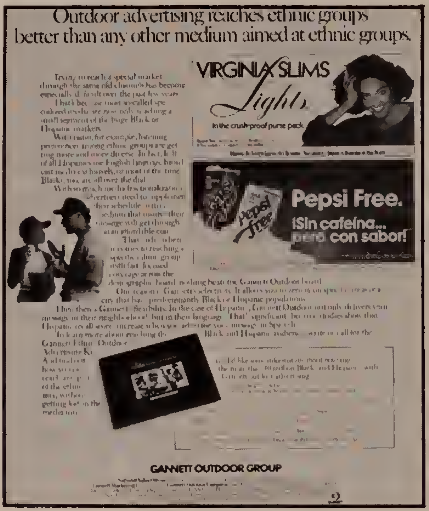
25. Haywood JL: Coronary heart disease mortality, morbidity, and risk in blacks. I. Clinical manifestations and diagnostic criteria: the experience with the Beta Blocker Heart Attack Trial. *Am Heart J* 1974; 108:787–796.

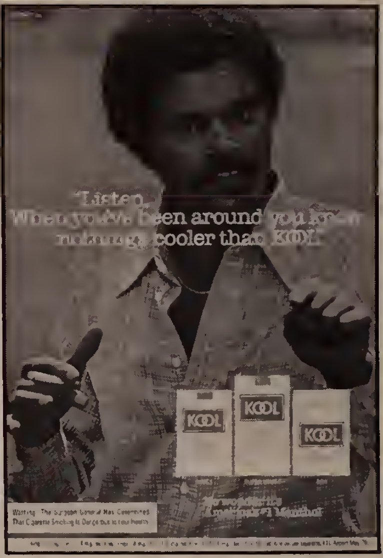
26. Tyroler HA, Heyden S, Bartel A, et al: Blood pressure and cholesterol as coronary heart disease risk factors. *Arch Intern Med* 1971; 128:907–914.

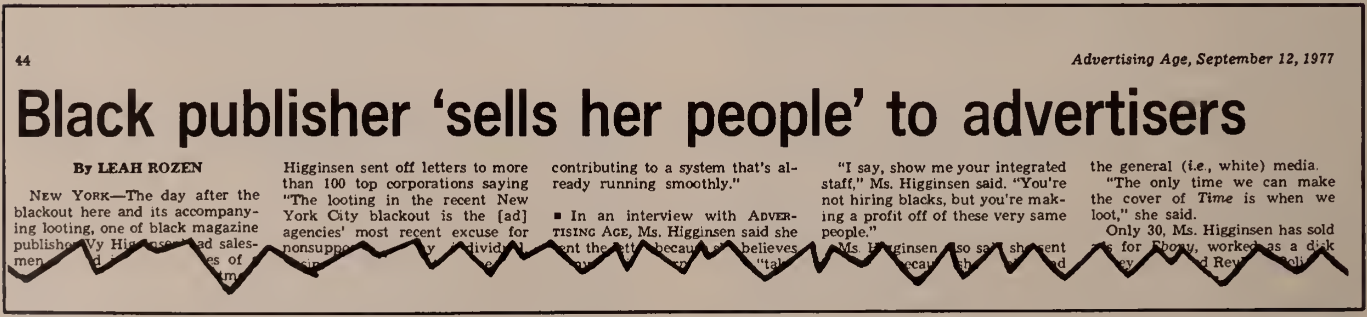
27. Heyden S, Cassel JC, Baryel A, Tyroler HA, Hames CG, Cornoni JC: Body weight and cigarette smoking as risk factors. *Arch Intern Med* 1971; 128:915–919.

28. Gartside PS, Khoury P, Glueck CJ: Determinants of high-density lipoprotein cholesterol in blacks and whites: the second National Health and Nutrition Examination Survey. *Am Heart J* 1984; 108:641–653.

29. Curry CL, Oliver J, Mumtaz FB: Coronary artery disease in blacks: risk factors. *Am Heart J* 1984; 108:653–657.


Outdoor advertising reaches ethnic groups better than any other medium aimed at ethnic groups.
VIRGINIA SLIMS
Lights
In the crush-proof purse pack
Pepsi Free.
¡Sin cafeína... pero con sabor!
GANNETT OUTDOOR GROUP




44 *Advertising Age, September 12, 1977*

Black publisher 'sells her people' to advertisers

By LEAH ROZEN

NEW YORK—The day after the blackout here and its accompanying looting, one of black magazine publishe

Higginsen sent off letters to more than 100 top corporations saying "The looting in the recent New York City blackout is the [ad] agencies' most recent excuse for nonsuppo

contributing to a system that's already running smoothly."

■ In an interview with ADVERTISING AGE, Ms. Higginsen said she ent the believes

"I say, show me your integrated staff," Ms. Higginsen said. "You're not hiring blacks, but you're making a profit off of these very same people."

the general (*i.e.*, white) media.

"The only time we can make the cover of *Time* is when we loot," she said.

Only 30, Ms. Higginsen has sold s for *Ebony*, worked as a d

The rise in heart disease among blacks has also been paralleled by the rise in cigarette advertising directed toward the black consumer market. Billboards, nearly all of which promote either cigarettes or alcohol, are far more prevalent in black and Hispanic communities than predominantly white neighborhoods. In a report on marketing to minorities (November 19, 1984) an *Advertising Age* reporter wrote, "Many experts in the outdoor ad industry believe outdoor boards geared to the black consumer measure the pulse of corporate America's attitude toward the black public."

THE GENE DOCTORS: MEDICAL GENETICS AT THE FRONTIER

By Yvonne Baskin. 288 pp. New York, William Morrow & Company, 1984. $13.95 (softcover).

In her first full-length book, popular science writer and reporter Yvonne Baskin traces the major events during the past century that led up to what promises to become one of the most exciting and controversial developments in modern medicine: the prevention and treatment of disease by primary genetic manipulation.

After a brief introductory overview, the author outlines the development of the current understanding of the relationship between genetics and disease. This includes chapters on inborn errors of metabolism; the genetics of common multifactorial diseases such as diabetes, heart disease, and cancer; and a review of current approaches to the management of genetic disease, including organ and bone marrow transplantation. The third part of the book recounts, in greater depth, the history of the isolation and localization of specific genes, the discovery of restriction fragment length polymorphisms, and the potential application of these discoveries in the diagnosis and prevention of genetic disease. Part four contains a review of the methods by which foreign genes have been introduced into the eukaryotic genome. It includes an engaging chapter that describes the events leading up to Martin Cline's controversial attempts to treat thalassemia by exposing the patient's bone marrow cells to purified human beta-globin genes in vitro and re-infusion of the gene-treated material. Other chapters in the section address the regulation of gene expression and Mulligan's and Jaenisch's elegant studies on the retrovirus. In the fifth and final part of the book, Baskin speculates on what might be expected in the future with respect to gene transfer therapy, and what is clearly not feasible in the foreseeable future. Included are some thought-provoking comments on the moral, ethical, social, and religious implications of gene therapy.

Throughout the book, Baskin employs a brisk journalistic style which heightens the drama and excitement of the events. For the most part, however, she lets the events and personalities involved speak for themselves, often dramatizing, but only rarely sensationalizing them. The considerable use of material obtained by personal interviews provides an intimacy that adds to the appeal of this story. On the other hand, the quick journalistic pace is not well suited to the exploration of conceptually difficult issues, such as restriction fragment length polymorphisms, or philosophically abstruse matters relating to the ethics of gene therapy. For all the detailed information packed into the book, it contains surprisingly few errors most of which, such as the date of the first use of insulin in humans, are relatively trivial. The mixing of a discussion of chromosomal abnormalities with the review of inherited metabolic diseases in chapter 6 is unconventional and potentially confusing.

The conspicuous efforts to simplify the jargon of modern genetics and the liberal and often imaginative use of analogies to explain various genetic concepts indicate that the book is intended to appeal to laymen. However, the density of the presentation along with the total absence of any diagrams or figures is likely to make it rather arduous for all but the most biologically sophisticated. On the other hand, physicians, geneticists, and others with a reasonably sound background in biology will find plenty to hold their attention.

Overall, Baskin has succeeded in producing an interesting and personalized, though well balanced account of developments in the history of gene therapy which should appeal to a range of biology-watchers.

J. T. R. CLARKE, MD, PhD
The Hospital for Sick Children
Toronto, Canada M5G 1X8

AFRICAN FEVER: A STUDY OF BRITISH SCIENCE, TECHNOLOGY, AND POLITICS IN WEST AFRICA, 1781–1864.

By Dennis G. Carlson. 108 pp. Canton, MA, Science History Publications, 1984. $15.95 (softcover)

Anyone familiar with the history of West Africa is aware that malaria had an enormous impact on early exploration, travel, and colonial annexation. West Africa was known as "The White Man's Grave" because of the staggering mortality among Europeans who either visited or resided there. In 1805, for example, the Scottish physician-explorer Mungo Park set out for the interior of West Africa, to explore the course of the Niger River. Of the 44 Europeans who started out with him, only four were still alive six months later when he arrived at Bamako, now the capital of Mali; most had succumbed to fever.

It is now known that most episodes of fever suffered by these early travelers were due to malaria. Less frequently the explorers suffered from yellow fever. Although it had long been known that cinchona bark was effective in treating fever, it was rarely used appropriately by 18th and early 19th century travelers in Africa. As the author effectively describes, this was due to several factors, among which were the theoretical chaos that dominated European and American medicine concerning the causes of fever, the dominance of bleeding, use of mercury and tonics in the treatment of fevers, and the lack of convincing objective evidence that cinchona bark was specific for African fever.

For much of the 19th century, miasms and foul air were thought to be the cause of what we now know to be malaria. Although quinine was isolated from cinchona bark in 1820 by Pelletier and Caventou, its specificity and effectiveness against malaria were not accepted for more than 30 years. Why this was so and how and why medical thinking about quinine slowly changed constitute the central theme of this book. The book is divided into seven chapters. In "Lure and Danger," the author succinctly describes the influence of febrile illnesses in West Africa on several important early British exploratory and trade expeditions. These early experiences were characterized by staggering mortality among Europeans and the observation that Africans were fairly immune to fevers. In the next chapter, "Marshes and Miasms," prevailing early 19th century views of malaria are discussed. This is followed by two chapters, "Theoretical Chaos" and

"Bleeding, Mercury and Tonics" that clearly explain why it took so long for medicine to accept the fact that quinine was an effective and specific agent for both the prevention and treatment of malaria.

In chapter five the author details the remarkable progress of British statistical studies in the 1830s and 1840s and their influence in changing attitudes of medical thinkers about many aspects of disease. As far as malaria was concerned, the real breakthrough came when the British military decided to analyze morbidity and mortality data on their personnel in various parts of the world. The man they placed in charge of this effort, Alexander Murray Tullock, was a graduate of Sandhurst with a bent for reform. He analyzed data from 1817 through 1836 over a five-year period (systematic reporting of the British Army's health matters began in 1816). The data were analyzed from the points of view of geographic location, diet, tours of duty, accommodations, temperature, humidity, rainfall, soils, altitude, and agriculture.

Tullock's report was presented to Parliament in 1836 and published in 1838. Although it did not lead to changes in how African fever was treated and prevented, it did bring about less reliance on anecdote and subjective impressions, replacing them with analyses of carefully collected data.

Further progress in establishing quinine's effectiveness came a decade later with Dr Alexander Bryson's studies of the health records of British navy personnel assigned to ships in African waters. He came to the firm conclusion, based not only on his studies, but also on his first hand experiences aboard ships off the African coast, that quinine was extremely effective in preventing febrile illness in the tropics. Bryson also recognized the 14- to 20-day incubation period of malaria and recommended in his 1847 report that the drug be administered for at least 14 days after personnel had returned to ships after being on land.

As the author states, a new paradigm emerged for the explanation of febrile illness in Africa. Bryson's views took root in a receptive medical environment and in the company of Peter Panum's studies of measles on the Faroe Islands (1846) and John Snow's initial cholera studies (1848–1849).

By 1848, Bryson's conclusions had been translated into medical policy in the British army and navy. Prevention and treatment guidelines using quinine for malaria were established. The results were spectacular. This breakthrough had an enormous impact on the history of subsequent European-African contacts. Commerce and trade, evangelism, and colonial annexation were all accelerated once the major impediment to European health had been removed. It would be another 33 years before Laveran would discover malarial parasites in human red blood cells, and a half century before the transmission cycle of malaria would be fully understood.

From one perspective, this book is an account of how epidemiologic investigations produced data that influenced the beliefs of a new generation of medical thinkers for whom the old dogmas had failed. Thus, it was not only the discoveries themselves and their dissemination that made them successful, but the receptive environment into which they were introduced.

Carlson is a skilled medical historian who has used meticulous scholarship to produce a concise and clear account of the triumph of science over dogma. Written in an engaging and easy to read style, this book will have broad appeal to physicians and those interested in medical history.

PASCAL JAMES IMPERATO, MD
Deputy Editor

PRETERM BIRTH: CAUSES, PREVENTION AND MANAGEMENT

Edited by Fritz Fuchs, MD and Phillip G. Stubblefield, MD. 392 pp, illustrated. New York, Macmillan Publishing Company, 1984. $48.00 (hardcover)

This book is a timely publication on the subject of prematurity. The editors and invited authors are recognized for their contributions in this field.

The first section of the book is an excellent review of physiologic and endocrinologic mechanisms; however, the section on sociologic factors is cumbersome and makes for difficult reading. The discussion of infections is brief and omits a significant amount of recent laboratory and clinical information pertaining to the role of infection in preterm births. Similarly, the relationship of smoking and preterm birth is given only brief consideration.

The section on tocolytic therapy is concise and contains helpful tables, figures, and references. The drugs used, and their specific actions, indications, and side effects are described in detail. This section should be very instructive for clinicians caring for patients who undergo premature labor.

The last section on management and outcome lacks detail and is incomplete. More emphasis should have been given to the use of steroids for the induction of fetal lung maturation, amniocentesis, and care of patients during delivery.

A concluding chapter on future treatment methods for premature labor, especially relating to prevention and advances in neonatal care, would have been appropriate. Overall, this book offers a good review of preterm birth, and it would be an excellent addition to the library of those caring for patients with preterm labor.

JULES MOODLEY, MD
STEPHAN R. SANKO, MD
University of Rochester
School of Medicine
Rochester, NY 14627

MAKING AN ISSUE OF CHILD ABUSE. POLITICAL AGENDA SETTING FOR SOCIAL PROBLEMS

By Barbara J. Nelson. 169 pp. Chicago, The University of Chicago Press, 1984. $17.50 (hardcover)

The author did not write this book out of interest in the problems of child abuse or to explain this disturbing phenomenon. Rather, she sought to answer more general questions about policy initiation. She views the issue of child abuse as a good example of governmental influences on agenda-setting and as a subject all but absent from the literature on the setting of priorities. The author approaches the subject from the perspective of her stated political views. She characterizes the United States as a country with no successful socialist tradition and little support for redistribution of wealth. In view of this, she claims that "there seemed to be a need for research on smaller, categorical programs where group demands for special social intervention are met." Thus, child abuse was seen as a compelling issue on which to base a study.

The author's basic perspective on child abuse is summarized as follows: the public use of private deviance. She concludes that "the scenario of having done too much and too little is one consequence of liberal reform. Lacking support for significant social reordering, American reformers are faced with two unsatisfactory alternatives." Incremental change, which may not be enough, or comprehensive change, which may never happen. Her position appears to coincide with her statement that "socialist critics add that individually centered responses to social problems are designed to maintain existing relations."

The presentation and analysis of historical data suffer from the author's lack of the historian's skills. Many significant developments in the recent evolution of child protective services and child abuse policy are overlooked, and her gathering of facts is highly selective. Nelson's knowledge is limited by issues or individuals related to her sources, which results in an unreliable presentation. For example, her discussion of legislation never mentions the individual who has made the most influential contribution, Douglas Besharov—who drafted New York State's Child Protective Services Act and subsequently became the first director of The National Center for the Prevention of Child Abuse and Neglect. This contribution became the most widely used model in the development of child abuse legislation in many states during the 1970s.

The material in the book is too incomplete to rely on as an accurate history of child abuse in the United States or of how our society has responded to it. Another book would be needed to explain and correct all the omissions and errors in this one. The book has no interest or utility for clinicians who treat victims of child abuse and neglect, and it is too flawed to be of any help to policy makers.

VINCENT J. FONTANA, MD
The New York Foundling Hospital
New York City 10021

THE AGING MUSCULOSKELETAL SYSTEM: PHYSIOLOGICAL AND PATHOLOGICAL PROBLEMS

Edited by Carl L. Nelson, MD and Anthony P. Dwyer, MD. 178 pp, illustrated. Lexington, MA, The Collamore Press, 1984. $30.00 (hardcover)

This book summarizes the results of a conference held at the University of Arkansas in September of 1981. The chapters address issues related to aging: the demographics of aging, psychosocial factors, the biology and physiology of aging, the biology of osteoarthritis and osteoporosis, and the use of surgery and rehabilitation techniques in managing problems associated with aging. The contributors to this volume are among the outstanding specialists in the field. They comment on topics such as osteoporosis; the kinematics of aging, with specific emphasis on the lumbar spine; the pathogenesis of osteoarthritis, with some suggestions for treatment; total hip replacement in the elderly; trauma; hip fractures; and rehabilitation medicine in the aging population.

One of the major defects of the book is the failure to discuss inflammatory musculoskeletal disease in the elderly, including rheumatoid arthritis, polymyalgia rheumatica, and polymyositis. All of these conditions are common in the elderly and can be reversed with appropriate therapy. Some of the chapters, such as those dealing with osteoporosis, are rich in detail, whereas others, such as the one on the pathoanatomy of aging, are sketchy.

This volume is an excellent summary of the 1981 conference and contains a comprehensive review of the aging musculoskeletal system, pinpointing some, but not all, major areas. It is a useful addition to the libraries of those interested in problems of the aging. However, a volume that is more comprehensive and better organized would be more useful.

HARRY SPIERA, MD
The Mount Sinai Medical Center
New York City 10029

COLOR ATLAS OF GERIATRIC MEDICINE

By Asif Kamal, MD, J. C. Brocklehurst, MD. 176 pp, illustrated. Oradell, Medical Economics Books, 1984. $59.95 (hardcover)

The purpose of this book is to aid those who care for elderly persons who are frail and ill. The authors present the significant differences in the clinical patterns of disease that reflect the normal and abnormal changes associated with aging. Emphasis is given to a range of illustrative material supported by descriptions of the etiology, clinical features, differential diagnosis, and importance of significant conditions affecting the aged.

The authors are experienced geriatricians. Dr Brocklehurst is an internationally recognized authority and a professor of geriatrics at the University of Manchester, England.

The book consists of three brief introductory chapters: "Geriatric Medicine," "Special Features of Illness in Old Age," and "Assessment," followed by an extensive series of illustrated chapters dealing with specific areas of the body. There is a suggested reading list and an index. There are no references in the text, which I believe is an advantage in this type of publication.

The chapter on geriatric medicine is well done, with a primary emphasis on the nature of the subject in Great Britain. The chapter on the special features of medicine in old age is an excellent discussion of the multiple causes and multisystemic effects of disease in the aged, including incontinence. A range of other conditions, such as infections, constipation, and musculoskeletal, cardiac, and psychogeriatric disorders, as well as malignancies are discussed. The chapter on assessment deals with history taking, and physical and mental examination—with special attention to postural hypotension. Hypothermia, a condition of significance in Great Britain, is given special emphasis.

Other chapters deal with various anatomic areas of the body including the head, face, neck, chest, abdomen, upper limbs, lower limbs, and skin.

Each chapter is illustrated with photographs and micrographs of the highest technical quality, and x-rays and diagrams, with accompanying brief descriptions of relevant clinical features.

The book meets the authors' stated objectives, as noted in the preface, of providing a valuable aid to caregivers to the aged, including practicing and house physicians, medical students, nurses, and physical and occupational therapists. I think that it will be a useful and attractive addition to any personal or institutional library.

MANUEL RODSTEIN, MD
Kingsbridge Center
The Jewish Home and
Hospital for the Aged
New York City 10468

PRISON HEALTH CARE

By Richard Smith, BSc, MB ChB. 182 pp, illustrated. London, UK, The Devonshire Press, 1984. (softcover)

This book is an informative and readable work focusing on health issues in British and Welsh prisons. A unique approach is used by the author (a doctor and a journalist) in relating health concerns to an overall comprehensive description of the life of incarcerated individuals. In these institutions the priority is containment of dangerous men. Health care is an afterthought and even a contradiction in this arena.

Dr Smith visited 20 prisons in five countries in preparing this book. The work is based on a series that previously appeared as articles in the *British Medical Journal*, where he is an assistant editor. These articles have been revised and augmented, and the resulting work describes the British prison medical service, with references to several Dutch and American penal institutions.

The author addresses several questions: What do prison doctors do? What types of medical problems are most common in the prison? Is it possible to practice good medicine in spite of the difficult conditions in the prison? How can suicide be prevented? The analysis of these issues is valuable because health problems are related to broader issues within prisons. These problems include overcrowding, living conditions, rioting, and lack of leadership and poor morale among correctional staff. These factors of prison life are as important to health as the medical services available to the incarcerated population. Throughout the book the contradiction between a concern for health within the prison walls and the institutional priority for confining dangerous men is correctly shown to be the basic impediment. The author describes with clarity and depth conditions in British prisons and the viewpoints of those who provide security and those who provide health care.

The work is less comprehensive in its treatment of the physical health of prisoners and the organization of medical services. The author rightfully refers to the hazy picture of the health of prisoners but does not present information that adds clarity to this subject. The organization of medical services is treated in the book largely within the confines of the British system and lacks a full exposition of alternative methods of contracting for prison health care and the utilization of alternative providers. The chapters on prison doctors, ethics, and women in prison are helpful and stimulating to anyone with an interest in improving health services for the incarcerated.

There is a paucity of published observations on the interactions between the health of prisoners and the correctional institution. There is a similar lack of attention to the provision of services themselves. This book addresses both of these issues.

Overall, this book presents a positive and knowledgeable outlook, with useful recommendations for improving medical services in prisons and relating these improvements to the overall penal institution. Health services are not treated in a vacuum but as a component of the larger system that is refractory to health concerns.

LLOYD NOVICK, MD
Director
Department of Health Services
Phoenix, AZ 85007

CARDIOLOGY 1984

Edited by William C. Roberts, MD. 512 pp, illustrated. New York, Yorke Medical Books, 1984. $49.50 (hardcover)

Unfortunately, the editor of this volume does not provide a stated purpose for this book. One has the impression from the preface that the editor and six other eminently qualified experts reviewed the leading American and British cardiology and general medical journals and selected the most important articles published in 1983. They have written summaries of these, with the intent of providing a digest for busy cardiologists.

I chose three summaries at random from the book and compared them to the original journal articles. In each instance, except for the initial sentence and the occasional use of different abbreviations, each summary was a verbatim transcript of the original article abstract. This sample leads one to suspect that the majority, if not all, of the summaries are of the same type. If this in fact is true, it should be clearly and explicitly stated in the preface.

The abstracts sampled were, for the most part, short terse paragraphs containing a large amount of information and statistical data telescoped in such a way that it made reading the book difficult. There is little critical comment by the editor or the other contributors.

This volume is the fourth in a series published annually and must be compared with the long running *Yearbook of Cardiology*. The 1984 edition of the latter reviews many of the same original articles selected for *Cardiology 1984* but does so with longer, well-written summaries which include useful, critical comments by the reviewers; as a result, it is much more interesting and readable.

The usefulness of *Cardiology 1984* lies primarily in learning which journal articles published in 1983 were considered by a group of prominent specialists to be the most important original contributions to the cardiology literature. Otherwise, this book cannot be recommended.

JOSEPH SCHLUGER, MD
The Long Island College Hospital
New York City 11201

SUMMER POSITION FOR A MEDICAL STUDENT AT THE *JOURNAL*

Applications are invited from medical students (including those who will be entering medical school in the fall) for a 10-week editorial assistantship at the *Journal* for an issue on alcohol problems. Inquiries should be directed to the Editor. Applications should consist of a curriculum vitae and a covering letter. Deadline: May 20.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

Schedule of Sessions 171
Officers, Councilors, and Trustees 173
Members of the House 176
Reference Committees 180
Nominating Committee 182
Report of the House Committee on Bylaws 183

HOUSE OF DELEGATES

June 6 through 9, 1985
New York Hilton

SCHEDULE of SESSIONS

Thursday, June 6	
Opening Session	1:00 p.m.
Friday, June 7	
Reference Committee Hearings	8:30 a.m.–12:30 p.m.
Reference Committee Hearings	1:30 p.m.–6:00 p.m.
Reference Committee Hearings	8:00 p.m. (to completion)
Saturday, June 8	
Second Session	9:00 a.m.
Third Session	1:30 p.m.
Fourth Session	8:00 p.m. (If necessary)
Sunday, June 9	
Closing Session	9:00 a.m.

A Guide to the 1985 Annual Meeting

Policy-making in The House of Delegates

This section of the New York State Journal of Medicine is a call to the officers, delegates, and members of the Medical Society of the State of New York to attend the 179th Annual Meeting of the Society and the meeting of its House of Delegates; 293 delegates representing 61 county medical societies, 9 district branches, specialty societies, the hospital medical staff section, 12 medical schools, the resident and medical student membership will meet at the Hilton Hotel in New York City on June 6–9, 1985.

The officers, members of the Council and the Board of Trustees, past presidents of the MSSNY, the Commissioner of the New York State Department of Health, and the Past President of the American Medical Association from New York, Gerald D. Dorman, MD, are also official representatives to the House.

Mandated by the Bylaws of the Society, this section lists the names of elected representatives, delegates who are serving on reference committees, and selections of the Nominating Committee for the election of Officers, Councilors, Trustees and AMA Delegates. Also included are the amendments to the Bylaws which will be considered by the House of Delegates.

Since the House of Delegates is the legislative body of the Society, charged with the general management of the State Society and its affairs, members of the Society are urged to use this information to consult with and advise their representatives. Resolutions introduced by representatives bring members' concerns to the attention of the entire Society. Representatives not only have the responsibility to present the views of their constituents, but also to inform them of the actions taken by this governing body. The House of Delegates also formulates policy for the Society. No officer, councilor, board, commission, committee, or employee may initiate any policy unless such policy has been expressly approved by the House of Delegates or by the Council of the Society.

If one were to choose the most important work of the House of Delegates, it would have to be that of the reference committees. Any member of the Medical Society of the State of New York has the right to appear before any reference committee and participate in its deliberations. It is also in the reference committees that people outside of the State Society are asked to give their views. Nonmember physicians, guests or interested observers who wish to express their opinions may, with permission, come before the reference committee and speak to the business of the committee. I strongly urge members to use this forum to express their views.

John L. Clowe, MD, Speaker
House of Delegates

MEDICAL SOCIETY OF THE STATE OF NEW YORK

ORGANIZED 1807

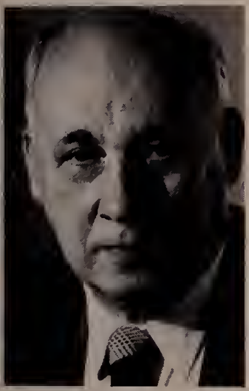
Officers 1984–1985

Allison B. Landolt

Westchester
President

Daniel F. O'Keeffe

Warren
President-Elect



Bernard J. Pisani

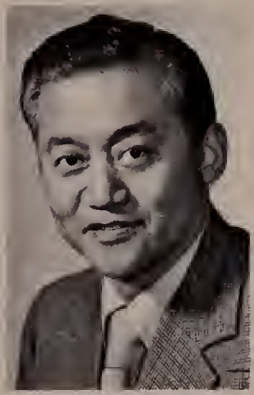
New York
Past-President

John A. Finkbeiner

New York
Vice-President

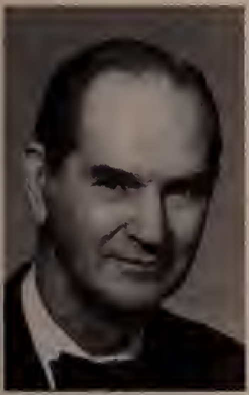
John H. Carter

Albany
Secretary



George Lim

Oneida
Assistant Secretary

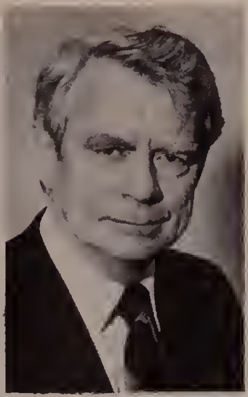


Charles D. Sherman, Jr.

Monroe
Treasurer

Morton Kurtz

Queens
Assistant Treasurer



John L. Clowe

Schenectady
Speaker

Charles N. Aswad

Broome
Vice-Speaker

Board of Trustees 1984–1985

Carl Goldmark, Jr.

New York
Chairman

G. Rehmi Denton

Albany

Ralph M. Schwartz

Kings

Edgar P. Berry

New York

Richard D. Eberle

Onondaga

Joseph F. Shanaphy

Richmond

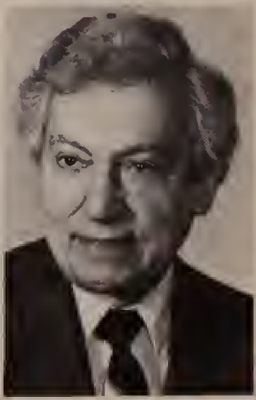
Victor J. Tofany

Monroe

Councilors 1984–1985

James H. Cosgriff, Jr.

Erie



Samuel M. Gelfand

Nassau

Richard A. Hughes

Warren

Antonio F. LaSorte

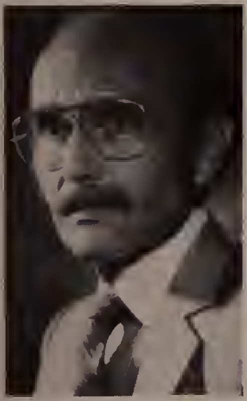
Broome

Robert A. Mayers

Westchester

Thomas D. Pemrick

Rensselaer



Ralph E. Schlossman

Queens

Stanley A. Steckler

Suffolk

David M. Benford

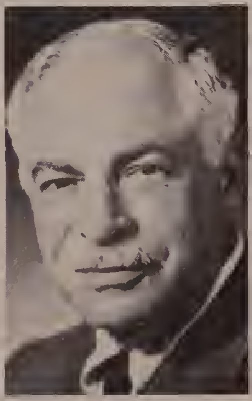
New York

Duane M. Cady

Onondaga

James M. Flanagan

Wayne



M. Theodore Tanenhaus

Kings

1985 HOUSE OF DELEGATES MEMBERS

The following is a list of members of the 1985 House of Delegates of the Medical Society of the State of New York:

Officers 1984–1985
President—Allison B. Landolt, Westchester
President-Elect—Daniel F. O'Keeffe, Warren
Vice-President—John A. Finkbeiner, New York
Secretary—John H. Carter, Albany
Assistant Secretary—George Lim, Oneida
Treasurer—Charles D. Sherman, Jr., Monroe
Assistant Treasurer—Morton M. Kurtz, Queens
Speaker—John L. Clowe, Schenectady
Vice-Speaker—Charles N. Aswad, Broome

Councilors
Term Expires 1985
James H. Cosgriff, Jr., Erie
Samuel M. Gelfand, Nassau
Richard A. Hughes, Warren
Antonio F. LaSorte, Broome

Term Expires 1986
Robert A. Mayers, Westchester
Thomas D. Pemrick, Rensselaer
Ralph E. Schlossman, Queens
Stanley A. Steckler, Suffolk

Term Expires 1987
David M. Benford, New York
Duane M. Cady, Onondaga
James M. Flanagan, Wayne
M. Theodore Tanenhaus, Kings

Trustees
Carl Goldmark, Jr., New York, *Chairman*
G. Rehmi Denton, Albany
Ralph M. Schwartz, Kings
Edgar P. Berry, New York
Richard D. Eberle, Onondaga
Joseph F. Shanaphy, Richmond
Victor J. Tofany, Monroe

Past Presidents
1959–1960—Henry I. Fineberg, Suffolk
1960–1961—Norman S. Moore, Tompkins
1961–1962—John M. Galbraith, Nassau
1966–1967—James M. Blake, Schenectady
1969–1970—Walter T. Heldmann, Richmond
1970–1971—Walter Scott Walls, Erie
1971–1972—George Himler, New York
1972–1973—Edward Siegel, Clinton
1974–1975—Lynn R. Callin, Monroe
1975–1976—Ralph S. Emerson, Nassau
1976–1977—George L. Collins, Jr., Erie
1977–1978—Carl Goldmark, Jr., New York
1978–1979—George T. C. Way, Dutchess
1979–1980—G. Rehmi Denton, Albany
1980–1981—Ralph M. Schwartz, Kings
1982–1983—Richard D. Eberle, Onondaga
1983–1984—Bernard J. Pisani, New York

Past President of the American Medical Association
Gerald D. Dorman, New York

Commissioner, New York State Department of Health

District Delegates
(*Elected Delegates of District Branches*)
First—Roger D. Billig, Richmond
Second—Melvyn Bruckstein, Suffolk
Third—Thomas W. Greenlees, Schoharie
Fourth—Michael J. Lynch, Washington
Fifth—Frederic A. Stone, Jefferson
Sixth—Vincent I. Maddi, Broome
Seventh—John E. Horn, Steuben
Eighth—Kenneth J. Clark, Orleans
Ninth—Joel E. Mandel, Rockland

Specialty Society Delegates
New York State Society of Anesthesiologists—Edward C. Sinnott, Nassau
New York State Society of Dermatology—Franklin S. Glickman, Kings
New York Chapter–Emergency Physicians—Ralph Altman, New York
New York State Academy of Family Physicians—James R. Nunn, Erie
New York State Society of Internal Medicine—Robert M. Kohn, Erie
New York State Neurological Society—Russel H. Patterson, Jr., New York
American College of Obstetricians and Gynecologists—Leonard S. Weiss, Orange
New York Occupational Medical Association, Inc.—Thomas J. Doyle, Queens
New York State Ophthalmological Society—Francis J. Gilroy, Broome
New York State Society of Orthopaedic Surgeons—John D. States, Monroe
New York State Society of Otolaryngology—Head and Neck Surgery—Fouad B. Michael, Nassau
New York State Society of Pathologists—Herbert Lansky, Erie
American Academy of Pediatrics–District II—David Annunziato, Nassau
New York Society of Physical Medicine and Rehabilitation—David Ernstoff, Queens
New York Regional Society of Plastic and Reconstructive Surgery—

New York State Psychiatric Association—Harold E. Berson, Kings
New York State Chapter-American College of Radiology—Victor A. Panaro, Erie
New York State Society of Surgeons—Theodore C. Max, Oneida
New York State Urological Society—Lawrence Ravich, Nassau

Delegates from Component County Medical Societies

Albany (*4*)
John W. Abbuhl, Albany
William O'Dwyer, Latham
Nathan P. Reed, Albany
Anthony P. Tartaglia, Albany

Allegany (*1*)
Muhammad Akmal Khan, Wellsville

Bronx (*10*)
John P. Albanese, Bronx
Marcos A. Charles, Bronx
Dattatraya G. Lanjewar, Pelham Manor
Michael N. Lavacca, Bronx
Charles Laverdi, Bronx
Hugo M. Morales, Bronx
Govindappa Rangaiah, Scarsdale
Howard S. Schwartz, Bronx
Tullio F. Tartaglia, Bronx
Juana Toporovsky, Bronx

Broome (*4*)
Louis W. Giordano, Endicott
Theodore R. Poritz, Binghamton
Jeffrey A. Ribner, Binghamton
Irwin J. Rosenberg, Endicott

Cattaraugus (*1*)
Leo David Moss, Olean

Cayuga (*1*)
Richard J. Freeman, Auburn

Chautauqua (*2*)
James G. Dahlie, Jamestown
Charles R. Sinatra, Jamestown

Chemung (*2*)
Donald L. Brooks, Jr., Elmira
Gerald P. Schneider, Elmira

Chenango (*1*)
Norman C. Lyster, Jr., Norwich

Clinton (*1*)
Robert T. Potter, Plattsburgh

Columbia (*1*)
Rosewell D. Shaw, Stottville

Cortland (*1*)
Patrick J. Hayes, Cortland

Delaware (*1*)
Gary Preiser, Walton

Dutchess (*4*)
Eric R. Brocks, Fishkill
Stephen R. Chernay, Fishkill
Arthur W. Menken, Poughkeepsie
Seymour R. Stall, Poughkeepsie

Erie (*9*)
Victorino Anllo, Amherst
Robert A. Caputi, Buffalo
Anthony J. Federico, Buffalo
Allen L. Lesswing, Buffalo
Milford C. Maloney, Buffalo
Leo E. Manning, Amherst
James F. Phillips, Jr., Buffalo
Joseph A. Prezio, Buffalo

Essex (*1*)
Erwin Lange, Mineville

Franklin (*1*)
Alfred A. Hartmann, Sr., Malone

Fulton (*1*)
Armand J. D'Errico, Gloversville

Genesee (*1*)
Alfred L. George, Batavia

Greene (*1*)
Shanmuga S. G. Sundaram, Cairo

Herkimer (*1*)
Krishna K. Vadlamudi, Ilion

Jefferson (*2*)
William Bruce Carter, Watertown
Eugene Michael Renzi, Watertown

Kings (*19*)
Leslie L. Alexander, Brooklyn
Grace A. Altenau, Brooklyn
Lawrence Ames, Brooklyn
Norman S. Blackman, Brooklyn
Randall D. Bloomfield, Brooklyn
Joseph R. Brennan, Brooklyn
Matthew Brody, Brooklyn
Vernal G. Cave, Brooklyn
Duncan W. Clark, Brooklyn
Paul Finkelstein, Brooklyn
Stanley I. Fishman, Brooklyn
Robert E. Gordon, Brooklyn
Ladislav P. Hinterbuchner, Brooklyn
A. W. Martin Marino, Jr., Brooklyn
Martin Markowitz, Brooklyn
Bentley D. Merrim, Brooklyn
Hubert S. Pearlman, Brooklyn

Giovanna Rasile, Brooklyn
Leo J. Swirsky, Brooklyn

Lewis (*1*)
John C. Herrman, Lowville

Livingston (*1*)
Sheppard Arluck, Geneseo

Madison (*1*)
Leonard A. Argentine, Oneida

Monroe (*7*)
William A. Dolan, Rochester
Nathaniel J. Hurst, Rochester
William A. Kern, Jr., Rochester
Vito P. Laglia, Rochester
Mario B. LoMonaco, Rochester
Joyce M. McChesney, Rochester
Charles R. Salamone, Rochester

Montgomery (*1*)
Feori F. Pipito, Amsterdam

Nassau (*11*)
Lee S. Binder, Valley Stream
Ray S. Crampton, Syosset
Morton Goldfarb, Massapequa Park
Paul C. Lehmuller, Levittown
Arnold D. Lurie Baldwin
Alfred W. Marks, Rockville Centre
Sidney Mishkin, Fort Salonga
Felix A. Monaco, Rockville Centre
John A. Ostuni, Freeport
Patricia G. Squillace, Rockville Centre
Jerome Zwanger, Massapequa

New York (*23*)
Arnold L. Bachman, New York City
Melvin H. Becker, Manhasset
Robert S. Bernstein, New York City
Irwin J. Cohen, New York City
Hugh Clark Davidson, New York City
Wilbur J. Gould, New York City
George Hyams, New York City
Virginia Kanick, New York City
Vance Lauderdale, Jr., New York City
Samuel H. Madell, New York City
Aaron G. Meislin, New York City
George W. Melcher, Jr., New York City
Alfred Moldovan, New York City
Richard B. Nolan, New York City
Donna B. O'Hare, New York City
Stuart I. Orsher, New York City
Richard N. Pierson, Jr., New York City
William B. Rawls, Pleasant Valley
Isadore Rosenfeld, New York City
Albert M. Schwartz, New York City
Miriam Z. Seborer, New York City
Clifford L. Spingarn, New York City
Meyer Texon, New York City

Niagara (*3*)
Claus Michael Fichte, Niagara Falls
James J. Kropelin, Niagara Falls
Clark G. Triftshauser, Lewiston

Oneida (3)
Robert D. Brennan, Utica
Gerald C. Gant, New Hartford
Robert M. George, Utica

Onondaga (*6*)
Daniel L. Dombroski, Syracuse
Richard A. Konys, Syracuse
Arthur Lehrman, Syracuse
Carl E. Marlow, North Syracuse
John T. Prior, Syracuse
George P. Tilley, Syracuse

Ontario (*2*)
Thomas A. McCormick, Geneva
Kenneth T. Steadman, Geneva

Orange (*4*)
Stanley F. Brunn, Port Jervis
Stanley L. Grossman, Newburgh
Jack Manpel, New Windsor
Ansel R. Marks, Middletown

Orleans (*1*)
David B. L. Meza, III, Albion

Oswego (*1*)
James F. Grant, Oswego

Otsego (*1*)
Rodman D. Carter, Cooperstown

Putnam (*1*)
Hari P. Chakravorty, Mahopac

Queens (*16*)
Daniel Chansky, Forest Hills
John W. V. Cordice, Jr., St. Albans
Alexander M. De la Garza, Flushing
Robert M. Farrell, Flushing
Fred S. Fensterer, Glendale
Fred N. Flatau, Flushing
Irving G. Frohman, Rockaway Beach
Leo Arthur Green, Jackson Heights
Victor Guarneri, Bayside
Edith Gutman, Beechhurst
Joseph Klements, Bayside
Nicetas H. Kuo, Great Neck
Hilda Ratner, Beechhurst
Sheldon D. Sax, Whitestone
Ezra A. Wolff, Manhasset

Rensselaer (*2*)
William M. McCarty, Troy
William A. Whyland, Troy

Richmond (*4*)
B. Basil Hyman, Staten Island

Gerald J. Lustig, Staten Island
William A. Schwarz, Southbury, Connecticut
Robert J. Silich Staten Island

Rockland (4)
Robert J. Brenner, New City
Michael A. Kalvert, Bardonia
Roy H. Lieberman, Nanuet
Stanley H. Oransky, Bardonia

St. Lawrence (1)
Maurice J. Elder, Massena

Saratoga (1)

Schenectady (3)
Richard H. Lange, Schenectady
John M. Spring, Schenectady
Donald Wexler, Schenectady

Schoharie (1)
Frederick J. Michel, Cobleskill

Schuyler (1)

Seneca (1)
Donald W. Bidwell, Seneca Falls

Steuben (2)
Jack D. O'Neil, Corning
Wayne C. Templer, Corning

Suffolk (11)
William H. Bloom, Bay Shore
Stephen I. Braitman, Islip Terrace
Clive D. Caplan, Islip Terrace
Frank R. Collier, Port Jefferson
John G. Egner, West Islip
Robert E. Fear, Southampton
James A. Holleran, East Setauket
Elise H. Korman, Southampton
Phillip I. Levitan, Smithtown
Nathaniel B. Messinger, Islip Terrace
Melvin Shuter, Islip Terrace

Sullivan (1)
Seymour Cohen, Monticello

Tioga (1)

Tompkins (2)
Wendell L. Bryce, Ithaca
Hope Craig Perry, Ithaca

Ulster (2)
Joseph B. Basile, Kingston
Stephen L. Hermele, Kingston

Warren (2)
Larry J. Greenberg, Glens Falls
Raymond A. Maddocks, Glens Falls

Washington (1)
Arnold B. Wise, Cambridge

Wayne (1)
Norman R. Loomis, Ontario

Westchester (9)
Edwin I. Cleveland, Bronxville
Peter B. Farnsworth, Valhalla
Richard L. Fenton, Tarrytown
Armond V. Mascia, Tarrytown
Stanley M. Mendelowitz, Tarrytown
Richard L. Petrillo, Mount Vernon
John A. Ramsdell, White Plains
Thomas Dignan Rizzo, Bronxville
Michael Sierp, Yonkers

Wyoming (1)

Yates (1)
William Dean, Jr., Penn Yan

Medical School Delegates — 12
Albany Medical College—Samuel Steven Ciccio, Albany
Albert Einstein College of Medicine—Melvin Nat Zelefsky, Bronx
Columbia University—Linda D. Lewis, New York
Cornell University Medical College—Jack Richard, New York
Mount Sinai School of Medicine—David A. Dreiling, New York
New York Medical College—Michael L. Friedland, Westchester
New York University School of Medicine—Stanford Wessler, New York
State University of New York at Brooklyn—
State University of New York at Buffalo—
State University of New York at Stony Brook—Tamarath Knigin Yolles, Suffolk
State University of New York at Syracuse—George F. Reed, Onondaga
University of Rochester—Thomas E. Cardillo, Monroe

Medical Student Delegates — 3
Douglas M. Gordon, Syracuse
Joel Seth Greenwald, Bronx
Elizabeth Anne Jennison, Rochester

Resident Membership Delegate — 1

Hospital Medical Staff Delegate — 1

REFERENCE COMMITTEES

Reports of Officers

Anthony J. Federico, Erie, *Chairman*
Stanley F. Brunn, Orange
Michael L. Friedland, New York Medical College
Isadore Rosenfeld, New York
Edward C. Sinnott, NYS Society of Anesthesiologists

ANNUAL REPORTS REFERRED TO COMMITTEE:
- *President*
- *President-Elect*
- *Secretary*
- *Treasurer*
- *Board of Trustees*
- *Budget and Finance*
- *Executive Vice-President*
- *New York Delegation to the AMA*
- *District Branches*
- *Physicians' Home*
- *Long-Range Planning*
- *Résumé of Instructions from the 1984 House of Delegates*
  - *Ad Hoc Committee to Study the Feasibility of Relocating MSSNY Headquarters*
  - *Ad Hoc Committee on Physician/Patient Concerns*

Bylaws

Stanley A. Steckler, Suffolk, *Chairman*
William Bruce Carter, Jefferson
James R. Nunn, NYS Academy of Family Physicians
Hilda Ratner, Queens
Nathan P. Reed, Albany

ANNUAL REPORT REFERRED TO COMMITTEE:
- *Résumé of Instructions from the 1984 House of Delegates*

Governmental Affairs and Legal Matters

Sidney Mishkin, Nassau, *Chairman*
William A. Dolan, Monroe
B. Basil Hyman, Richmond
David B. L. Meza, III, Orleans
Robert T. Potter, Clinton

ANNUAL REPORTS REFERRED TO COMMITTEE:
- *Ethics*
- *Judicial Council*
- *Legislation*
- *Liaison Committee with HANYS*
- *New York State Association of the Professions*
- *Professional Medical Liability Insurance and Defense Board*
- *Résumé of Instructions from the 1984 House of Delegates*
  - *Special Ad Hoc Committee to Evaluate Nurse Practitioner Legislation*

Medical Services

Alfred W. Marks, Nassau, *Chairman*
John P. Albanese, The Bronx
Donald L. Brooks, Jr., Chemung
Hugh Clark Davidson, New York
William A. Kern, Jr., Monroe

ANNUAL REPORTS REFERRED TO COMMITTEE:
- *Alternative Health Care Delivery Systems*
- *Health Planning*
- *Hospital and Professional Relations*
- *Interspecialty*
- *Medicaid*
- *Medical Care Insurance*
- *Social Security Administration Disability Benefits Program, Committee to Monitor and Provide Input Into*
- *Workers' Compensation and Occupational Health*
- *Résumé of Instructions from the 1984 House of Delegates*
  - *Ad Hoc Committee to Review Applications from Specialty Societies Seeking Representation in the MSSNY House of Delegates*

Membership Benefits, Publications, and Miscellaneous

Stephen R. Chernay, Dutchess, *Chairman*
Randall D. Bloomfield, Kings
John E. Horn, Seventh District Branch
James J. Kropelin, Niagara
Phillip I. Levitan, Suffolk

ANNUAL REPORTS REFERRED TO COMMITTEE:
- *Administrative Committee for the MSSNY Pension Program*
- *American Association of Medical Assistants, New York State Society, Inc., Advisory Committee to*
- *Foreign Medical Graduates*
- *General Insurance*
- *Library, Archives, and Publications*
- *Membership Benefits*
- *Résumé of Instructions from the 1984 House of Delegates*

Public Health and Education

Stuart I. Orsher, New York, *Chairman*
David Annunziato, American Academy of Pediatrics – District II
Nicetas H. Kuo, Queens
Ansel R. Marks, Orange
John T. Prior, Onondaga

ANNUAL REPORTS REFERRED TO COMMITTEE:
- *Accident and Injury Prevention*
- *Aging and Nursing Homes*
- *Alcoholism*
- *Cancer*
- *Cardiovascular Disease*
- *Child Abuse*

Drug Abuse
Emergency Medical Services
Environmental Quality
Home Health Care and Hospice
Maternal and Child Health
Mental Health
Physical Medicine and Rehabilitation
Physicians' Committee
Preventive Medicine
School Health and Sports Medicine

Continuing Medical Education
Forensic Medicine
Information Technology in Medicine
Medical School Relations
Scientific Program
Résumé of Instructions from the 1984 House of Delegates

CONVENTION COMMITTEES

Credentials

William A. Schwarz, Richmond, *Chairman*
Stephen I. Braitman, Suffolk
A. W. Martin Marino, Kings
George W. Melcher, Jr., New York
Frederick J. Michel, Schoharie

Tellers

Leo E. Manning, Erie, *Chairman*
John C. Herrman, Lewis
Richard H. Lange, Schenectady
Armand V. Mascia, Westchester
Howard S. Schwartz, The Bronx

Sergeant at Arms

Stanley H. Oransky, Rockland, *Chairman*
Arnold L. Bachman, New York
Joseph R. Brennan, Kings
Louis W. Giordano, Broome
John A. Ostuni, Nassau

Rules and Order of Business

Robert M. George, Oneida, *Chairman*
Rodman, D. Carter, Otsego
Samuel C. Ciccio, Albany Medical College of Union University
Richard L. Petrillo, Westchester
Richard N. Pierson, Jr., New York

NOMINATING COMMITTEE

To the House of Delegates, Ladies and Gentlemen:

In accordance with Article XI, Section 2 of the Bylaws of the Medical Society of the State of New York, the Nominating Committee met at the Society's headquarters, 420 Lakeville Road, Lake Success, New York, on Wednesday, January 23, 1985, at 10:30 a.m.

Members of the committee present were:
Member-at-Large—**Bernard J. Pisani, M.D.,** New York, *Chairman*
First District Branch—**Edgar P. Berry, M.D.,** New York
Second District Branch—**Samuel M. Gelfand, M.D.,** Nassau
Third District Branch—**Stephen L. Hermele, M.D.,** Ulster
Fourth District Branch—**Robert A. Reid, M.D.,** Warren
Fifth District Branch—**George Lim, M.D.,** Oneida
Sixth District Branch—**Paul M. DeLuca, M.D.,** Broome
Seventh District Branch—**Victor J. Tofany, M.D.,** Monroe
Ninth District Branch—**John A. Ramsdell, M.D.,** Westchester
Member-at-Large—**Ralph M. Schwartz, M.D.,** Kings

Excused was: James J. Kropelin, M.D., Eighth District Branch. Allison B. Landolt, M.D., was designated to represent the interests of that District.

After careful consideration of the recommendations submitted by the district branches and the county medical societies in response to a memorandum, dated October 29, 1984, and nominations from the floor, your committee respectively nominated the following candidates for election on June 9, 1985:

President—**Daniel F. O'Keeffe, M.D.,** Warren
President-Elect—**John A. Finkbeiner, M.D.,** New York
Vice-President—**Samuel M. Gelfand, M.D.,** *Nassau*
Secretary—**John H. Carter, M.D.,** Albany
Assistant Secretary—**George Lim, M.D.,** Oneida
Treasurer—**Charles D. Sherman, Jr., M.D.,** Monroe
Assistant Treasurer—**Morton Kurtz, M.D.,** Queens
Speaker—**John L. Clowe, M.D.,** Schenectady
Vice-Speaker—**Charles N. Aswad, M.D.,** Broome
Trustees (*one for five years*)—**Bernard J. Pisani, M.D.,** New York
Councilors (*four for three years*)—**James H. Cosgriff, Jr., M.D.,** Erie
Richard A. Hughes, M.D., Warren
Antonio F. LaSorte, M.D., Broome
Sidney Mishkin, M.D., Nassau
Councilors (*two for one year*)—**Thomas D. Pemrick, M.D.,** Rensselaer
Ralph E. Schlossman, M.D., Queens

Delegates to the American Medical Association for two years commencing January 1, 1986:
David Benford, M.D., New York
John H. Carter, M.D., Albany
Duncan W. Clark, M.D., Kings
Irwin J. Cohen, M.D., New York
James H. Cosgriff, Jr., M.D., Erie
G. Rehmi Denton, M.D., Albany
John A. Finkbeiner, M.D., New York
James M. Flanagan, M.D., Wayne
Samel M. Gelfand, M.D., Nassau
Robert M. George, M.D., Oneida
Morton Kurtz, M.D., Queens
Allison B. Landolt, M.D., Westchester
Antonio F. LaSorte, M.D., Broome
George Lim, M.D., Oneida
Daniel F. O'Keeffe, M.D., Warren
Stuart Orsher, M.D., New York
Bernard J. Pisani, M.D., New York
***Elizabeth A. Prezio,** Onondaga
Charles D. Sherman, Jr., M.D., Monroe
Seymour R. Stall, M.D., Dutchess
M. Theodore Tanenhaus, M.D., Kings
Victor J. Tofany, M.D., Monroe

The eleven nominees receiving the largest number of votes will be delegates; the second eleven will be alternates.

Bernard J. Pisani, M.D., Chairman

---

* Medical Student

HOUSE COMMITTEE ON BYLAWS

To the House of Delegates, Ladies and Gentlemen:

The members of the House Committee on Bylaws are as follows:

George Lim, M.D., *Chairman*	Oneida
Edgar P. Berry, M.D.	New York
Robert Kohn, M.D.	Erie
Sidney Mishkin, M.D.	Nassau
Daniel F. O'Keeffe, M.D.	Warren
Donna B. O'Hare, M.D.	New York
Seymour Stall, M.D.	Dutchess
Stanley A. Steckler, M.D.	Suffolk
Allison B. Landolt, M.D., President, ex officio	Westchester
John H. Carter, M.D., Secretary, ex officio	Albany
John L. Clowe, M.D., Speaker, ex officio	Schenectady
Milton Rosenberg, M.D., Executive Vice-President, ex officio	Suffolk
Donald R. Moy, J.D., General Counsel, ex officio	Nassau

A meeting of the Reference Committee on Bylaws was held at the Hilton Hotel, New York, NY, on October 15, 1984. The Reference Committee was appointed to receive input from delegates as to their thoughts on the resolutions referred to the House Committee on Bylaws. The input received from the meeting of the Reference Committee on Bylaws proved to be of great assistance to the House Committee on Bylaws when they met on January 9, 1985.

In all, twelve resolutions asking for amendments to the Bylaws were referred to the committee for its consideration.

The following recommendations were made with respect to the resolutions referred to this committee:

Resolution 84-1, Certain Officers of the Medical Society of the State of New York Serving on the Board of Directors of the Medical Liability Mutual Insurance Company

Introduced by Lawrence Ravich, M.D., as an Individual

Whereas, The medical liability insurance crisis is a continuing problem; and

Whereas, The Medical Liability Mutual Insurance Company was established by members of the Medical Society of the State of New York as a separate corporation with its own Board of Directors; and

Whereas, Decisions made by the MLMIC Board of Directors are made in the best interests of that corporation even if these interests are not consonant with the best interests of the members of the MSSNY; and

Whereas, The President, President-Elect, and Vice-President of the MSSNY may also be members of the Board of Directors of MLMIC; and

Whereas, This situation constitutes a conflict of interest in the instance where on the one hand these officers of MSSNY, as directors of MLMIC, vote with good conscience and with their fiduciary responsibility for increases in the medical liability insurance premiums and on the other hand, as spokesmen for and as leaders of MSSNY, must also decry and fight for a decrease rather than for an increase in medical liability insurance premiums; and

Whereas, Despite the fact that these officers of MSSNY are acting in good conscience in both instances, their position as spokesmen for MSSNY physician interests is diffused and confused in the minds of the membership of MSSNY and in the eyes of the general public; therefore be it

Resolved, That Article V of the Bylaws of the Medical Society of the State of New York be amended by inserting the following paragraph after paragraph two of Article V: (additions italicized)

It is the policy of the Medical Society of the State of New York that its president, president-elect, or vice-president may not concurrently serve as an officer or a member of the Board of Directors of the Medical Liability Mutual Insurance Company. If the president, president-elect or vice-president of the Medical Society of the State of New York is also an officer or a member of the Board of Directors of MLMIC, he must resign from the position on the MLMIC or he shall be presumed to have resigned from his office in the Medical Society of the State of New York.

In the opinion of the committee, it is essential to have representation from the Medical Society of the State of New York on the Medical Liability Mutual Insurance Company Board in order to protect the interests of our members. The committee also felt that there is no true conflict of interest for the officers of the MSSNY who serve on the Board of Directors of MLMIC that would possibly hinder their acting in a responsible manner in both positions.

The Committee unanimously recommends that Resolution 84-1 **NOT BE ADOPTED.**

Resolution 84-2, Councilors Representing the Student Membership and the Resident Membership

Introduced by Daniel F. O'Keeffe, M.D., as an Individual

Whereas, It is important to involve medical students and graduates of medical schools who are in continuous residency training in organized medicine; and

Whereas, The future of organized medicine requires the active participation of the younger physicians; and

Whereas, The American Medical Association has set the lead for involving student and resident members in the decision-making process by amending its Bylaws to provide for a resident member and a non-voting student member on the AMA Board of Trustees; and

Whereas, The Medical Society has recognized this need by providing for student and resident membership in the Medical Society of the State of New York; and

Whereas, The interest of medical students and residents in the Medical Society can be greatly increased by providing for their representation in the Council of the MSSNY; therefore be it

Resolved, That the Bylaws of the Medical Society of the State of New York be amended to provide for a Councilor representing the student membership and a Councilor representing the resident membership on the MSSNY Council; and be it further

Resolved, That Article IV, Section 1, paragraph 1 of the MSSNY Bylaws be amended by deleting the number "12" and by inserting the number "14" in its place. Article IV, Section 1, paragraph 1 would then read: (additions italicized, deletions in brackets)

"The Council shall be composed of the president, president-elect, vice-president, immediate past president, secretary, assistant secretary, treasurer, assistant treasurer, speaker, vice-speaker, chairman of the Board of Trustees and [12] *14* councilors elected by the House of Delegates."

and be it further

Resolved, That Article IV, Section 1, paragraph 3 of the Bylaws be amended by adding a new sentence after the first sentence, to read:

"Two councilors, one councilor representing the student membership and one councilor representing the resident membership to the Medical Society of the State of New York, shall be elected every third year by the House of Delegates, each for a term of three years."

Article IV, Section 1, paragraph 3 would then read: (additions italicized)

"Four councilors shall be elected annually by the House of Delegates, each for a term of three years. *Two councilors, one councilor representing the student membership and one councilor representing the resident membership to the Medical Society of the State of New York, shall be elected every third year by the House of Delegates, each for a term of three years.* In the event of a vacancy, a councilor shall be elected by the Council to serve until the next meeting of the House of Delegates, at which time the House of Delegates shall elect a councilor to fill the unexpired term."

and be it further

Resolved, That Article IV, Section 1, paragraph 6 of the Bylaws be amended by adding a new sentence after the last sentence to read:

"The councilor representing the student membership must be a student member in the Medical Society of the State of New York; the councilor representing the resident membership must be a resident member of the Medical Society of the State of New York."

Article IV, Section 1, paragraph 6 of the Bylaws would then read: (additions italicized)

"Councilors shall be assigned to specific county societies as liaison for the Council in accordance with the provisions of Article V, Section 2. Councilors shall be required to disseminate information of Council activity as well as returning information to the Council, and shall report regularly to the Council on their activity. *The councilor representing the student membership must be a student member in the Medical Society of the State of New York; the councilor representing the resident membership must be a resident member of the Medical Society of the State of New York."*

Daniel F. O'Keeffe, M.D., who introduced the resolution, submitted an additional "Resolve" to the Reference Committee on Bylaws, as follows:

"**Resolved,** That using the AMA as a guide, MSSNY allow the resident membership to have voice and vote and that the student membership have voice and vote, to be determined by the Council by issue."

After considerable discussion, the House Committee on Bylaws voted separately on the question of residents and students sitting on the Council. Their decisions, which were in agreement with the recommendations of the Reference Committee on Bylaws, are as follows:

RESIDENTS	Seat on Council	– Yes
	Voice and Vote	– Yes
	Term of Office	– 1 year. May be re-elected for a total of three years.
STUDENTS	Seat on Council	– Yes
	Voice	– Yes
	Vote	– No
	Term of Office	– 1 year. May be re-elected for a total of three years.

In accordance with the above recommendation, the committee recommends that Article IV, Section 1, paragraph 2 of the Bylaws be amended by the addition of the following sentence: "The councilor from the resident membership shall attend all meetings of the Council with voice and with vote. The councilor from the student membership shall attend all meetings of the Council with voice but without vote." Article IV, Section 1, paragraph 2 shall now read: (additions italicized)

"The trustees, the executive vice-president, the deputy executive vice-president, and the general counsel of the Medical Society of the State of New York shall attend all meetings of the Council with voice but without vote. *The councilor from the resident membership shall attend all meetings of the Council with voice and with vote. The councilor from the student membership shall attend all meetings of the Council with voice but without vote."*

The committee amended the third "Resolve" by deleting the phrase, "shall be elected every third year by the House of Delegates, each for a term of three years," and inserted the phrase in its place to read, "shall be elected every year by the House of Delegates, each for a term of one year." Article IV, Section 1, paragraph 3 of the Bylaws would then read: (additions italicized)

"Four councilors shall be elected annually by the House of Delegates, each for a term of three years. *Two councilors, one councilor representing the student membership and one councilor representing the resident membership to the Medical Society of the State of New York, shall be elected every year by the House of Delegates, each for a term of one year. Article IV, Section 1, paragraph 4 is not applicable to the term of office of a resident or student councilor.* In the event of a vacancy, a councilor shall be elected by the Council to serve until the next meeting of the House of Delegates, at which time the House of Delegates shall elect a councilor to fill the unexpired term."

The committee also amended the fourth "Resolve" with the addition of the phrase, "other than the councilor representing the student membership and the councilor representing the resident membership" to be incorporated in the first sentence of Article IV, Section 1, paragraph 6 of the Bylaws. Article IV, Section 1, paragraph 6 would then read: (additions italicized)

> "Councilors, *other than the councilor representing the student membership and the councilor representing the resident membership,* shall be assigned to specific county societies as liaison for the Council in accordance with the provisions of Article V, Section 2. Councilors shall be required to disseminate information of Council activity as well as returning information to the Council, and shall report regularly to the Council on their activity. *The councilor representing the student membership must be a student member in the Medical Society of the State of New York; the councilor representing the resident membership must be a resident member of the Medical Society of the State of New York."*

The Committee unanimously recommends **ADOPTION** of Resolution 84-2, as amended.

Resolution 84-11, Deletion of Reinstatement Penalty

Introduced by Milton Rosenberg, M.D., as an Individual

Whereas, Physicians dropped from membership for nonpayment of dues currently are required to, in effect, pay two year's dues to be reinstated; and

Whereas, This requirement does not apply to members who resign; and

Whereas, Few members are aware of this requirement; and

Whereas, There is little practical difference to the MSSNY between a resignation and nonpayment of dues; and

Whereas, This requirement serves as a barrier to reinstatement and thus actually imposes a financial penalty on the MSSNY; therefore be it

Resolved, That Article II, Section 2, paragraph 14 of the Bylaws of the Medical Society of the State of New York be amended as follows: (additions italicized, deletions in brackets)

> Medical Society of the State of New York dues of a member reinstated after October 1 shall be credited to the succeeding year, all rights and privileges of membership, however [dating] *shall date* from the time of reinstatement. [but no member dropped for nonpayment of dues and assessments shall be reinstated until he has, in addition, paid his dues and assessments for the year in which he was dropped,];

and be it further

Resolved, That Article XV, paragraph 8, of MSSNY's Bylaws be amended as follows: (deletions in brackets)

> Dues and State Society assessments of a member elected or reinstated after October 1 shall be credited to the succeeding year. [but no member dropped for nonpayment of dues and assessments shall be reinstated until he has, in addition, paid his dues and assessments for the year in which he was dropped.]

The committee agreed with the spirit of the resolution since the deletion of paragraph 14 could encourage former members who were dropped for non-payment of dues and assessments to rejoin. However, the Bylaws Committee is concerned that the deletion of paragraph 14 could have unintended adverse consequences. The committee believes that the provisions in paragraph 14 should be waived for a *finite* period. Therefore, the committee recommends that the first resolve of Resolution 84-11 be amended as follows: (additions italicized)

Article II, Section 2, paragraph 14 to read:

> Medical Society of the State of New York dues of a member reinstated after October 1 shall be credited to the succeeding year, all rights and privileges of membership, however dating from the time of reinstatement; but no member dropped for nonpayment of dues and assessments shall be reinstated until he has, in addition, paid his dues and assessments for the year in which he was dropped.

and by the addition of new paragraph 15 to read:

> *"The provision in Article II, Section 2, paragraph 14 that no member dropped for non-payment of dues and assessments shall be reinstated until he has, in addition, paid his dues and assessments for the year in which he was dropped, shall not be in effect for the period beginning June 6, 1985 through April 1, 1986."*

The committee further recommends that the second resolve of Resolution 84-11 be amended as follows: (additions italicized)

Article XV, paragraph 8 to read:

> Dues and State Society assessments of a member elected or reinstated after October 1 shall be credited to the succeeding year; but no member dropped for nonpayment of dues and assessments shall be reinstated until he has, in addition, paid his dues and assessments for the year in which he was dropped.

and by the addition of new paragraph 9 to read:

> *"The provision in Article XV, paragraph 8 that no member dropped for non-payment of dues and assessments shall be reinstated until he has, in addition, paid his dues and assessments for the year in which he was dropped, shall not be in effect for the period beginning June 6, 1985 through April 1, 1986."*

The Committee recommends that Resolution 84-11 be **ADOPTED,** as amended. The vote was 5 in favor, 1 against.

Resolution 84-16, Equalization of Special Dues Status to All New Members

Introduced by New York County Medical Society

Whereas, Article XV of the Bylaws of the Medical Society of the State of New York grants special dues status to active members for the first two years of solo practice; and

Whereas, Many younger physicians today do not practice as solo practitioners but practice in other types of practice; therefore be it

Resolved, That Article XV of the Bylaws of the Medical Society of the State of New York be amended to equalize treatment of all new members regardless of the type of practice by eliminating the word solo, thus granting special dues status for all active members during the first two years of practice. Article XV, paragraph 3 would then read: (additions italicized, deletions in brackets)

"The dues and assessments of those active members who have been graduated from medical or osteopathic college not more than five calendar years, not counting active service in the armed forces of the United States or in the United States Public Health Service, shall be one third the amount levied on all other active members. The dues and assessments of those active members who have not completed their continuous residency training shall be one tenth the amount levied on all other active members. In no case may an active physician continue to pay such reduced dues more than seven years after the date of graduation from medical or osteopathic school. The dues and assessments of [those] *all* active members entering [solo] practice for the first time, who are not otherwise eligible for reduced dues as set forth in prior provisions in this paragraph, shall be one half the amount levied on all other active members, for the first two years of [solo] practice; full dues shall be paid after the first two years of [solo] practice. Once an active member pays full dues, he shall no longer qualify for half-dues status."

The committee recognizes that differentiating between physicians in the first two years of practice is arbitrary, and therefore, no distinction should be made between those in private and group practice.

The Committee unanimously recommends **ADOPTION** of Resolution 84-16.

Resolution 84-21, Additional Councilor—Ninth District Branch

Introduced by Ninth District Branch

Whereas, The physician membership in the Ninth District Branch has exceeded 2800 for several years; and

Whereas, A comparable district branch in size and numbers (the Second District Branch) has had two councilors for many years; therefore be it

Resolved, That the Bylaws of the Medical Society of the State of New York be amended to increase the number of councilors for the Ninth District Branch to two; and be it further

Resolved, That councilor assignment within the district be such that Westchester will have one councilor and other counties within the district shall have one councilor; and be it further

Resolved, That Article IV, Section 1, paragraph 1 of the Bylaws be amended to read as follows (additions italicized, deletions in brackets)

"The Council shall be composed of the president, president-elect, vice-president, immediate past-president, secretary, assistant secretary, treasurer, speaker, vice-speaker, chairman of the Board of Trustees and [12] *13* councilors elected by the House of Delegates.";

and be it further

Resolved, That Article IV, Section 1, paragraph 3 be amended to read as follows (additions italicized, deletions in brackets)

"[Four] *five* councilors shall be elected by the House of Delegates [annually] *one year and four councilors shall be elected in the two subsequent years*, each for a term of three years. In the event of a vacancy, a councilor shall be elected by the Council to serve until the next meeting of the House of Delegates, at which time the House of Delegates shall elect a councilor to fill the unexpired term."

Fiscal Note: $400 to $500 per annum

and

Resolution 84-22, Council Representation

Introduced by Medical Society of the County of Westchester, Inc.

Whereas, There has been a large increase in membership in the Ninth District Branch (over 2800 physicians in the district are MSSNY members); and

Whereas, The Second District Branch with a similar number of members has two councilors; and

Whereas, Fairness dictates that equal representation be a matter of policy for the Medical Society of the State of New York; therefore be it

Resolved, That the Ninth District Branch be assigned two councilors, one to serve Westchester County and the other to serve the other four counties in the District; and be it further

Resolved, That the Bylaws of the Medical Society of the State of New York be amended to provide for another councilor for the Ninth District Branch; and be it further

Resolved, That Article IV, Section 1, paragraph 1 of the MSSNY Bylaws be amended by deleting the number "12" and by inserting the number "13" in its place. Article IV, Section 1, paragraph 1 would then read: (additions italicized, deletions in brackets)

"The Council shall be composed of the president, president-elect, vice-president, immediate past-president, secretary, assistant secretary, treasurer, assistant treasurer, speaker, vice-speaker, chairman of the Board of Trustees and [12] *13* councilors, elected by the House of Delegates.

and be it further

Resolved, That Article IV, Section 1, paragraph 3 be amended to read: (additions italicized, deletions in brackets)

"[Four] *five* councilors shall be elected by the House of Delegates [annually] *one year and four councilors shall be elected in the two subsequent years*, each for a term of three years. In the event of a vacancy, a councilor shall be elected by the Council to serve until the next meeting of the House of Delegates, at which time the House of Delegates shall elect a councilor to fill the unexpired term."

Fiscal Note: $400 — $500 per annum

The committee reviewed these two resolutions at the same time because of the similarity of content. After careful consideration, it was the opinion of the committee that this is an internal problem, amenable to local resolution, and, therefore, does not require any action by the House of Delegates.

The Committee recommends that Resolutions 84-21 and 84-22 **NOT BE ADOPTED.**

Resolution 84-27, Four-Year Membership Option for Freshman Medical Students Applying to the Medical Society of the State of New York

Introduced by Jefferson J. Katims, Robert Newborn and Elizabeth Anne Prezio, Medical Student Delegates

Whereas, Other student membership organizations, including the American Medical Student Association, offer four-year memberships to coincide with a student's enrollment in school; and

Whereas, Most medical students who join the Medical Society of the State of New York do so at the time of freshman orientation; and

Whereas, Allowing these students the option of prepaying for four years' membership could serve as a valuable pilot for multiple years' membership option among other categories of MSSNY members; therefore be it

Resolved, That the Bylaws of the Medical Society of the State of New York be amended to provide first-year medical students the option of applying for four-year membership as an alternative to the single-year membership currently available; and be it further

Resolved, That Article XV of the Bylaws of the Medical Society of the State of New York be amended by adding a new closing paragraph to read: (additions italicized)

"Notwithstanding the terms of Article II and XV of these Bylaws, the House of Delegates or the Council may establish schedules for multiple year dues options for student members;" and be it further

Resolved, That students who choose the four-year membership option be required to attest annually to the fact that they are still enrolled in an accredited school of medicine or osteopathy in the State of New York and therefore still qualified for student membership.

The members of the committee agree that this option is needed for membership recruitment and retention, and that the administration should have the ability to recruit new members.

The committee, however, wishes to amend the resolution by deleting the last "Resolve" and inserting a new "Resolve" to read as follows:

"**Resolved,** That a student who elects to pay dues on a multiple year option must annually attest that he qualifies for student membership in accordance with Article II, Section 1, paragraph 4 of these Bylaws."

The committee unanimously recommends **ADOPTION** of Resolution 84-27, as amended.

Resolution 84-43, Recision of CME Mandate

Introduced by The Council

Whereas, Since 1981, all active members of the Medical Society of the State of New York have been mandated to fulfill certain CME requirements in order to continue their membership; and

Whereas, The reporting of CME by MSSNY members in these past three years has demonstrated that they are continuing their medical educational needs; and

Whereas, The MSSNY has been in the forefront in providing high quality CME programs; and

Whereas, Educators, practitioners and legislators have recognized the fallacy of trying to measure physician competency by mandated continuing medical education; therefore be it

Resolved, That the Medical Society of the State of New York continue to support and encourage voluntary continuing medical education; and be it further

Resolved, That the MSSNY rescind its mandate requiring CME for continuing membership; and be it further

Resolved, That the Bylaws of MSSNY be amended to effectuate this change by deleting Article II, Section 3 of the Bylaws which reads as follows:

SECTION 3. CONTINUING MEDICAL EDUCATION

An active member who fails to fulfill the requirements for continuing medical education established by the Council of the Medical Society of the State of New York shall not be in good standing. This section shall become effective on March 1, 1981.

An active member as defined in Article II, Section 1, who is found not to be in good standing as of March 1, 1981, or March 1 of any subsequent year, for failing to comply with paragraph 1 of this Section shall receive reasonable notice that he must fulfill continuing medical education requirements by the last day of December of the year in which he is found not to be in good standing. An active member who does not fulfill continuing medical education requirements by the last day of December of the year in which is is found not to be in good standing shall automatically cease to be a member of the component county medical society and the State Society, unless, in the view of the Council, specific mitigating circumstances exist that the physicians could not complete the CME requirements.

The House Comittee on Bylaws unanimously agrees that continuing medical education should not be mandated, and, therefore, believes that Article II, Section 3 of the MSSNY Bylaws should be deleted. The House Committee on Bylaws is concerned, however, that the deletion of Article II, Section 3 may be mistakenly interpreted by some as a repudiation of continuing medical education. It should be clear that the discontinuance of mandated participation in continuing medical education is not a repudiation of continuing medical education itself.

The House Committee on Bylaws, therefore, recommends that the Medical Society, through the appropriate committee, adopt a position statement encouraging physicians to participate in continuing medical education activities.

Accordingly, the House Committee on Bylaws unanimously recommends that Resolution 84-43 be **ADOPTED.**

Resolution 84-50, Prorated Dues Payment

Introduced by Milton Rosenberg, M.D., as an Individual

Whereas, It is in the best interests of the Medical Society of the State of New York to attract new members; and

Whereas, Some physicians may be hesitant to pay full dues for only a half-year of membership; and

Whereas, Rhode Island is the only other state medical society that does not provide for half-year dues payment; therefore be it

Resolved, That the Bylaws of the Medical Society of the State of New York be amended to provide for dues payment for membership on a prorated basis for new members who have never previously been members of the Medical Society; and be it further

Resolved, That Article XV of the Bylaws of the Medical Society of the State of New York be amended by adding a new paragraph 10 to read: (additions italicized)

"Notwithstanding the terms of Articles II and XV of these Bylaws, the House of Delegates or the Council may establish schedules for the prorated payment of dues for new members who have never previously been members of the Medical Society of the State of New York. Such prorated dues payments established by the House of Delegates or the Council may only be exercised by the new members in the year that they apply for membership."

In the opinion of the committee, approval of this resolution would be a means of increasing new membership in the MSSNY, and would also bring additional revenue to the State Society.

The Committee, therefore, recommends **ADOPTION** of Resolution 84-50.

Resolution 84-60, Actions That May be Taken Without Meetings

Introduced by Milton Rosenberg, M.D., as an Individual

Whereas, Subdivisions (b) and (c) of Section 708 of the Not-for-Profit Corporation Law provide:

"(b) Unless otherwise restricted by the certificate of incorporation or the by-laws, any action required or permitted to be taken by the board or any committee thereof may be taken without a meeting if all members of the board or the committee consent in writing to the adoption of a resolution authorizing the action. The resolution and the written consents thereto by the members of the board or committee shall be filed with the minutes of the proceedings of the board or committee.

(c) When authorized by the certificate of incorporation or the by-laws, any one or more members of the board or any committee thereof may participate in a meeting of such board or committee by means of a conference telephone or similar communications equipment allowing all persons participating in the meeting to hear each other at the same time. Participation by such means shall constitute presence in person at a meeting."; and

Whereas, The Medical Society of the State of New York is governed by the Not-for-Profit Corporation Law; and

Whereas, It is not always possible for the Council, the Board of Trustees or any committee of the House of Delegates, the Council, or the Board of Trustees to meet; and

Whereas, Circumstances may require the Council, the Board of Trustees or any committee of the House of Delegates, the Council or the Board of Trustees to take action without a meeting or take action by telephone conference; therefore be it

Resolved, That the Bylaws of the MSSNY be amended to enable the Council, the Board of Trustees or any committee of the House of Delegates, Council or Board of Trustees, to take action without meetings if all members of the Council, Board of Trustees or the committee consent in writing to the adoption of a resolution authorizing the action; and to enable the Council, the Board of Trustees or any committee of the House of Delegates, Council or Board of Trustees to participate in meetings by conference telephone; and be it further

Resolved, That the Bylaws of the MSSNY be amended by adding a new Article XIII-A to read: (additions italicized)

ARTICLE XIII-A — Actions that May be Taken Without Meetings

"Any action required or permitted to be taken by the members of the Council, the Board of Trustees or any committee of the House of Delegates, the Council, or the Board of Trustees, may be taken without a meeting if all the members of the Council, the Board of Trustees or the committee shall individually or collectively consent in writing to the adoption of a resolution authorizing the action. The resolution and the written consents thereto by the members of the Council, Board of Trustees or committee, shall be filed with the minutes of the proceedings of the Council, Board of Trustees or the committee.

Any one or more members of the Council, the Board of Trustees or any committee of the House of Delegates, the Council or the Board of Trustees may participate in a meeting of such Council, Board of Trustees or committee by means of a conference telephone or similar communications equipment allowing all persons participating in the meeting to hear each other at the same time. Participation by such means shall constitute presence in person at the meeting."

The authority of the board, or any committee thereof, of a not-for-profit corporation to take action without a meeting, is provided by section 708 of the Not-for-Profit Corporation Law. According to subdivision (c) of section 708, a meeting by means of a conference telephone or similar communications equipment may only be had when it is authorized by the bylaws of the corporation.

The House Committee on Bylaws realizes that, as a practical necessity, Resolution 84-60 must be adopted so that the Medical Society will have the means of responding to emergency situations that require immediate action or resolution. It is realized that the adoption of Resolution 84-60 does not mean that meetings by means of the conference telephone will substitute or replace the regular meetings of the Council, Board of Trustees, or any committee of the Medical Society. The adoption of Resolution 84-60, however, will enable the Medical Society to conduct meetings by the means of the conference telephone when the need exists.

Accordingly, the House Committee on Bylaws unanimously recommends that Resolution 84-60 be **ADOPTED.**

Resolution 84-62, Consultation

Introduced by Joyce McChesney, M.D., as an Individual

Whereas, Section 8.03 of the Current Opinions of the Judicial Council of the American Medical Association (1984) provides:

"Physicians should obtain consultation whenever they believe that it would be helpful in the care of the patient or when requested by the patient or the patient's representative. When a patient is referred to a consultant, the referring physician should provide a history of the case and such other information as the consultant may need and the consultant should advise the referring physician of the results of the consultant's examination and recommendations relating to the management of the case. A physician selected by a patient for the purpose of obtaining a second opinion on an elective procedure is not obligated to advise the patient's regular physician of the findings or recommendations."; and

Whereas, Section 4 of Chapter II of the Principles of Professional Conduct of the Medical Society of the State of New York provides:

"Doctors of medicine should request consultation when necessary or upon request of patient and/or family."; and

Whereas, The position adopted by the American Medical Association regarding consultation is more comprehensive than the position on consultation adopted in the Principles of Professional Conduct; and

Whereas, The position adopted by the AMA specifically recognizes the need for the consulting physician to confer with the referring physician, while the position in the current Principles of Professional Conduct omits any reference to such a need; therefore be it

Resolved, That Section 4 of Chapter II of the Principles of Professional Conduct be deleted and replaced by a new Section 4 to incorporate the position provided in the Current Opinions of AMA Judicial Council;

Section 4 of Chapter II would then read: (additions italicized; deletions in brackets)

[Doctors of medicine should request consultations when necessary or upon request of patient and/or family.] *Physicians should obtain consultation whenever they believe that it would be helpful in the care of the patient or when requested by the patient or the patient's representative. When a patient is referred to a consultant, the referring physician should provide a history of the case and such other information as the consultant may need and the consultant should advise the referring physician of the results of the consultant's examination and recommendations relating to the management of the case. A physician selected by a patient for the purpose of obtaining a second opinion on an elective procedure is not obligated to advise the patient's regular physician of the findings or recommendations.*

During the discussion of this resolution, it was pointed out that the American Medical Association will be discussing its position regarding "Consultation" and similar topics in the June 1985 meeting of its House of Delegates. It is recommended, therefore, that consideration of this resolution be postponed until the next session of the MSSNY House of Delegates.

Accordingly, therefore, the House Committee on Bylaws recommends that Resolution 84-62 be **POSTPONED** until the next meeting of the House of Delegates.

Resolution 84-65, AMA Officers, Trustees, and Speakers Members of the MSSNY House of Delegates

Introduced by Bernard J. Pisani, M.D. as an Individual

Wheres, Officers and speakers of the American Medical Association, as well as members of the AMA Board of Trustees, are encouraged to remain members of their respective State House of Delegates; and

Whereas, At times it may not be feasible for such a physician so honored by election by the AMA to maintain an elected position in the MSSNY House of Delegates or Council; therefore be it

Resolved, That the Bylaws of the Medical Society of the State of New York be amended so that all New York State physicians who are elected officers, trustees or speakers of the American Medical Association, by virtue of those offices become voting members of the House of Delegates of the Medical Society of the State of New York; and be it further

Resolved, That Article III, Section 1, paragraph 1 of the Bylaws of the Medical Society of the State of New York be amended as follows: (additions italicized, deletions in brackets)

The House of Delegates shall be composed of: (a) duly designated delegates from the component county medical societies; (b) officers of the Medical Society of the State of New York, councilors, and trustees; (c) a duly designated delegate from each district branch; (d) a duly designated delegate from each recognized specialty society; (e) duly designated delegates from the student membership; (f) the Commissioner of Health of the State of New York, or a deputy designated by him, provided that any representative shall be a member of the State Society; (g) past-presidents of the State Society and any past president of the American Medical Association, provided that individual is a member of the Medical Society of the State of New York who shall be members for life; (h) a representative from each of the medical schools in New York State, provided said representative is a member of the Medical Society of the State of New York; (i) a delegate representing the resident membership; [and] (j) a delegate representing the hospital medical staff section *and (k) officers, trustees, and speakers of the American Medical Association, provided those individuals are members of the Medical Society of the State of New York.*

The committee agrees that the MSSNY would benefit from the experience of those members who are officers of the American Medical Association remaining as members of the MSSNY House of Delegates.

Therefore, the committee recommends **ADOPTION** of Resolution 84-65.

Your chairman would like to thank, in addition to the members of the Committee and the physicians who testified before it, those members of staff who have greatly helped this Committee; Donald R. Moy, J.D., General Counsel and Mrs. Joan Grimm for her secretarial assistance to the Committee.

Respectfully submitted,

George Lim, M.D., Chairman

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

cies in the Aged. Mount Sinai Medical Center. Info: Page & William Black Post-Graduate School of Medicine, One Gustave L. Levy Pl, New York, NY 10029. Tel: (212) 650-6737.

May 21. **Family Systems—Perspectives on Psychophysiologic Disorders.** Lenox Hill Hospital. Info: Mrs V. Matthes, 542 Baldwin Ave, Baldwin, NY 11510. Tel: (516) 379-1787.

May 23–24. **Vascular Surgery 1985—Current State of the Art, Second International Vascular Symposium.** Waldorf Astoria. Info: Ann J. Boehme, CME, LIJ-Hillside Medical Center, New Hyde Park, NY 11042. Tel: (212) 470-2114.

BROOKLYN

May 1. **Infectious Complications of Neoplastic Diseases.** Info: Interfaith Medical Center, 555 Prospect Place, Brookyn, NY 11238. Tel: (718) 240-1000.

May 13. **Anesthesia for Patients Undergoing Carotid Endarterectomies.** *May 14.* **Malignant Hyperthermia.** Info: Abraham I. Raskob, MD, CME, SUNY-Downstate Medical Center, Department of Anesthesiology, Box #6, 450 Clarkson Ave, Brooklyn, NY 11203. Tel: (718) 270-1934.

SUFFOLK

May 7. **Managing Newborn Respiratory Problems in the Level II Hospital.** *May 14.* **Follow-Up Care of the Sick Newborn.** Info: Arthur Schwager, MD, Pediatric CME, Huntington Hospital, 270 Park Ave, Huntington, NY 11743. Tel: (516) 427-0530.

May 13. **Pathophysiology & Management of Pulmonary Embolism.** Southampton Hospital. 1½ Cat 1 credits. Info: Donna C. Joslin, CME, Southampton Hospital, 240 Meeting House Lane, Southampton, NY 11968. Tel: (516) 283-2600, ext 505.

ALBANY

May 16. **Capital District Dermatological Society Annual Teaching Day.** Albany. Info: Robert G. Shoss, MD, Executive Park, North, Albany, NY 12203. Tel: (518) 482-8631.

CHIEF EXECUTIVE OFFICER

The Medical Society of the State of New York invites applications for the position of Executive Vice-President. The EVP is the Chief Executive Officer and directs the activities of the 27,000 member Society, located at Lake Success, N.Y.

Salary and benefits negotiable. All replies will be considered in confidence.

Send Curriculum Vitae to:
Chairman, EVP Search Committee

Medical Society of the
State of New York
420 Lakeville Road
Lake Success, NY 11042

BROOME

May 17–22. **1985 Convention New York State Academy of Family Physicians.** Monticello. Info: Bruce A. Bagley, MD, NYS Academy of Family Physicians, 30 W. State St, Colonial Plaza, Binghamton, NY 13901-3223. Tel: (607) 722-7205.

ERIE

May 9. **Advances in Cancer Treatment.** Info: Gayle Bersani, RN, CME, Roswell Park Memorial Institute, 666 Elm St, Buffalo, NY 14263. Tel: (716) 845-2339.

ONONDAGA

May 9. **Loretto Geriatric Center/Upstate Medical Center Geriatric Medicine Teaching Day.** Syracuse. Info: Office of CME, SUNY Upstate Medical Center, 766 Irving Ave, Syracuse, NY 13210. Tel: (315) 473-4606.

May 22. **Ischemic Heart Disease—1985—Diagnosis and Management.** Syracuse. Info: Ross B. Moquin, MD, 301 Prospect Ave, Syracuse, NY 13203.

Index to Advertisers

Ayerst Laboratories . . . 2A,3A,4A,30A,31A,32A
Blue Cross/Blue Shield of Greater NY 3rd Cover
Classified Advertising
. . . . 5A,17A,18A,20A,22A,24A,26A,28A
CyCare 29A
Knoll Pharmaceuticals 6A,7A,8A
Eli Lilly and Co. . . . . 2nd Cover
Marion Laboratories 9A,10A
Medical Quadrangle 20A
Northwest Mutual Life 5A
Pertec Computer Corp. 23A
Sarantos and Company 21A
Stuart Pharmaceuticals 25A,26A
Upjohn Company . . . . 4th Cover
Wyeth Laboratories 27A,28A

Medical Society of the
State of New York

Annual Dinner Dance

in honor of
Allison B. Landolt, M.D.
President

Thursday
June 6, 1985
Trianon Ballroom
The New York Hilton
53rd Street & Avenue of America

Cocktail Hour
7 P.M.

Superlative
Dinner

Subscription $70.00 per person

Reservation Form

Make check payable to:
Medical Society of the State of New York
420 Lakeville Road
Lake Success, New York 11042

Please send me ________ tickets for the
Annual Dinner Dance on June 6, 1985
Check enclosed for $__________

PLEASE NOTE: Reservations and ticket sales will close at 12 Noon on Thursday, June 6, one hour before the House Convenes.

Name____

Address______________________________________

______________________________Zip______________

NATIONAL EMERGENCY SERVICES, INC. (N.E.S.)
Directorships, full- and part-time emergency department opportunities are available throughout New York State and the country for primary care physicians with ER experience. Enjoy a variety of practice settings, competitive remuneration, paid malpractice insurance, and flexible scheduling. For information and application materials contact the recruitment department at (516) 365-4010, (Outside N.Y. State 1-800-645-4848) or send C.V. to N.E.S., One Hollow Lane, Suite 304, Lake Success, N.Y. 11042.

OTSEGO

May 16. **Ninth Annual Fox/Bassett/Otsego County Medical Society Teaching Day—Dermatology for Clinicians.** Oneonta. 3 Cat 1 credits. Info: Bonnie Kaido, CME, 1251, The Mary Imogene Bassett Hospital, Atwell Road, Cooperstown, NY 13326-1394. Tel: (607) 547-3764.

ROCKLAND

May 31. **Current Concepts in Myocarditis.** Nyack. Info: Ms Myrna Hahn, Nyack Hospital, N. Midland Ave, Nyack, NY 10960. Tel: (914) 358-6200, ext 2509.

ST. LAWRENCE

May 9. **Update in Asthma Therapy.** Canton-Potsdam Hospital. *May 16.* **Computers in Medicine.** Medical Society of the County of St. Lawrence. Info: Sebastian A. Mazzotta, MD, Medical Society of the County of St. Lawrence, 53 South Main St, Norwood, NY 13668. Tel: (315) 353-2332.

AROUND THE NATION

CALIFORNIA

May 2–4. **Postgraduate Course in General Surgery.** *May 11–17.* **San Francisco Orthopaedic Review Course—1985.** *May 23–24.* **Symposium on Current Issues in Anatomic Pathology.** *May 30–31.* **Neurosurgery Postgraduate Course.** San Francisco. Info: University of California San Francisco Extended Programs in Medical Education, Room 569-U, San Francisco, CA 94143. Tel: (415) 666-4251.

FLORIDA

May 10–12. **Cardiology Update: Diagnosis & Treatment of Conduction Disturbances.** Clearwater Beach. Info: Anne Criss Taylor, Rogers Heart Foundation, St. Anthony's Hospital,

STRENGTHEN YOUR ROLE IN
HOSPITAL MEDICAL STAFF LEADERSHIP...

INFLUENCE AMA POLICY

Participate • Influence Organized Medicine •
Participate • Solve Medical Staff Concerns •
Participate • Face Medical Staff Issues •
Participate • Predict Medical Staff Trends •
Participate •

HMSS

AMA Hospital Medical Staff Section
Fifth Assembly Meeting
June 13-17, 1985
Hyatt Regency Hotel
Chicago

For Information Contact:

American Medical Association
Hospital Medical Staff Services
535 North Dearborn Street
Chicago, Illinois 60610
Phone (312) 645-4747 or (312) 645-4753

FOR PHYSICIANS AND SURGEONS WITH A PART-TIME EXPOSURE ONLY

Now offering Medical Malpractice Insurance on an ***occurrence basis*** at drastically reduced rates thru Frontier Insurance Co., Monticello, NY.

With the tail on claims made policies, you might lose your shirt.

Call or write for further information and application.

MEDICAL MQ QUADRANGLE

Medical Quadrangle, Inc.
P. O. Box 456
318 Bedford Road
Bedford Hills, New York 10507
914 666-0170
212 829-3654
"Specializing in Medical Liability Insurance"

601 12th St North, St. Petersburg, FL 33705. Tel: (813) 894-0790.

ILLINOIS

May 8–10. **Second Biennial Chicago Cardiovascular Symposium.** *May 31–June 1.* **Clinical Approaches to Arterial Blood Gas Interpretation and Electrolytes.** Chicago. Info: Registrar, American College of Physicians, 4200 Pine St, Philadelphia, PA 19104. Tel: (215) 243-1200.

13th World Congress
ISRAEL MEDICAL ASSOCIATION
MAY 19 - 24, 1985
Jerusalem - Israel
All medical specialties have session
C.M.E. Credit Approved
For complete travel details
and special conference rates contact:
TRAVELAND
4270 Main St., Bridgeport, CT 06606
203-371-1114 / Out-of-State 800-421-0374
Tour Scientific Coordinator:
Professor Kier — Yale University

LOUISIANA

May 13–17. **Family Practice Update.** New Orleans. Info: Family Practice Update CME, LSU School of Medicine, 1542 Tulane Ave, New Orleans, LA 70112. Tel: (504) 568-6085.

MARYLAND

May 1–3. **Management of Complex Vascular Problems.** *May 8–10.* **Clinical Applications of Doppler Echocardiography—1985.** *May 20–22.* **Clinical Applications of Stress Testing Techniques in Coronary Heart Disease.** Bethesda. Info: American College of Cardiology Learning Center Registrar, 9111 Old Georgetown Rd, Bethesda, MD 20814. Tel: (301) 897-5400, ext 241.

MASSACHUSETTS

May 16–18. **Topics in Internal Medicine.** 16 Cat 1 credits. Boston. Info: Lahey Clinic Medical Center, 41 Mall Rd, Burlington, MA 01803. Tel: (617) 273-8807.

PENNSYLVANIA

May 15–17. **Update in Infectious Diseases.** Philadelphia. Info: Registrar, American College of Physicians, 4200 Pine St, Philadelphia, PA 19104. Tel: (215) 243-1200.

TENNESSEE

May 10–11. **Critical Hemodynamic Diagnosis, Monitoring and Management.** Chattanooga. Info: Registrar, American College of Physicians, 4200 Pine St, Philadelphia, PA 19104. Tel: (215) 243-1200.

TEXAS

May 15–17. **Clinical Approaches to Arterial Blood Gas Interpretation and Electrolytes:** Dallas. Info: Registrar, American College of Physicians, 4200 Pine St, Philadelphia, PA 19104. Tel: (215) 243-1200.

"Why your investments aren't as successful as you are."

Face it. Few problems are as complex as the management of your practice, money and taxes. And few health care practitioners have the extensive specialized expertise necessary for success in precisely these disciplines. Expertise which is vital to achieving financial growth, and, ultimately, independence.

As one of the health care practitioners who has been fortunate enough to gain these crucial financial skills, I'd like to share a few observations on why so many otherwise successful practitioners don't have similar success when it comes to their investments.

Strategies to make your earnings grow

A successful practice alone is not enough to guarantee your financial independence. That requires a careful balance of prudent, disciplined tax planning and professional management of your retirement assets.

Too many of you have relied far too much on the stock market...and paid the price for its unpredictability. Why assume such risk–when you can pursue a conservative, non-stock market approach that provides for a reliable, real rate of return? A diversified portfolio based on short-term guaranteed investment vehicles, as well as real estate investments designed specifically for retirement plans, can satisfy both your immediate and future financial needs.

Call in the right professionals now

Deciding how to invest isn't easy. Financial strategies present a complex picture–but the right professionals can help you focus in on the best solutions for you. I've compiled "25 Investment Pitfalls and How the Health Care Practitioner Can Avoid Them." Your phone call or coupon will bring these valuable insights free of charge–and start you on the path to making more of what you earn with the help of our team of qualified specialists.

So call me, Dr. Randy Sarantos, or my colleague, Jay Cruice, Certified Financial Planner, today. Whatever your concerns–practice evaluation, investment counseling, or any other aspect of personal financial planning–one firm does have the financial solutions for the health care practitioner...Sarantos & Company.

Sarantos & Company, Inc.
Registered investment advisors
800-223-0164 212-484-9759

"Total financial planning for the health care practitioner"

Mail To: Sarantos & Company, Inc.
733 Third Avenue, Suite 600
New York, NY 10017

☑ YES, send me my free copy of:
"25 Investment Pitfalls and How the Health Care Practitioner Can Avoid Them."

Also send me information on:
☐ Personal Financial Planning
☐ Investment Management for Retirement Plans

Name____________________
Address____________________
City__________ State__________ Zip__________
Phone: Office ( )________ Home ( )________
NYJM-485

POSITIONS WANTED

SURGERY—Senior Resident in General Surgery at N.C.M.C., will complete residency in June 1986, seeking practice opportunity. Lawrence P. Kelly, M.D., 200 Carman Ave., East Meadow, NY 11554, (516) 542-0733.

ANESTHESIOLOGIST: BE Proficient in all areas, excellent training. 5-years group practice, looking to relocate. Available within 2-months or negotiable later date. Prefers solo or group private practice. Reply Dept. 386 c/o NYSJM.

PRACTICES WANTED

YOUNG INTERNIST, wishes to purchase Nassau County office based or nursing home based private practice. Available immediately. Strictly confidential. Reply Dept. 365, c/o NYSJM.

OPHTHALMOLOGIST, BOARD CERTIFIED desires to purchase practice in New York City or suburban area. Confidence maintained. Please contact Dept. 168, c/o NYSJM.

JUNE 15, 1985
5th Annual Advances in Gastroenterology
Golden Nugget Hotel
Atlantic City, New Jersey

Sponsored by the Gastrointestinal Section of the Hospital of the University of Pennsylvania

and

the Continuing Medical Education Department of the Underwood Memorial Hospital, Woodbury, New Jersey

Category 1 credit offered

Information: Registration Supervisor, SLACK, Incorporated, 6900 Grove Road, Thorofare, New Jersey 08086, 609-848-1000.

Need A Temporary Physician?
During: vacations, CME's, illness, recruiting.
Want Free Time While You Practice Medicine?
Join CompHealth's Locum Tenens Physician Group.
For further information, call 412-741-3310.

CompHealth A Physician Group
WILSON ROSS, Regional Administrator
114 Centennial Ave., Sewickley, PA 15143

PRACTICES AVAILABLE

SELLING PRACTICES IS OUR ONLY BUSINESS—Want to maximize the return on your investment? Before you buy or sell, call for a consultation to assure the best financial and transfer terms. We guide you through the entire sales process from initial meeting to closing. Serving 100 mile radius of New York City. Countrywide Business Brokerage, Inc., 319 East 24th Street, Suite 23-G, New York, NY 10010 (212) 686-7902.

INTERNAL MEDICINE practice for sale. Well-established in Nassau County. Home-office, fully equipped for one or two physicians. Telephone (516) 747-3072.

PRACTICE FOR SALE: Active lucrative primary care internal medicine; prime office location in prime area west county, 40 minutes from Manhattan. Excellent opportunity for most subspecialties especially ID. Will spend 3–5 years transferring practice. Opportunity for creative, vigorous, young M.D. Send CV to Dept. 385 c/o NYSJM.

EQUIPMENT

WANTED: a good fluoroscopic screen in place of my old one. Prof-Xray screen needed 12 in. x 12 in., Direction 11824A, Code 10-3050. Urgent! Call (607) 756-9941, collect.

NEW LAW CUTS TAX BREAKS FOR LUXURY CARS

If the car you want costs more than $16,667.00 don't spend your money, use ours—we still pass the investment tax credit to you.

If you're in the 50% tax bracket no longer can you save half the depreciation on anything over $16,677.00 for the first 3 years:

HOWEVER,

When you lease you can continue to deduct your monthly rental payments in excess of $16,000.00 *provided* you conform to the limits and rules set by the I.R.S. regarding usage. The leasee must use the vehicle more than 50% of the time for his business or profession.

SO DOESN'T IT MAKE SENSE TO LEASE NOW!

ALL MAKES & MODELS AVAILABLE.

CALL CAROL LEASING INC.

(516) 222-9041

ROOSEVELT RACEWAY, WESTBURY, NY 11590

The **Professional** Answer To Medical Office Efficiency…

There's a lot of talk today about computerizing medical practices. But what do you really need?

As specialists in medical office automation we want you to know about the **professional** answer: the Basic Oriented Medical Business System (BOMBS)*…one of the Specialist Series of software products for the healthcare field.

BOMBS will streamline your paperwork, eliminate backlogs, increase your cash flow, expedite collections, and facilitate insurance payments.

Because BOMBS is modular, you can add software for appointment scheduling, medical profiling, medical recall, and even letter writing.

The BOMBS system has the endorsement of the American Society of Internal Medicine, and is currently being evaluated by several other medical societies. A stable, proven system, BOMBS is currently installed in over twenty states.

BOMBS operates on the outstanding Pertec 3200 Cable Computer System. This multi-user, state-of-the-art system expands as your practice grows, without sacrifice of performance. The system can accommodate up to 100,000 patients, with an unlimited number of services for each patient.

* BOMBS is a product of C&S Research Corp.

If you want your busy medical office to experience a new level of efficiency, send back the coupon today; and we'll rush you our informative, four-color brochure: MEDICAL PRACTICE MANAGEMENT.

PERTEC

Computers for Business Growth

Yes, I'm interested in what the Basic Oriented Medical Business System (BOMBS) can do for my practice.

☐ Please send your informative four-color brochure

☐ Have a representative call

Practice name____________________

Contact____________________

Address____________________

City__________State____Zip____

Telephone___ ____________
Area Number

Number of physicians____

Specialty__________

PERTEC COMPUTER CORPORATION
17112 Armstrong Avenue,
Mail Stop 71-81
Irvine, CA 92713-9602

NYJ4

U.S. SAVINGS BONDS

MONEY MARKET RATES. GUARANTEED RETURN. YOU STILL CAN'T LOSE WITH SAVINGS BONDS.

While the interest rates have dramatically changed, U.S. Savings Bonds still represent the safest investment in our country. That's because Savings Bonds receive the full backing of the U.S. Government—and offer you the added security of a guaranteed return.

After you hold your Savings Bonds for only five years, you always earn at least 7½% interest—no matter what's happening to other interest rates in the financial markets. Before your Bonds reach the five-year point, they earn interest on a fixed graduated scale, rising from 5.5% after one year to the guaranteed return of 7.5% at five years. Also, since every Bond is registered, Bonds can be replaced free if lost, stolen or destroyed.

GET THE ADDED VALUE OF TAX ADVANTAGES.

Here's even more good news about U.S. Savings Bonds. The interest you earn from your Bonds is not subject to state or local income taxes. This increases the effective yield you'll receive with Savings Bonds' variable rates. There's another tax advantage, too. With Savings Bonds, you can defer your federal tax on the interest you earn.

BACK TO THE BASICS OF U.S. SAVINGS BONDS.

After all this talk about Savings Bonds, maybe you're wondering what they actually are. Bonds are accrual-type registered securities. This means that the interest you earn is paid only when the Bond is cashed in. And you must hold Bonds for at least six months before they can be redeemed.

The purchase price of Bonds is always 50 percent of the face amount. And Bonds are available in eight denominations —$50, $75, $100, $200, $500, $1,000, $5,000, $10,000. With such a wide range of purchase prices, Savings Bonds are an affordable investment for everyone. And with the new variable interest rates, they're a smart investment, too.

Series EE-Monthly Accumulations

MONTHLY	AT 7.5%			AT 10%
SAVINGS	1 YEAR	5 YEARS	10 YEARS	10 YEARS
$ 6.25	75.94	434.70	1074.00	1196.90
12.50	152.82	874.52	2161.04	2411.42
25.00	306.60	1759.34	4348.46	4863.34
50.00	613.20	3518.68	8696.92	9726.68

Series EE-BiWeekly Accumulations

BIWEEKLY	AT 7.5%			AT 10%
SAVINGS	1 YEAR	5 YEARS	10 YEARS	10 YEARS
6.25	172.09	957.55	2356.91	2633.95
12.50	344.62	1920.00	4727.96	5286.86
25.00	690.12	3846.04	9469.92	10597.26
50.00	1380.24	7692.08	18939.84	21194.52

MISCELLANEOUS

JUNE 10-14, 1985, UPDATE YOUR MEDICINE 1985. Association of Practicing Physicians of the New York Hospital and Cornell University Medical College. 37 hours Category 1 credit, 49 if both optional workshops are taken. 9 major review lectures, 16 workshops, 2 Meet the Professor luncheons, Grand Rounds and optional practical workshops in breast/pelvic and male genitorectal examinations. Held at New York Hospital-Cornell, 1300 York Avenue at 69th Street, New York. Information: Office of CME, (212) 472-6119 or 5600. Dr. Lila Wallis, Course Director. Also call us for information about Update Your Medicine, Ithaca course July 22-26, 1985.

ARCHITECTURE & PLANNING, Comprehensive services from analysis of alternative office locations through design and construction. Experienced in both new construction and renovation. Lewis & Gould (212) 807-6588.

TAX ACCOUNTANT-ATTORNEY, formerly with I.R.S., announces his availability to prepare taxes, accounting services, advise on tax shelters, pension plans and represent buyers or sellers of medical practices. I have other doctor clients. Gerald Goldberg, (212) 406-1156 and (516) 365-5679.

1985 CME CRUISE/CONFERENCES on Selected Medical Topics—Caribbean, Mexican, Hawaiian, Alaskan, Mediterranean. 7-14 days year-round. Approved for 20-24 CME Cat. 1 credits (AMA/PRA) & AAFP prescribed credit. Distinguished professors. Fly Roundtrip Free on Caribbean, Mexican & Alaskan Cruises. Excellent group fares on finest ships. Registration Limited. Pre-scheduled in compliance with present IRS requiremnets. Information: International Conferences, 189 Lodge Ave., Huntington Station, NY 11746. (516) 549-0869.

AVOID MALPRACTICE SUITS—Free 30 Day Trial "Guidelines For Medical and Surgical Emergencies". Write professional type orders for laboratory and treatment. Masson Publishing Company—U.S.A., 133 East 58th Street, New York, NY 10022.

ARCHITECTURAL DESIGN AND CONSTRUCTION: Designers, builders of custom professional offices. Consulting services available. Whether you're opening a new office, relocating, remodeling or expanding an existing office in New York City area and adjacent New Jersey counties. Donald Young, Inc. Tel: (718) 948-5191.

BORROW $50,000 (or less) on your signature alone. Doctor? Lawyer? Executive earning $40,000? You could qualify for Profesco Professional Funding. No pre-payment penalty; Competitive rates; Financing arranged in 72 hours. For details; application: M. Fisher, Account Executive, John Hancock-Profesco, Suite 505, 3601 Hempstead Turnpike, Levittown, NY 11756, (516) 579-4455 or reply Dept. 381 c/o NYSJM.

EXECUTIVE DIRECTOR—Non-profit professional health review organization in Manhattan seeks to fill the position of Executive Director. Individual functions as chief executive officer reporting to Board of Directors. Extensive previous experience with peer review activity required. Managerial experience and familiarity in dealing with governmental and private health care agencies and providers preferred. Excellent salary and full fringe benefits package. Interested candidates should submit a Curriculum Vitae to Dept. 382 c/o NYSJM. An equal opportunity employer.

Angina comes in many forms...

So does SORBITRATE®

(ISOSORBIDE DINITRATE)

Unsurpassed flexibility in nitrate therapy.

2.5 mg 5 mg 10 mg
Sublingual Tablets

5 mg 10 mg
Chewable Tablets

5 mg 10 mg 20 mg 30 mg 40 mg
Oral "Swallow" Tablets

40 mg
Sustained Action
"Swallow" Tablets

© 1985 ICI AMERICAS INC.

See following page for brief summary of prescribing information.

SORBITRATE®
(ISOSORBIDE DINITRATE)

Please consult full prescribing information before use. A summary follows:

INDICATIONS AND USAGE: SORBITRATE (isosorbide dinitrate) is indicated for the treatment and prevention of angina pectoris All dosage forms of isosorbide dinitrate may be used prophylactically to decrease frequency and severity of anginal attacks and can be expected to decrease the need for sublingual nitroglycerin

The sublingual and chewable forms of the drug are indicated for acute prophylaxis of angina pectoris when taken a few minutes before situations likely to provoke anginal attacks Because of a slower onset of effect, the oral forms of isosorbide dinitrate are not indicated for acute prophylaxis

CONTRAINDICATIONS: SORBITRATE is contraindicated in patients who have shown purported hypersensitivity or idiosyncrasy to it or other nitrates or nitrites. Epinephrine and related compounds are ineffective in reversing the severe hypotensive events associated with overdose and are contraindicated in this situation

WARNINGS: The benefits of SORBITRATE during the early days of an acute myocardial infarction have not been established If one elects to use organic nitrates in early infarction, hemodynamic monitoring and frequent clinical assessment should be used because of the potential deleterious effects of hypotension

PRECAUTIONS: General: Severe hypotensive response, particularly with upright posture, may occur with even small doses of SORBITRATE The drug should therefore be used with caution in subjects who may have blood volume depletion from diuretic therapy or in subjects who have low systolic blood pressure (eg, below 90 mmHg) Paradoxical bradycardia and increased angina pectoris may accompany nitrate-induced hypotension Nitrate therapy may aggravate the angina caused by hypertrophic cardiomyopathy

Marked symptomatic, orthostatic hypotension has been reported when calcium channel blockers and organic nitrates were used in combination Dose adjustment of either class of agents may be necessary

Tolerance to this drug and cross-tolerance to other nitrates and nitrites may occur Tolerance to the vascular and antianginal effects of isosorbide dinitrate or nitroglycerin has been demonstrated in clinical trials, experience through occupational exposure, and in isolated tissue experiments in the laboratory The importance of tolerance to the appropriate use of isosorbide dinitrate in the management of patients with angina pectoris has not been determined However, one clinical trial using treadmill exercise tolerance (as an end point) found an 8-hour duration of action of oral isosorbide dinitrate following the first dose (after a 2-week placebo washout) and only a 2-hour duration of effect of the same dose after 1 week of repetitive dosing at conventional dosing intervals On the other hand, several trials have been able to differentiate isosorbide dinitrate from placebo after 4 weeks of therapy and, in open trials, an effect seems detectable for as long as several months

Tolerance clearly occurs in industrial workers continuously exposed to nitroglycerin. Moreover, physical dependence also occurs since chest pain, acute myocardial infarction, and even sudden death have occurred during temporary withdrawal of nitroglycerin from the workers In clinical trials in angina patients, there are reports of anginal attacks being more easily provoked and of rebound in the hemodynamic effects soon after nitrate withdrawal The relative importance of these observations to the routine, clinical use of isosorbide dinitrate is not known However, it seems prudent to gradually withdraw patients from isosorbide dinitrate when the therapy is being terminated, rather than stopping the drug abruptly

Information for Patients: Headache may occur during initial therapy with SORBITRATE Headache is usually relieved by the use of standard headache remedies or by lowering the dose and tends to disappear after the first week or two of use

Drug Interactions: Alcohol may enhance any marked sensitivity to the hypotensive effect of nitrates.

Isosorbide dinitrate acts directly on vascular smooth muscle, therefore, any other agent that depends on vascular smooth muscle as the final common path can be expected to have decreased or increased effect depending on the agent.

Carcinogenesis, Mutagenesis, Impairment of Fertility: No long-term studies in animals have been performed to evaluate the carcinogenic potential of this drug A modified two-litter reproduction study in rats fed isosorbide dinitrate at 25 or 100 mg/kg/day did not reveal any effects on fertility or gestation or any remarkable gross pathology in any parent or offspring fed isosorbide dinitrate as compared with rats fed a basal-controlled diet

Pregnancy Category C: Isosorbide dinitrate has been shown to cause a dose-related increase in embryotoxicity (increase in mummified pups) in rabbits at oral doses 35 and 150 times the maximum recommended human daily dose There are no adequate and well-controlled studies in pregnant women SORBITRATE should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: It is not known whether this drug is excreted in human milk Because many drugs are excreted in human milk, caution should be exercised when SORBITRATE is administered to a nursing woman.

Pediatric Use: The safety and effectiveness of SORBITRATE in children has not been established

ADVERSE REACTIONS: Adverse reactions, particularly headache and hypotension, are dose-related In clinical trials at various doses, the following have been observed

Headache is the most common (reported incidence varies widely, apparently being dose-related, with an average occurrence of about 25%) adverse reaction and may be severe and persistent Cutaneous vasodilation with flushing may occur Transient episodes of dizziness and weakness, as well as other signs of cerebral ischemia associated with postural hypotension, may occasionally develop (the incidence of reported symptomatic hypotension ranges from 2% to 36%) An occasional individual will exhibit marked sensitivity to the hypotensive effects of nitrates and severe responses (nausea, vomiting, weakness, restlessness, pallor, perspiration, and collapse) may occur even with the usual therapeutic dose Drug rash and/or exfoliative dermatitis may occasionally occur. Nausea and vomiting appear to be uncommon Case reports of clinically significant methemoglobinemia are rare at conventional doses of organic nitrates The formation of methemoglobin is dose-related and, in the case of genetic abnormalities of hemoglobin that favor methemoglobin formation, even conventional doses of organic nitrate could produce harmful concentrations of methemoglobin.

DOSAGE AND ADMINISTRATION: For the treatment of angina pectoris, the usual starting dose for sublingual SORBITRATE is 2 5 to 5 mg; for chewable tablets, 5 mg, for oral (swallowed) tablets, 5 to 20 mg, and for controlled-release forms, 40 mg

SORBITRATE should be titrated upward until angina is relieved or side effects limit the dose In ambulatory patients, the magnitude of the incremental dose increase should be guided by measurements of standing blood pressure

The initial dosage of sublingual or chewable SORBITRATE for prophylactic therapy in angina pectoris patients is generally 5 or 10 mg every 2 to 3 hours Adequate controlled clinical studies demonstrating the effectiveness of chronic maintenance therapy with these dosage forms have not been reported

SORBITRATE in oral doses of 10 to 40 mg given every 6 hours or in oral controlled-release doses of 40 to 80 mg given every 8 to 12 hours is generally recommended The extent to which development of tolerance should modify the dosage program has not been defined *The oral controlled-release forms of isosorbide dinitrate should not be chewed*

DOSAGE FORMS AVAILABLE: Sublingual Tablets (2 5, 5, 10 mg), Chewable Tablets (5, 10 mg), Oral Tablets (5, 10, 20, 30, 40 mg), Sustained Action Tablets (40 mg)

STUART PHARMACEUTICALS
Division of ICI Americas Inc.
Wilmington, DE 19897

STR-2282

MISCELLANEOUS—CONT'D

DOCTOR, YOU CAN'T BEAT the quality or the price! Holter Monitor Scanning Service. Physician owned, trained and supervised. No contracts to sign. We can arrange for lease/purchase of Holter equipment. New dual channel recorders with test cables $1,550. Now using UP Service for faster turn around time. DCG Interpretation (313) 879-8860.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuariat certification and employee statements of participation—partnership agreements and professional incorporation—No insurance required—references upon request—Jack Wachstock, PC, 3000 Marcus Ave., Lake Success, N.Y. 11530 (516) 437-4848.

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate, with no points, fees or charges of any kind. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

MEDSTAR: THE CUSTOMIZED COMPUTER SYSTEM for medical office management. Designed by a physician and his staff. Completely customized for each practice. Affordable, Reliable, Easy to use. Leasing available. Medstar, 363 Route 111, Smithtown, NY 11787. (516) 360-1422.

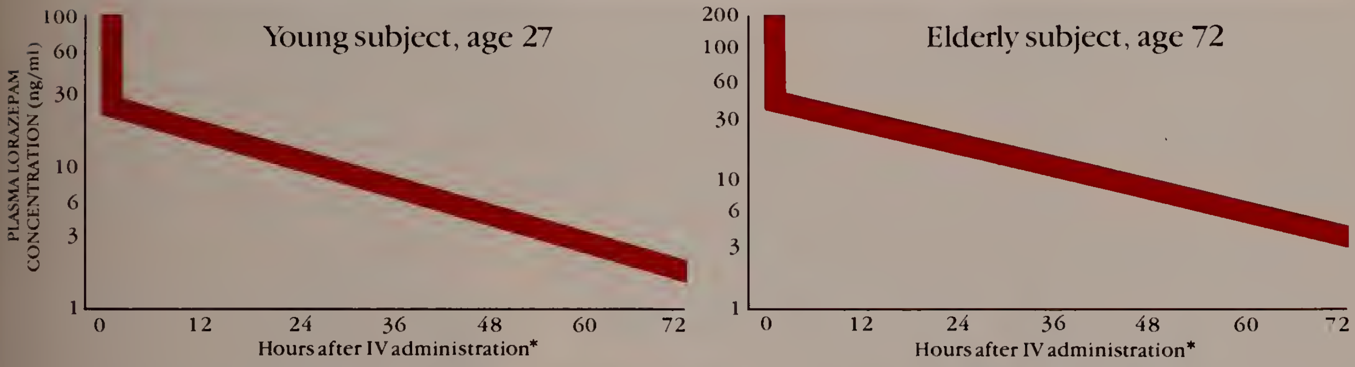
REAL ESTATE FOR SALE OR RENT

GASTROINTESTINAL ENDOSCOPY SUITES. Rent by procedure or hour. Fully equipped with latest Olympus equipment including video. Full-time gastrointestinal assistants. "Park Avenue Brownstone" location. Endo-Center, Inc. (212) 751-0365.

FLUSHING, QUEENS: for rent, fully equipped dental office or can be used for any other professional. 2 blocks fron Northern Blvd. Call (718) 939-5210.

Among leading benzodiazepines, only Ativan has proof... pharmacokinetics not significantly altered by age.[1]

Representative charts of comparison testing



Lorazepam is nearly 100 percent bioavailable by the intramuscular and oral routes, as compared to the intravenous. Therefore, data for clearance of intravenous lorazepam are equally applicable to oral lorazepam.

- **Clearance not significantly delayed by age, liver or kidney dysfunction**
- **Cumulative sedative effects seldom a problem**
- **Available in 0.5-mg tablets to facilitate the recommended geriatric starting dosage**

[1] Greenblatt DJ: Clinical study, pharmacokinetics and bioavailability in the elderly, Ativan® (lorazepam). Data on file, Wyeth Laboratories.

*Fourteen subjects, aged 60 to 84 years, participated in the study. Twelve subjects, aged 19 to 32 years, served as "young controls." Subject dosage was adjusted for body weight and ranged from 1.5 mg to 3.0 mg of lorazepam. Within the study, lorazepam clearance was monitored following IV, IM and oral administration in the elderly group and following IV administration in the control group. The effect of aging on total clearance of lorazepam was relatively small and not statistically significant. Half-life values following the three different routes of administration were essentially identical.

Only Ativan® (lorazepam) Ⓒ IV

Wyeth Laboratories
Philadelphia, PA 19101

© 1985, Wyeth Laboratories.

See important information on following page.

The appearance of these tablets is a registered trademark of Wyeth Laboratories

DISTINCTIVE DESIGN® 0.5, 1.0 and 2.0 mg

Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence. Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Transient amnesia or memory impairment has been reported in association with the use of benzodiazepines.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® Ⓒ IV
for (lorazepam)
Anxiety

DOSAGE: Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.

HOW SUPPLIED: 0.5, 1.0 and 2.0mg tablets.

Wyeth Laboratories
Philadelphia, PA 19101

REAL ESTATE FOR SALE OR RENT—CONT'D

MANHATTAN
WEST 55 ST.
STREET LEVEL
1,000 SQ. FT.
Principals only
Lerner (212) 736-8400

WANTED: MEDICAL OFFICE BUILDINGS. As principals we are seeking "existing buildings"—fully tennanted by physicians, dentists and health care services. Contact: Paul Gellert, Gelco Associates, 155 West 68th St., N.Y., N.Y. 10023; (212) 724-7900.

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

OZONE PARK, QUEENS: Lower level of busy Medical Building, stable community, for any medical related activity. 3,400 square feet available. Will subdivide to suit and consider any arrangements. Very favorable terms, great opportunity. Call (718) 848-6790 days or (718) 835-5259 nights and weekends. Ample finders fee available.

BAY SHORE, leasing new luxurious medical building. Located near Southside Hospital. Minimum suite 740 sq. ft. T.V. Monitored hallway. Large parking area, handicapped designed. Individual gas heat, a/c-electric. (516) 665-4064, 10 A.M. to 5 P.M., Monday to Friday.

BEAUTIFULLY DECORATED SPACIOUS OFFICE, 57 West 57th Street, Center of town. Hospital, Lab. and x-ray in building. All mornings $1250.00. Some afternoons if required. Call EL 5-7979, 2–4:30 P.M. (except Wednesday) (212) 580-8870 Mornings and Evenings.

COUNTRY ESTATE: for gracious living or weekend retreat. Catskills. 100 miles from N.Y.C. 9 room ranch, large living room with fireplace, dining room, 4 bed rooms, 3 baths, superb kitchen with Jenair stove and microwave, family room, large storage cellar, separate 4 room guest house. 4 car garage, large barn, trout pond, bass pond, skiing, snowmobiling, deer, wild turkey, magnificent view. $239,500 (201) 391-4801 or (914) 351-2346.

FIRST OFFERING: CENTRAL PARK WEST MEDICAL—86W. & C.P.W. Prime location, subway & buses at door. 100–12,000 sq. ft. for rent, time sharing, built to suit, doorman, concierge. MUSEUM WEST MEDICAL—79W. & Columbus. 1 suite remaining. LANDMARK MEDICAL—Carroll Gardens, Court St., join major radiology group. 2 suites built to suit available. FLUSHING MEDICAL ARTS CENTER—across from Flush. Hospital, suite available. Call Professional Enterprises award winning developers of Prof. Space (718) 229-8440.

We've been studying medicine almost as long as you have.

Over 17 years and untold manhours. That's what CyCare has invested in the study of health care. We've long since earned our diploma. While many aspiring competitors failed to make the grade.

We chose one specialty. The delivery of health care is a specialized business. Your data processing company should understand it thoroughly. That's tough to do if they're also marketing to banks, factories and the like.

From the beginning, CyCare decided to commit only to the medical industry. Our staff of over 600 has been living and breathing it ever since.

Experience only CyCare can claim. CyCare has studied with thousands of physicians, administrators, and nurses. We've worked with nearly 850 clients of all specialties and size. In 17 years, we've treated data processing challenges of every kind.

You know experience is the best teacher. So choose a company that's been around long enough to learn.

Our knowledge benefits you. Our experience taught us that each client is different. We designed systems that easily accomodate any type of case. We learned you didn't want useless "bells and whistles", so we developed practical software that enhances the delivery of patient care. We discovered the fear of system obsolescence. So we created modular systems that can be expanded at any time.

Learning never stops. Like you, we never stop learning. We invest more in research each year than most of our competitors gross in sales. We listen to your ideas, look for new ways to improve your practice, and stay abreast of industry needs. It's the only way to take the lead.

Compare our credentials. Examine CyCare thoroughly. Demand as much from us as you demand from yourself. Look at our experience, our financial stability. Examine our products and talk to our clients. Find out why CyCare is the leading supplier to medical group practices, HMOs and ambulatory care facilities nationwide.

Put us to the test. We're prepared.

Ask about CyCare's C100 APPOINTMENT SCHEDULING. The physician time management system for small and medium size practices.

☐ Rush free details to me about CyCare.
☐ Have a representative contact me.
My business card/letterhead is attached.
No. of Phys.____
Mail to: CyCare, 520 Dubuque Building Dubuque, Iowa 52001
319/556-3131
NYSJM 4/85

Sales and Service Offices:
Atlanta, GA; Cherry Hill, NJ; Chicago, IL; Dallas, TX; Denver, CO; Miami, FL; Minneapolis, MN; New York, NY; Portland, OR; San Diego, CA; Spokane, WA; Canada: Toronto, Ont.

Authorized WANG National ISO

cycare
a step ahead...

Right from the start in hypertension…

Once-daily INDERAL LA (propranolol HCl) for smooth blood pressure control without the potassium problems of diuretics

Once-daily INDERAL LA (propranolol HCl) avoids the risk of diuretic-induced ECG abnormalities due to hypokalemia.[1,2] In addition, INDERAL LA preserves potassium balance without additive agents or supplements while providing simple, well-tolerated therapy with broad cardiovascular benefits.

Once-daily INDERAL LA for the cardiovascular benefits of the world's leading beta blocker

Simply start with 80 mg once daily. Dosage may be increased to 120 mg to 160 mg once daily as needed to achieve additional control.

Like conventional INDERAL tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.

For beta-1/beta-2 blockade

Once-daily

INDERAL LA

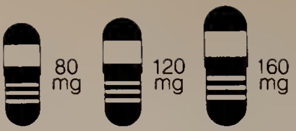
(PROPRANOLOL HCl)

80 mg 120 mg 160 mg

The appearance of these capsules is a registered trademark of Ayerst Laboratories

Please see brief summary of prescribing information on the next page for further details

Once-daily
For beta-1/beta-2 blockade INDERAL® LA
(PROPRANOLOL HCl) LONG ACTING CAPSULES



The appearance of these capsules is a registered trademark of Ayerst Laboratories

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*
INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use Effects on plasma volume appear to be minor and somewhat variable INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential. The significance of the membrane action in the treatment of arrhythmias is uncertain

The mechanism of the antimigraine effect of propranolol has not been established Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient. But there are also situations in which sympathetic stimulation is vital. For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

Propranolol is not significantly dialyzable

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina palpitations, and syncope. INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE continued use of beta blockers can in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)— PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g , dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General:* Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure

Clinical Laboratory Tests Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy Pregnancy Category C INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman.

Pediatric Use Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular bradycardia, congestive heart failure, intensification of AV block, hypotension; paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type

Central Nervous System: lightheadedness, mental depression manifested by insomnia, lassitude, weakness, fatigue, reversible mental depression progressing to catatonia, visual disturbances, hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics

Gastrointestinal. nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory: bronchospasm

Hematologic agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous. alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION—*Dosage must be individualized* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic The dosage may be increased to 120 mg once daily or higher until adequate blood pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

REFERENCES

1. Holland OB, Nixon JV, Kuhnert L: Diuretic-induced ventricular ectopic activity. *Am J Med* 1981,70:762-768. **2.** Holme I, Helgeland A, Hjermann I, et al: Treatment of mild hypertension with diuretics. The importance of ECG abnormalities in the Oslo study and in MRFIT. *JAMA* 1984,251.1298-1299

Ayerst®
AYERST LABORATORIES
New York, N Y 10017

9411/1184

Copyright © 1984 AYERST LABORATORIES
Division of AMERICAN HOME PRODUCTS CORPORATION

From Blue Cross and Blue Shield of Greater N.Y.

The last claim form!

Introducing

Amicus 1™

MEDICAL OFFICE MANAGEMENT SYSTEM

A computer system that does something you could never do before

For over 50 years, Blue Cross and Blue Shield of Greater New York has been the most consistently innovative force in helping physicians and patients meet the financial demands of progressive medical care. Now, with advances in the technology of computer networking, we can help you do something never before possible. Amicus 1 lets you transmit claims directly by phone to our computer for faster, more efficient processing of our medical and Medicare B* claims.

Direct communication provides better service

Just as direct communication between people is preferred for accuracy, computer-to-computer communication reduces the potential for errors and delays. When you purchase Amicus 1, you gain significant benefits. Faster collections and fewer errors. Greater productivity. And significant relief of cash flow pain.

Handles most of your accounting and financial management functions

Of course, Amicus 1 does more than just send claims to our computer. It assists you in all your billing and accounts receivable needs. It provides up to the minute financial reports on your practice. And more. All with just a few short entries.

A complete system featuring the IBM® Personal Computer XT

We've developed a ready-to-use system featuring an IBM Personal Computer XT. In addition, Amicus 1 includes a high speed printer; a tape backup storage unit; and a fast modem for telephone data transmission and access to medical and financial services. The Amicus 1 system includes installation with a three-day training program for your office staff. It also includes an on-site service contract plus a special telephone response line available during business hours.

Fill out your last claim form.

Just complete and mail the coupon below. Or, call us at **212 490-4180** to learn how Amicus 1 can help reduce your paperwork, increase your productivity and improve your cash flow.

Tell me how Amicus 1 can work for my practice.
☐ Yes, I'd like to see a personal demonstration.
☐ Please send me more information. NYSJM

1. NAME

2. SPECIALTY

3. ADDRESS *(Street, city, state, ZIP code)*

4. TELEPHONE ( )

5. OFFICE MANAGER

Send completed form to: **Medical Business Services**
P.O. Box 1707 Grand Central Station
New York, N.Y. 10163

Medical Business Services
Div. of
Blue Cross®
Blue Shield®
of Greater New York

*Applies to the 16 counties where Blue Cross and Blue Shield of Greater N.Y. is the Medicare carrier
IBM® is a registered trademark of International Business Machines Corporation
Amicus 1™ is a trademark of Blue Cross and Blue Shield of Greater New York

Motrin®

ibuprofen, Upjohn

600 mg Tablets

More convenient for your patients.

Upjohn

© 1984 The Upjohn Company

The Upjohn Company • Kalamazoo, Michigan 49001 USA

4944 January 1984

STACKS

Annual CME Assembly, June 7–9, New York Hilton Hotel

(See March issue for Preliminary Program)

NEW YORK STATE JOURNAL OF MEDICINE

MAY 1985 • Volume 85, Number 5

Contents

COMMENTARIES

Practical aspects of immunohistochemistry 191
ANN E. WALTS, MD

Ureteropyeloscopy 193
DEMETRIUS H. BAGLEY, MD

Transurethral endoscopy of the upper urinary tract 194
JEFFRY L. HUFFMAN, MD

Long-term survival with metastatic uterine sarcoma 195
EDWARD V. HANNIGAN, MD

RESEARCH PAPERS

Accidents in the skilled nursing facility 202
JACOB DIMANT, MD

Rigid ureteropyeloscopy 205
FUAD J. KHURI, MD, ROBERT J. PEARTREE, MD, RONALD A. RUOTOLO, MD, JOHN R. VALVO, MD

HISTORY

The poultice 207
JOHN S. HALLER, Jr

LEGAL ISSUES

Informed consent: The New York approach 210
JAMES M. MORRISSEY, ESQ

ESSAYS IN PEDIATRICS

Normal adolescent development: Helping teenagers cope with change 214
RICHARD E. KREIPE, MD

CASE REPORTS

Leiomyosarcoma of the ovary 218
SUDHAKAR A. REDDY, MD, TUNG PUI POON, MD, GITA RAMASWAMY, MD, VICTOR TCHERTKOFF, MD

Metastatic leiomyosarcoma of the iliac bone 220
VINITA PATANAPHAN, MD, MOHAMMAD ABDUL HAFIZ, MD, OMAR M. SALAZAR, MD

Recurrent chronic epididymitis as an initial symptom of colovesical fistula 222
CIRIL J. GODEC, MD, DAN RICE, MD, STEVEN YOUNGER, MD

Stage II benign cystic teratoma with malignant squamous degeneration 224
VICKI SELTZER, MD, STEVE VOGL, MD

Posterior dislocation of the sternoclavicular joint 225
MARVIN J. WEINGARTEN, MD, ROBERT TASH, MD, ROBERT M. KLEIN, MD, RICHARD J. KEARNS, MD

Abdominal distention associated with lactose malabsorption in a premature infant 227
STUART BEREZIN, MD, ANITA BHOLE, MD, TZEE CHUNG WU, MD, LEONARD J. NEWMAN, MD

LETTERS TO THE EDITOR

Neutropenia associated with procainamide 197
DAVID P. KIMMEL, MD, LOUIS V. ANTIGNANO, MD, S. SERGE BAROLD, MD, LASZLO BOROS, MD, JOHN P. OLSON, MD

Symposium on violence 197
ANTHONY M. IMPARATO, MD

The Berwid case 198
NATHANIEL S. LEHRMAN, MD
DANIEL W. SCHWARTZ, MD

Utilization review mini quiz 198
JOHN E. JACOBY, MD
CATHERINE GIORDANO, RN

Esophageal ulceration associated with oral penicillin in Marfan's syndrome 199
PERRY C. GOULD, MD, ROBERT S. BARTOLOMEO, MD, HOWARD M. SKLAREK, MD

Advances in the treatment of diabetes mellitus 200
RICHARD K. BERNSTEIN, MD

AIDS: a disease of ancient Egypt? 200
RICHARD J. ABLIN, PHD, MAURICE J. GONDER, MD, RONALD S. IMMERMAN, MD

Dr Stiles D. Ezell 201
PETER H. STERN, MD

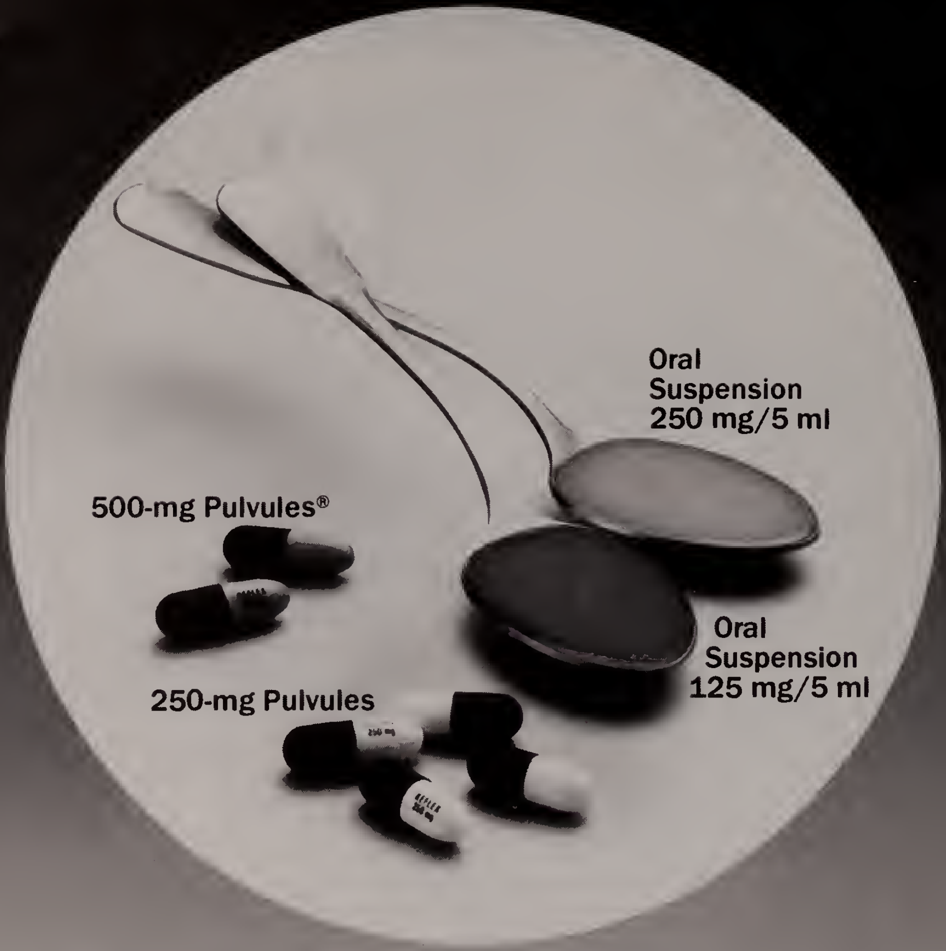
BOOK REVIEWS 228

M. D. S.

LIBRARY OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA
MAY 22 1985

Library of the College Physician
of Philadelphia Dec. 85
19 S. 22nd St.
Phila, PA 19103

Easy To Take



Keflex®
cephalexin

Additional information available to the profession on request.

DISTA
420113

Dista Products Company
Division of Eli Lilly and Company
Indianapolis, Indiana 46285
Mfd. by Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
GERALD P. MURPHY, MD
ELIZA H. CALDWELL, MD
RICHARD B. NOLAN, MD
URSULA MATULONIS (Medical Student)
CARL E. POCHEDLY, MD
JOHN T. PRIOR, MD

Editor	ALAN BLUM, MD
Deputy Editor and Book Review Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Managing Editor	VICKI GLASER
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Administrative Assistant	IMELDA MORRISSEY
Editorial Assistant	JEANNE CONNERY
Librarian	ELLA ABNEY
Library Assistant	ELEANOR BURNS
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1985

RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MARK C. HOMONOFF, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. Synergistic Media Sales, 50 East 42nd St., Suite 504, New York, N.Y. 10017. Telephone: (212) 682-2200.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

ALLISON B. LANDOLT, MD, Westchester	*President*
BERNARD J. PISANI, MD, New York	*Past-President*
DANIEL F. O'KEEFFE, MD, Warren	*President-Elect*
JOHN A. FINKBEINER, MD, New York	*Vice-President*
JOHN H. CARTER, MD, Albany	*Secretary*
GEORGE LIM, MD, Oneida	*Assistant Secretary*
CHARLES D. SHERMAN, JR, MD, Monroe	*Treasurer*
MORTON KURTZ, MD, Queens	*Assistant Treasurer*
JOHN L. CLOWE, MD, Schenectady	*Speaker*
CHARLES N. ASWAD, MD, Broome	*Vice-Speaker*

Councilors

Term Expires 1985	JAMES H. COSGRIFF, JR, MD, Erie SAMUEL M. GELFAND, MD, Nassau RICHARD A. HUGHES, MD, Warren ANTONIO F. LaSORTE, MD, Broome
Term Expires 1986	ROBERT A. MAYERS, MD, Westchester THOMAS D. PEMRICK, MD, Rensselaer RALPH E. SCHLOSSMAN, MD, Queens STANLEY A. STECKLER, MD, Suffolk
Term Expires 1987	DAVID M. BENFORD, MD, New York DUANE M. CADY, MD, Onondaga JAMES M. FLANAGAN, MD, Wayne M. THEODORE TANENHAUS, MD, Brooklyn

Trustees

CARL GOLDMARK, JR, MD, New York, *Chairman*
G. REHMI DENTON, MD, Albany
RALPH M. SCHWARTZ, MD, New York
EDGAR P. BERRY, MD, New York
RICHARD D. EBERLE, MD, Onondaga
JOSEPH F. SHANAPHY, MD, Richmond
VICTOR J. TOFANY, MD, Monroe

Executive Vice-President	GEORGE J. LAWRENCE, JR, MD
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Associate	IRMA A. ERICKSON
Director, Division of Scientific Publications	ALAN BLUM, MD
Director, Division of Management Services	GEORGE FORREST, JR
Assistant Director, Division of Governmental Affairs	JERRY S. HOFFMAN, ESQ
Director, Division of Communications	EDWARD HYNES
Director, Division of Insurance and Membership Benefits	BERNARD M. JACKSON
Director, Division of Membership	EUNICE SKELLY
Director, Division of Finance	PHILIP G. KEATING
Asst. Director, Division of Scientific Activities	ELIZABETH DAEPP
Director, Division of Advertising-Sales and Meeting Services	ROBERT MILLER
General Counsel	DONALD R. MOY, JD
Director, Division of Computer Services	IVAN H. NAJMAN
Acting Director, Division of Medical Services	MARIO MENGHINI
Director, Division of Long-Range Planning	DEBORAH R. SIEGEL
Consultant	EDWARD SIEGEL, MD

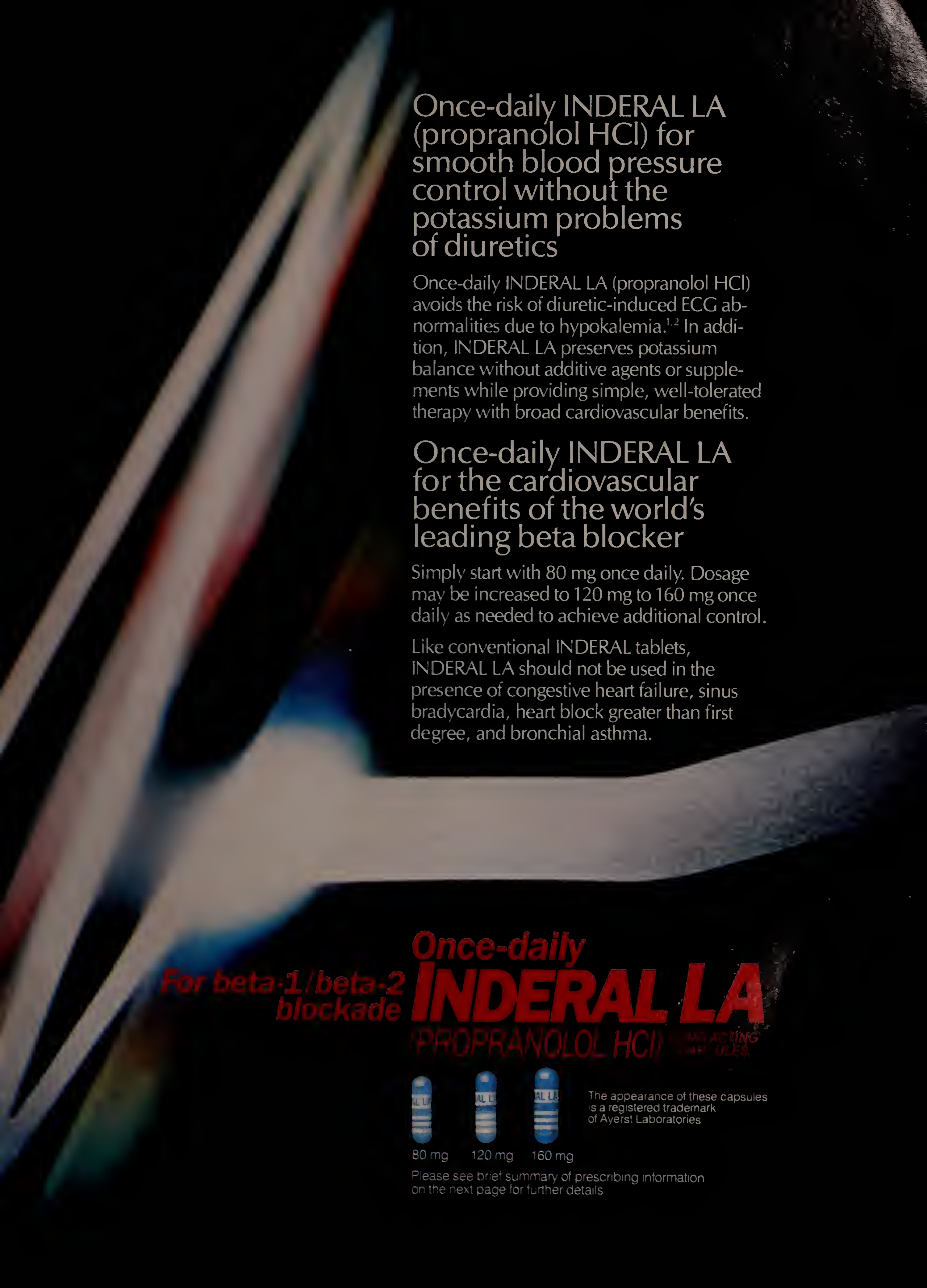
The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. Editorial material published in the *Journal* is covered by copyright. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

Right from the start in hypertension...



Once-daily INDERAL LA (propranolol HCl) for smooth blood pressure control without the potassium problems of diuretics

Once-daily INDERAL LA (propranolol HCl) avoids the risk of diuretic-induced ECG abnormalities due to hypokalemia.[1,2] In addition, INDERAL LA preserves potassium balance without additive agents or supplements while providing simple, well-tolerated therapy with broad cardiovascular benefits.

Once-daily INDERAL LA for the cardiovascular benefits of the world's leading beta blocker

Simply start with 80 mg once daily. Dosage may be increased to 120 mg to 160 mg once daily as needed to achieve additional control.

Like conventional INDERAL tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.

For beta-1/beta-2 blockade

Once-daily

INDERAL LA

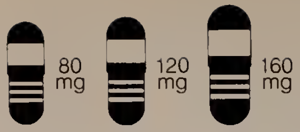
(PROPRANOLOL HCl) LONG-ACTING CAPSULES

80 mg 120 mg 160 mg

The appearance of these capsules is a registered trademark of Ayerst Laboratories

Please see brief summary of prescribing information on the next page for further details

Once-daily
For beta-1/beta-2 blockade
INDERAL® LA
(PROPRANOLOL HCl) LONG ACTING CAPSULES



The appearance of these capsules
is a registered trademark
of Ayerst Laboratories

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use Effects on plasma volume appear to be minor and somewhat variable INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction. Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential The significance of the membrane action in the treatment of arrhythmias is uncertain

The mechanism of the antimigraine effect of propranolol has not been established Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm

Propranolol is not significantly dialyzable.

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE continued use of beta blockers can in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General:* Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure

Clinical Laboratory Tests Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy Pregnancy Category C INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose

There are no adequate and well-controlled studies in pregnant women INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman

Pediatric Use Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular. bradycardia, congestive heart failure, intensification of AV block, hypotension, paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type

Central Nervous System lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances, hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics

Gastrointestinal nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory bronchospasm

Hematologic agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION—*Dosage must be individualized* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic The dosage may be increased to 120 mg once daily or higher until adequate blood pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

REFERENCES

1. Holland OB, Nixon JV, Kuhnert L: Diuretic-induced ventricular ectopic activity. *Am J Med* 1981,70:762-768. **2.** Holme I, Helgeland A, Hjermann I, et al Treatment of mild hypertension with diuretics. The importance of ECG abnormalities in the Oslo study and in MRFIT. *JAMA* 1984,251:1298-1299.

9411/1184

Ayerst®
AYERST LABORATORIES
New York, N Y 10017

Copyright © 1984 AYERST LABORATORIES
Division of AMERICAN HOME PRODUCTS CORPORATION

Classified Advertising

PHYSICIANS WANTED

MEDICAL DIRECTOR — EMERGENCY DEPARTMENT: Small community hospital located approximately 60 miles south of Rochester has an immediate position available for an emergency department Director. Responsibilities combine clinical and administrative functions. Post graduate training in a primary care area and emergency department experience required. Administrative experience desirable but not necessary. This career position offers an annual competitive income, director's stipend, paid professional liability insurance, paid health and life insurance benefits, plus more. For details contact Jean McGinty, Spectrum Emergency Care, Inc., P.O. Box 27352, St. Louis, MO 63141; 1-800-325-3982; 314-878-2280.

ANESTHESIOLOGIST: Upstate New York, Acute Medical/Surgical Hospital with active medical staff looking for Board Eligible or Certified Anesthesiologist. Excellent quality of life and setting. Interested candidates should send resume and economic expectations to Dept. 387 c/o NYSJM.

STAFF POSITIONS for Primary Care Physicians available at HMO Clinic Settings in Miami-Ft. Lauderdale Area. Attractive Salary, benefits and settings. Call or write Health-Med Systems, Inc., 19337 N.W. 2nd Ave., Miami, Fla. 33169. Phone (305) 651-8830.

DALLAS/FORT WORTH NEEDS PHYSICIANS. Full-time physician positions for general practice/internal medicine clinics. Partnership available in one year. Excellent opportunity. Write or call S. K. Kechejian, M.D., 609 S. Main St., Duncanville, TX 75116 or call (214) 780-0093.

PSYCHIATRISTS: Staff psychiatrists welcome. Excellent opportunity for building a practice. Call Nassau Hospital, Mineola, Long Island, New York, Department of Psychiatry at (516) 663-2691. Please ask for Nancy Yngstrom.

BOARD CERTIFIED INTERNIST: Preventive & Occupational Health—a leader in field offers varied opportunities (full-time, half-time or per diem) at our clinics and corporate medical units. Send C.V. to Executive Health Examiner, 777 Third Ave., N.Y., N.Y. 10017, Attn: Personnel Director N. [EOE M/F].

PHYSICIAN — EMERGENCY DEPARTMENT: Modern, well-equipped, mid-sized community hospital located one hour south of Buffalo has position available for an emergency department staff physician. Position requires some emergency department experience and two years post graduate training in a primary care specialty. This is a career opportunity for a physician dedicated to a career in emergency medicine. Competitive guaranteed income, paid professional liability insurance and other incentives provided. For details contact Jean McGinty, Spectrum Emergency Care, Inc., P.O. Box 27352, St. Louis, MO 63141; 1-800-325-3982; 314-878-2280.

PHYSICIANS WANTED—CONT'D

PRACTICE OPPORTUNITY: Family Practitioner, well established, multi-specialty group, with a developing Family Practice Department, is looking for a Family Practitioner for its satellite offices. Competitive salary. Liberal fringe benefits. Please send inquiry and Curriculum Vitae to Executive Director, Slocum-Dickson Medical Group P.C., 430 Court Street, Utica, NY 13502. Telephone area code 315-797-0500.

PHYSICIAN FOR CHILDREN'S SUMMER CAMP JULY AND/OR AUGUST 1985. Camp located at Beach Lake, Pennsylvania, accommodates 350 campers, ages 6–16. Complete modern health center; 2 R.N.'s in attendance. Will accept one M.D. for each month, no children accepted who are of camp age. Camp opens June 29th and closes August 23rd. Private room and facilities. Write to: Trail's End Camp, c/o Beach Lake, Inc., 215 Adams Street, Brooklyn, NY 11201. Please include your phone number.

ASSISTANT DIRECTOR, Department of Emergency Medicine in 950 bed voluntary hospital with 25,000 ER visits per year. Major affiliate of SUNY/Buffalo. BC/BE in IM, FM, EM or Surgery required. Teaching and research interests encouraged. Emergency service experience strongly preferred. Excellent opportunity in reorganized setting with developing trauma program. Position available July 1, 1985. Address inquiries to: Michael F. Noe, M.D., Vice President for Professional Affairs, Buffalo General Hospital, 100 High Street, Buffalo, N.Y. 14203 (716-845-2872). An Equal Opportunity Employer.

SOUTHERN CALIFORNIA: Prestigious HMO is seeking experienced specialists and general practitioners for our facilities in Los Angeles and Orange Counties. Located in close proximity to major teaching centers, we offer the opportunity for continued professional development and rewarding clinical practice. Excellent compensation and benefits including paid malpractice, life, disability, medical and dental coverage, paid vacations, sick leave, educational leave and retirement plan. Please send C.V. to: Director/Physician Recruitment, CIGNA Healthplans of California, 700 N. Brand Blvd., Suite 500-38, Glendale, CA 91203.

PRACTICES WANTED

YOUNG INTERNIST, wishes to purchase Nassau County office based or nursing home based private practice. Available immediately. Strictly confidential. Reply Dept. 365, c/o NYSJM.

PRACTICES AVAILABLE

RETIRING PHYSICIAN will sell building, practice and equipment in Queens Village, Queens, New York. Call evenings 8 to 10 P.M. (516) 627-5692.

FOREST HILLS, Internist retiring from long established practice. Completely equipped office in doorman building. Good location, will introduce. Reply Dept. 388 c/o NYSJM.

VIABLE FAMILY PRACTICE, home office, well equipped with x-ray, EKG, etc. North shore of Long Island. Call (516) 427-2180.

Don't let your lungs go to pot.

Smoking marijuana is a lot more dangerous than you think. And a lot less cool.

Recent studies show that pot can do a lot more damage to your lungs than anyone ever thought. Especially if you're young.

So, if you smoke, or if you're thinking about it, think again.

AMERICAN LUNG ASSOCIATION®
The Christmas Seal People®

Space contributed by the publisher as a public service

PRACTICES AVAILABLE—CONT'D

SELLING PRACTICES IS OUR ONLY BUSINESS—Want to maximize the return on your investment? Before you buy or sell, call for a consultation to assure the best financial and transfer terms. We guide you through the entire sales process from initial meeting to closing. Serving 100 mile radius of New York City. Countrywide Business Brokerage, Inc., 319 East 24th Street, Suite 23-G, New York, NY 10010 (212) 686-7902.

INTERNAL MEDICINE practice for sale. Well-established in Nassau County. Home-office, fully equipped for one or two physicians. Telephone (516) 747-3072.

FOR SALE LONG ESTABLISHED GENERAL PRACTICE with two story residence and attached office. Corner plot, separate entrances and addresses. Nassau County, New York, South Shore. Many extras, will introduce. Reply Dept. 380 c/o NYSJM.

EQUIPMENT

WANTED: a good fluoroscopic screen in place of my old one. Prof-Xray screen needed 12 in. x 12 in., Direction 11824A, Code 10-3050. Urgent! Call (607) 756-9941, collect.

ONLY DALMANE®
flurazepam HCl/Roche
IMPROVES SLEEP...

WITH
COMPLETE
SLEEP LABORATORY
PROOF[1-5]...

Copyright © 1984 by Roche Products Inc. All rights reserved

IN ALL THESE WAYS

- Rapid sleep onset[1-6]
- Effective for middle-of-the-night and early-morning awakenings[2,5]
- Undiminished efficacy for at least 28 nights[2-4]
- Patients usually awake rested and refreshed[7-9]
- Avoids rebound insomnia after discontinuation of therapy[3,5,10-12]

Caution patients about driving, operating hazardous machinery or drinking alcohol during therapy. Limit dose to 15 mg in elderly or debilitated patients. Contraindicated during pregnancy.

AND THE PREDICTABILITY THAT COMES WITH EXPERIENCE

FOR EFFECTIVE RELIEF OF INSOMNIA

DALMANE® Ⓘⓥ

flurazepam HCl/Roche

STANDS APART

15-MG/30-MG CAPSULES

See next page for references and summary of product information.

eferences: **1**. Kales J *et al. Clin Pharmacol*
ter 12:691-697, Jul-Aug 1971 **2**. Kales A *et al.*
'in Pharmacol Ther 18:356-363, Sep 1975.
Kales A *et al Clin Pharmacol Ther* 19
76-583, May 1976. **4**. Kales A *et al: Clin Phar-*
acol Ther 32:781-788, Dec 1982. **5**. Frost JD Jr,
eLucchi MR. *J Am Geriatr Soc* 27:541-546, Dec
979. **6**. Kales A, Kales JD. *J Clin Pharmacol*
140-150, Apr 1983. **7**. Monti JM: *Methods Find*
cp Clin Pharmacol 3:303-326, May 1981.
Greenblatt DJ *et al: Sleep* 5 (Suppl 1):S18-S27,
982. **9**. Kales A *et al: Pharmacology* 26:121-137,
983. **10**. Greenblatt DJ, Allen MD, Shader RI.
'in Pharmacol Ther 21:355-361, Mar 1977.
1. Zimmerman AM: *Curr Ther Res* 13:18-22,
n 1971. **12**. Amrein R *et al: Drugs Exp Clin Res*
1):85-99, 1983.

almane® Ⓒ
razepam HCl/Roche

efore prescribing, please consult complete product infor-
ation, a summary of which follows:

dications: Effective in all types of insomnia characterized by
ficulty in falling asleep, frequent nocturnal awakenings and/
early morning awakening; in patients with recurring insom-
or poor sleeping habits; in acute or chronic medical situa-
ns requiring restful sleep. Objective sleep laboratory data
ve shown effectiveness for at least 28 consecutive nights of
ministration. Since insomnia is often transient and intermit-
nt, prolonged administration is generally not necessary or rec-
mmended. Repeated therapy should only be undertaken with
propriate patient evaluation.

ntraindications: Known hypersensitivity to flurazepam HCl;
egnancy. Benzodiazepines may cause fetal damage when
ministered during pregnancy. Several studies suggest an
reased risk of congenital malformations associated with ben-
diazepine use during the first trimester. Warn patients of the
tential risks to the fetus should the possibility of becoming
egnant exist while receiving flurazepam. Instruct patient to
continue drug prior to becoming pregnant. Consider the pos-
ility of pregnancy prior to instituting therapy.

arnings: Caution patients about possible combined effects
th alcohol and other CNS depressants. An additive effect
y occur if alcohol is consumed the day following use for
httime sedation. This potential may exist for several days
lowing discontinuation. Caution against hazardous occupa-
ns requiring complete mental alertness (*e.g.*, operating
chinery, driving). Potential impairment of performance of
h activities may occur the day following ingestion. Not rec-
mmended for use in persons under 15 years of age. Though
ysical and psychological dependence have not been reported
recommended doses, abrupt discontinuation should be
oided with gradual tapering of dosage for those patients on
dication for a prolonged period of time. Use caution in
ministering to addiction-prone individuals or those who might
rease dosage.

ecautions: In elderly and debilitated patients, it is recom-
nded that the dosage be limited to 15 mg to reduce risk of
ersedation, dizziness, confusion and/or ataxia. Consider
tential additive effects with other hypnotics or CNS depres-
nts. Employ usual precautions in severely depressed patients,
in those with latent depression or suicidal tendencies, or in
ose with impaired renal or hepatic function.

lverse Reactions: Dizziness, drowsiness, lightheadedness,
ggering, ataxia and falling have occurred, particularly in
lerly or debilitated patients. Severe sedation, lethargy, dis-
entation and coma, probably indicative of drug intolerance or
erdosage, have been reported. Also reported: headache,
artburn, upset stomach, nausea, vomiting, diarrhea, constipa-
n, GI pain, nervousness, talkativeness, apprehension, irrita-
ity, weakness, palpitations, chest pains, body and joint pains
d GU complaints. There have also been rare occurrences of
ikopenia, granulocytopenia, sweating, flushes, difficulty in
using, blurred vision, burning eyes, faintness, hypotension,
ortness of breath, pruritus, skin rash, dry mouth, bitter taste,
cessive salivation, anorexia, euphoria, depression, slurred
eech, confusion, restlessness, hallucinations, and elevated
OT, SGPT, total and direct bilirubins, and alkaline phospha-
e; and paradoxical reactions, *e.g.*, excitement, stimulation
d hyperactivity.

osage: Individualize for maximum beneficial effect. *Adults:*
) mg usual dosage. 15 mg may suffice in some patients.
lerly or debilitated patients: 15 mg recommended initially
til response is determined.

pplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

ROCHE® Roche Products Inc
Manati, Puerto Rico 00701

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meetings and lectures unless the information is submitted at least two months prior to the event.*

JUNE 1985

AROUND THE STATE

MANHATTAN

June 1–2. **Pediatric Urology for the Practicing Urologist.** 12 Cat 1 credits. *June 3–5.* **Obstetrical Ultrasound.** 18 Cat 1 credits. *June 17–21.* **Emergency Medicine 1985.** 29 Cat 1 credits. *June 24–27.* **Pediatric & Adolescent Medicine.** 22 Cat 1 credits. Info: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

June 2. **New Frontiers in Pediatric Rheumatology.** 6 Cat 1 credits. Info: Elizabeth C. Gerst, PHD, Continuing Education Center, 630 W 168th St, New York, NY 10032. Tel: (212) 694-3682.

June 3, 4. **Care of the Patient with Amyotrophic Lateral Sclerosis.** 12 Cat 1 credits. *June 16–20.* **Rhinoplasty & Otoplasty.** 40 Cat 1 credits. *June 21, 22.* **Face Lift Surgery.** 14 Cat 1 credits. *June 24, 25.* **The Morris Feldstein Seminar on Cosmetic Surgery in the Aging Eye.** 14 Cat 1 credits. Mount Sinai Medical Center. Info: Page & William Black Post-Graduate School of Medicine, One Gustave L. Levy Place, New York, NY 10029. Tel: (212) 650-6737.

June 7–9. **CME Assembly—Health Care Today.** New York Hilton. Info: George J. Lawrence, Jr, MD, Medical Society of the State of New York, 420 Lakeville Rd, Lake Success, NY 11042. Tel: (516) 488-6100.

June 11. **ENT Pathology.** 2 Cat 1 credits. Info: Victor Tchertkoff, MD, Dept of Pathology, Metropolitan Hospital Center, 1901 First Ave, New York, NY 10029. Tel: (212) 360-6253.

June 14–15. **Breast Preservation: Workshop on the Technique of Conservative Surgery and Radiotherapy for Early Breast Cancer.** 9 Cat 1 credits. Info: CME Conference Planning Office, C-180, Memorial Sloan-Kettering Cancer Center, 1275 York Ave, New York, NY 10021. Tel: (212) 794-6754.

June 14–15. **Clinical Approaches to Arterial Blood Gas Interpretation and Electrolytes.** Info: Registrar, American College of Physicians, 4200 Pine St, Philadelphia, PA 19104. Tel: (215) 243-1200.

June 19. **Carcinogenesis and Host Response in Gynecologic Cancer.** Mount Sinai Medical Center. Info: Page & William Black Post-Graduate School of Medicine, One Gustave L. Levy Pl, New York, NY 10029. Tel: (212) 650-6737.

BROOKLYN

June 4. **Anesthesia for Thoracic Surgery.** *June 5.* **Management of the Patient with Coronary Artery Disease for Cardiac Surgery & Non-Cardiac Surgery.** Info: SUNY Downstate Medical Center, Dept of Anesthesiology, Box #6, 450 Clarkson Ave, Brooklyn, NY 11203. Tel: (718) 270-1233.

SUFFOLK

June 22. **Hypertensive Heart Disease, Comprehensive View of Clinical Aspects.** 6 Cat 1 credits. Info: Donna C. Joslin, CME, Southampton Hospital, 240 Meeting House LN, Southampton, NY 11968. Tel: (516) 283-2600, ext 505.

ERIE

June 9–22. **Methods of Immunologic Research and Diagnosis.** Buffalo. Info: James F. Mohn, MD, The Ernest Witebsky Center for Immunology, SUNY at Buffalo, 210 Sherman Hall, Buffalo, NY 14214. Tel: (716) 831-2848.

June 15. **Urology and Nephrology, Update 1985.** Amherst. 6 Cat 1 credits. Info: Rayna Saville, Pediatric Continuing Medical Education, Children's Hospital of Buffalo, 219 Bryant St, Buffalo, NY 14222. Tel: (716) 878-7630.

June 20. **Critical Care & Medical Management of the Cancer Patient.** Info: Gayle Bersani, RN, CME, Roswell

continued on p 22A

BALANCED CALCIUM CHANNEL BLOCKADE!

CARDIZEM®
(diltiazem HCl)
balances potent coronary vasodilation with a low incidence of side effects

Low incidence of side effects
CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

Reduces angina attack frequency*
42% to 46% decrease reported in multicenter study.[1]

Increases exercise tolerance*
In Bruce exercise test,[2] control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes ($P<.005$).

CARDIZEM®
(diltiazem HCl)

THE BALANCED CALCIUM CHANNEL BLOCKER

Please see full prescribing information on following page. 2/84

---

*Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

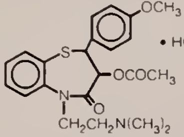
References:
1. Strauss WE, McIntyre KM, Parisi AF, et al: Safety and efficacy of diltiazem hydrochloride for the treatment of stable angina pectoris: Report of a cooperative clinical trial. Am J Cardiol 49:560-566, 1982.
2. Pool PE, Seagren SC, Bonanno JA, et al: The treatment of exercise-inducible chronic stable angina with diltiazem: Effect on treadmill exercise. Chest 78 (July suppl):234-238, 1980.

PROFESSIONAL USE INFORMATION

cardizem®
(diltiazem HCl)
30 mg and 60 mg tablets

DESCRIPTION

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one,3-(acetyloxy)-5-[2-(dimethylamino)ethyl]-2,3-dihydro-2-(4-methoxyphenyl)-, monohydrochloride,(+)-cis-. The chemical structure is:



Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform. It has a molecular weight of 450.98. Each tablet of CARDIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration.

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth muscle.

Mechanisms of Action. Although precise mechanisms of its antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways:

1. Angina Due to Coronary Artery Spasm: CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial and subendocardial. Spontaneous and ergonovine-induced coronary artery spasm are inhibited by CARDIZEM.
2. Exertional Angina: CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal and maximal exercise work loads.

In animal models, diltiazem interferes with the slow inward (depolarizing) current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Diltiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance.

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricular conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load. Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect; cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of diltiazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by diltiazem.

Intravenous diltiazem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Diltiazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, diltiazem significantly prolongs sinus cycle length (up to 50% in some cases).

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Diltiazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40%. CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 60 mg are given; a 120-mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem.

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm.** CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina).** CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS).

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia.
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing.
Cardiac Failure	Administer inotropic agents (isoproterenol, dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

The oral/LD_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort acute anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy**—CARDIZEM may be safely coadministered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

Cardizem 30-mg tablets are supplied in bottles of 100 (NDC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1771-49). Each green tablet is engraved with MARION on one side and 1771 engraved on the other. CARDIZEM 60-mg scored tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1772-49). Each yellow tablet is engraved with MARION on one side and 1772 on the other.

Issued 4/1/84

Another patient benefit product from

PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC
KANSAS CITY, MISSOURI 64137

1985 ANNUAL CME ASSEMBLY

HEALTH CARE TODAY

Medical Society of the State of New York
New York Hilton, New York City

FRIDAY, JUNE 7

8:00 a.m. - 10:00 a.m.	Meet the Professor Breakfast Meetings (5)
10:00 a.m. - 1:00 p.m.	Cardiovascular Diseases, *Beekman Parlor*
	Pediatrics/Dermatology, *Sutton Parlor North*
	Obstetrics/Gynecology, *Sutton Parlor South*
	Physical Medicine & Rehabilitation, *Sutton Parlor Center*
	Neurosurgery, *Regent Parlor*
2:00 p.m. - 5:00 p.m.	Neurology, *Sutton Parlor Center*
	Medical Legal & Workers' Compensation/Occupational Medicine, *Beekman Parlor*
	Cancer Update, *Sutton Parlor North*
	Chest Diseases, *Sutton Parlor South*
	Computers in Medicine, *Regent Parlor*

SATURDAY, JUNE 8

10:00 a.m. - 1:00 p.m.	Ophthalmology (All day), *Sutton Parlor North*
	Urology/Pathology, *Sutton Parlor Center*
	Internal Medicine (All day), *Beekman Parlor*
	General Surgery/Orthopedic Surgery/Hand Surgery/Gastroenterology Anesthesiology/Radiology, *Sutton Parlor South*
	Psychiatry/Alcoholism (All day), *Regent Parlor*
2:00 p.m. - 5:00 p.m.	Ophthalmology, *Sutton Parlor North*
	Otolaryngology — Head and Neck Surgery, *Sutton Parlor Center*
	Emergency Medicine, *Sutton Parlor South*
	Internal Medicine, *Beekman Parlor*
	Psychiatry/Alcoholism, *Regent Parlor*

SUNDAY, JUNE 9

8:00 a.m. - 10:00 a.m.	Meet the Professor Breakfast Meetings (5)
10:00 a.m. - 1:00 p.m.	Allergy & Immunology, *Beekman Parlor*
	Women's Medical Society of New York State/Family Practice, *Sutton Parlor Center*
	The Physician as a Witness, *Sutton Parlor North*

◆

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis.

◆

Technical Exhibits

General Information

The Medical Society of the State of New York's 1985 Annual CME Assembly sessions and exhibits are open to all physicians, their guests, and the allied professions. The two and one-half days of meetings will provide the individual physician with a possible 18 credits out of a total of 93 Category 1, CME credit hours being offered. Over 135 outstanding speakers, including a number from out-of-state, will present papers at 30 sessions.

On Friday, June 7th and Sunday, June 9th, five sessions of Meet the Professor will be held simultaneously. These two-hour sessions will include a continental breakfast.

The theme of the CME sessions will be *"Health Care Today"*. Some highlights of this year's sessions are, the Developments in the Relationship between Diet and Cancer; Update on Pacemakers; Computer Applications in Clinical Medicine; the Physician as a Witness; and today's concepts of Chest Diseases diagnosis and management. Our program will offer the clinician new approaches in diagnosis and treatment of diseases in a wide range of medical specialties. Don't miss this opportunity to gain Category 1 credits over a weekend when you are free from office hours. This year there is no registration fee for member physicians which makes it an economical way to brush up on your skills. Professional representatives from more than 70 companies will be in the Rhinelander Gallery Exhibit Hall to discuss their products and services. Technical Exhibits will include drug products, clinical laboratories, office equipment, computer services, educational and financial services.

COURTESY N.Y. CONVENTION & VISITORS BUREAU

PULITZER FOUNTAIN AT GRAND ARMY PLAZA

For the first time since 1979, the CME Assembly will be combined with the Annual Meeting of the House of Delegates, and the Auxilliary to the Medical Society.

SPECIAL EVENTS

Thursday, June 6: The Annual Dinner Dance will be held in the Trianon Ballroom of the New York Hilton, in honor of Allison B. Landolt, M.D., President, preceded by a reception in the Mercury Ballroom at 7:00 p.m. All attendees are welcome. Tickets should be ordered in advance from the Medical Society.

Friday, June 7 and Saturday June 8: A reception for physicians, the allied professions, and exhibitors will be held on both days in the Exhibit Hall, between 5:00 p.m. and 6:00 p.m., compliments of the Medical Society of the State of New York.

HOTEL RESERVATIONS

Physicians must make their own reservations with the New York Hilton no later than May 16, 1985. If making reservations by telephone, please mention that you are attending the Medical Society Convention for billing at the special rate.

REGISTRATION

CME Registration will take place in the Rhinelander Gallery Exhibit Hall, located on the second floor of the hotel and accessible by escalators from the lobby. Registration will take place at the following hours:

Friday, June 7th–
7:30 a.m.-5:00 p.m.
Saturday, June 8th–
8:00 a.m.-5:00 p.m.
Sunday, June 9th–
7:30 a.m.-12:30 p.m.

House of Delegates Registration will take place on the third floor, West Promenade, at the following hours: Wednesday, June 5 from 12 noon-7:30 p.m.; Thursday, June 6 from 8:30 a.m.-5:30 p.m.; Friday, June 7 from 8:30 a.m.-5:30 p.m.; Saturday, June 8 from 8:30 a.m.-5:30 p.m.; and Sunday, June 9 from 8:30 a.m.-12 noon.

CME MEETING HOURS

Friday, June 7th–
Meet the Professor–
8:00 a.m.-10:00 a.m.
All other sessions–
10:00 a.m.-1:00 p.m.
2:00 p.m.-5:00 p.m.

Saturday, June 8th–
All sessions–
10:00 a.m.-1:00 p.m.
2:00 p.m.-5:00 p.m.

Sunday, June 9th–
Meet the Professor–
8:00 a.m.-10:00 a.m.
All other sessions–
10:00 a.m.-1:00 p.m.

Meet the Professor – June 7th and 9th

at the Medical Society of the State of New York's Annual CME Assembly, June 7-9, 1985, the New York Hilton, New York City. Five continental breakfast meetings, 2 hours each, will be offered on Friday and Sunday mornings from 8:00 a.m.-10:00 a.m. Sessions are limited to 30 attendees each, and there is a $35.00 fee.

Hollander

Itskovitz

Lebovitz

Louria

Leevy

Aberman

FRIDAY, JUNE 7

Charles Hollander, M.D.

Professor of Medicine & Chief of Endocrinology, New York University School of Medicine, New York City. To provide a very brief review of our understanding of the physiology and pathophysiology of thyroid, pituitary and hypothalamic disorders.

Harold D. Itskovitz, M.D.

Professor of Medicine & Pharmacology, New York Medical College, Valhalla, New York. To familiarize the physician with new drugs used to treat hypertension; to stress changing concepts concerned with etiology, work-up and management of hypertension.

Harold E. Lebovitz, M.D.

Chief, Endrocrinology & Metabolism, SUNY, Downstate Medical Center, Brooklyn, New York. To provide an understanding of the pathophysiology of the two major types of diabetes, and to develop a rational set of goals for the treatment of patients with diabetes.

Donald Louria, M.D.

Professor & Chairman, Department of Preventive Medicine & Community Health, New Jersey Medical School, College of Medicine & Dentistry of New Jersey, Newark, New Jersey. To develop a programatic approach to the seriously ill patient with a putative infection.

Carroll Leevy, M.D.

Professor & Chairman, Department of Medicine, New Jersey Medical School, College of Medicine & Dentistry of New Jersey, Newark, New Jersey. Immunologic abnormalities are key factors in pathogenesis of hepatitus due to infections, alcoholism, and drug induced toxicity. Prevention, diagnosis and treatment have been greatly facilitated by recent advances in genetics, availabilty of monoclonal antibodies and the use of recombinant D.N.D. (Deoxyribonucleic Acid) technology.

SUNDAY, JUNE 9

Arnold Aberman, M.D.

Chief of Medicine, Mt. Sinai Hospital, University of Toronto Faculty of Medicine, Toronto, Canada. To approach the fluid and electrolyte management in the hospitalized patient with particular emphasis on hypo and hypernatremia.

Continued on next page

Meet the Professor - continued

Michael H. Grieco, M.D.

Director, R.A. Cooke Institute of Allergy, St. Luke's-Roosevelt Hospital Center, New York City. A review of current concepts concerning the immunologic and nonimmunologic mechanisms involved on pathogenesis of bronchial asthma, and review the management of ambulatory asthmatics.

Grieco

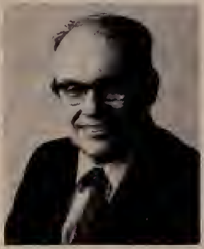
Kantor

Thomas Kantor, M.D.

Professor of Medicine; Chief, Division of Rheumatology, New York University School of Medicine, New York City. This session will demonstrate the all-purpose analgetic activity of the nonsteroidal, anti-inflammatory drugs in addition to their antiarthritic activity.

Simon Pollack, M.D.

Professor of Medicine, Albert Einstein College of Medicine, Bronx, New York. The objective of this presentation will be to explain current concepts of iron absorption and utilization, and the diagnosis and treatment of iron deficiency and iron overload.


Pollack

Rebuck

Anthony Rebuck, M.D.

Professor of Medicine, University of Toronto Faculty of Medicine, Toronto, Canada. Physicians attending this session will be made familiar with the proper use of oxygen in the every day care of caridac and pulmonary patients.

NEW YORK STATE
JOURNAL OF MEDICINE

May 1985 • Volume 85, Number 5

COMMENTARIES

Practical aspects of immunohistochemistry

The differential diagnosis of spindle cell malignancies poses a problem for the surgical pathologist. Accurate determination of cell type, frequently a prerequisite to therapy, may not be possible by routine light microscopy alone. Even a broad categorization of such tumor types as epithelial, mesenchymal, a mixture of both, or hematopoetic may be uncertain. Some poorly differentiated carcinomas and melanomas that lack obvious pigment may demonstrate striking spindle cell features in histologic section, thus making them indistinguishable from mesenchymal tumors, including sarcomas. Also, "large cell lymphomas" (histiocytic lymphomas in the Rappaport system) may be confused with poorly differentiated carcinomas, amelanotic melanomas, or sarcomas.

Recognizing that light microscopic appearances may be subjective and that histochemical staining (with phosphotungstic acid hematoxylin, for example) may not be specific, Reddy et al[1] relied on ultrastructural characteristics to support the diagnosis of ovarian leiomyosarcoma, as reported in this issue of the *Journal*. Electron microscopy, however, requires appropriately fixed tissue, expensive equipment, and specially trained personnel. For practical reasons, only a very small sample of tumor can be studied, thus introducing the potential for sampling errors. The authors apparently did not employ immunohistochemical techniques in the evaluation of this unusual ovarian lesion. Nevertheless, the application of immunohistochemical methods to the surgical pathologist's collection of "special stains" is suitable to such diagnostic problems and offers several advantages over electron microscopy.

The development of somatic cell hybridization techniques made possible the production of monoclonal antibodies against a variety of cell constituents and cell products. The successful demonstration of immunoglobulin in routinely processed tissue using the immunoperoxidase method[2] suggested that at least some "antigens" could survive fixation, embedding, and staining procedures and thus provided the impetus for the development of an evergrowing number of specific antisera, many of which are commercially available today.

A review of all antigenic markers used in the study of tumors constitutes much of the current literature on surgical pathology and is far beyond the scope of this commentary. However, it is of interest to focus on some markers that might be helpful in the evaluation of a spindle cell malignancy, be it in the ovary as reported by Reddy et al[1] or in bone as reported by Patanaphan et al,[3] both in this issue. These markers include intermediate filaments, carcinoembryonic antigen, S-100 protein, common leukocyte antigen, and estrogen receptors. These antibodies are most valuable when used as a panel rather than individually.

The cytoskeleton of animal cells is formed by a heterogeneous group of fibrillar proteins, collectively known as intermediate filaments.[4] Although similar in ultrastructure, measuring approximately 10nm in diameter, these proteins differ in molecular composition and configuration. Hence, they can be distinguished with immunologic tests, and specific antisera can be raised against each of the several subclasses of filaments: keratins, desmin, vimentin, neurofilament, and glial filament.[5] Keratins are a family of at least 19 polypeptides (ranging in molecular weight from 40 to 67 kd) present in various combinations in most epithelial cells.[6] Although they are most often found dispersed within the cytoplasm of epithelial cells,[7] these keratin filaments aggregate to form tonofilaments that can be identified by electron microscopy.[8] Thus, when compared to electron microscopy, immunohistochemical techniques provide a more sensitive method for the identification of epithelial cells. Recently, characteristic protein profiles of keratins with different molecular weights have been described in association with various stages of epidermal maturation and with different epithelia.[9-11] Keratins have been demonstrated in spindle cell carcinomas,[12] but with very few exceptions, such as synovial sarcomas and epithelioid sarcomas, they are absent from sarcomas and lymphomas.

Although at present keratins may be one of the most useful subclasses of intermediate filaments in surgical pathology, staining for vimentin and desmin, both commercially available as antisera, can provide complementary and supplementary information in tumor typing. Vimentin is the major intermediate filament of mesenchymal cells.[5] No subclasses of this protein have been described to date. Vimentin has been reported in sarcomas, melanomas, and hematopoietic tumors.[13] Thus, demonstration of vimentin in tumor cells of a spindle cell malignancy speaks against a diagnosis of spindle cell carcinoma. The expression of vimentin is not completely understood, however. Evidence is accumulating to suggest that under certain conditions (in tissue culture and body cavity fluids, for example) epithelial cells may express vimentin as well as keratin.[14] Desmin, similar in molecular weight to vimentin, and often coexpressed with it, is associated with differentiation of mesenchymal cells toward muscle.[15] Although present in large amounts in smooth as well as striated muscle cells, this protein is apparently absent from epithelial and mesothelial cells. Its usefulness is limited by the demonstration of desmin in some non-muscle sarcomas.[12] The specificity of antisera to neurofilament and glial filaments is somewhat uncertain at present.[16] Nevertheless, when testing for these proteins with the immunoperoxidase method, one can expect negative findings for carcinomas, leiomyosarcomas, melanomas, and lymphomas.

S-100 protein is an acidic protein, so named because of its solubility in 100% ammonium sulfate.[17] This protein has been demonstrated in a variety of lesions, including melanomas, granular cell tumors, some salivary gland tumors, and nerve sheath tumors.[16] Since most amelanotic and spindle melanomas yield positive results when stained for S-100 protein, whereas most spindle cell carcinomas and sarcomas are negative, S-100 protein may be helpful in certain differential diagnoses.

Antibodies to common leukocyte antigen (often designated as T200 antigen) are directed against a group of high molecular weight glycoproteins present in hematopoetic cells.[18] Positive staining for common leukocyte antigen suggests that a lesion is hematopoetic in origin and virtually eliminates undifferentiated carcinoma and sarcoma from diagnostic consideration.[19]

Carcinoembryonic antigen, originally considered a specific marker for adenocarcinomas of the gastrointestinal tract, is now recognized as being far less specific, but is useful as a marker for cells of epithelial origin.[20]

With appropriate modifications, immunohistochemical methods can be applied to fresh frozen or fixed embedded tissue. When the latter is used, the immunoperoxidase technique offers several advantages over alternative methods: no special equipment is needed; slides are reviewed using a routine light microscope; excellent histologic detail is preserved; accepted criteria can be applied to sections; permanent slide preparations are obtained; retrospective studies can be performed as long as several years after tissue embedding; and, finally, serial sections of tumor can be tested or studied with a panel of antisera as illustrated above.

The application of immunohistochemical methods to tumor diagnosis is in its infancy. Among the limitations in the use of these techniques are variation in results with different fixation methods, cross-reactivity of antisera, variation in antibody titer with different lots of antisera, and incomplete understanding of the factors that influence antigen expression. In the absence of meticulous controls, interpretation of immunoperoxidase staining is unreliable. Further, one must remember that none of the antisera available can be used to distinguish benign from malignant lesions.

Primary ovarian leiomyosarcoma accounts for only a small number of the mesenchymal lesions of the ovary. Metastatic or secondary involvement of the ovary (most often from a uterine or gastrointestinal lesion) must be excluded before a primary leiomyosarcoma of the ovary is diagnosed. Extraovarian leiomyosarcomas occur more frequently and may be difficult to distinguish from primary ovarian neoplasms.[21] Similarly, leiomyosarcomas that arise in the broad ligament[22] or paraovarian vessels[23] may masquerade as primary ovarian tumors. Despite advances in diagnosis, the histogenesis of these tumors remains uncertain and the prognosis is usually poor.

Patanaphan et al[3] report the use of large doses of radiation therapy to achieve long-term control of leiomyosarcoma that has metastasized to bone. Leiomyosarcoma is not considered a radiosensitive neoplasm and, although the reported findings are encouraging, one case requires the support of controlled studies before any conclusions can be drawn. On occasion, immunohistochemistry has been helpful in planning tumor therapy. Although most of this work concerns estrogen and progesterone receptor analysis in breast tumors, hormonal manipulation of other tumors—whether they be non-resectable or metastatic—may be attempted if in vitro receptor assays are positive.

ANN E. WALTS, MD
Department of Anatomic Pathology
Cedars-Sinai Medical Center
8700 Beverly Blvd.
Los Angeles, CA 90048

1. Reddy SA, Poon TP, Ramaswamy G, Tchertkoff V: Leiomyosarcoma of the ovary. *NY State J Med* 1985; 85:218-220.
2. Taylor CR: Immunoperoxidase techniques: theoretical and practical aspects. *Arch Pathol Lab Med* 1978; 102:113-121.
3. Patanaphan V, Hafiz MA, Salazar OM: Metastatic leiomyosarcoma of iliac bone. *NY State J Med* 1985; 85:220-222.
4. Franke WW, Schmid E, Osborn M, Weber K: Different intermediate-sized filaments distinguished by immunofluorescence microscopy. *Proc Natl Acad Sci USA* 1978; 75:5034-5038.
5. Lazarides E: Intermediate filaments as mechanical integrators of cellular space. *Nature* 1978; 283:249-256.
6. Moll R, Franke WW, Schiller DL, Geiger B, Krepler R: The catalog of human cytokeratins: patterns of expression in normal epithelia, tumors, and cultured cells. *Cell* 1982; 31:11-24.
7. Taxy JB, Battifora H, Hidvegi D: Nasopharyngeal carcinoma. Electron microscopy and a comparison of three antikeratin antisera. *Lab Invest* 1984; 50:59a.
8. Franke WW, Schmid E, Grund C, Geiger B: Intermediate filament proteins in nonfilamentous structures: transient disintegration and inclusion of subunit proteins in granular aggregates. *Cell* 1982; 30:103-113.

9. Banks-Schlegel SP: Keratin alterations during embryonic epidermal differentiation: a presage of adult epidermal maturation. *J Cell Biol* 1982; 93:551–559.

10. Tseng SC, Jarvinen MJ, Nelson WG, Huang JW, Woodcock-Mitchell J, Sun TT: Correlation of specific keratins with different types of epithelial differentiation: monoclonal antibody studies. *Cell* 1982; 30:361–372.

11. Sun TT, Eichner R, Nelson WG, et al: Keratin classes: molecular markers for different types of epithelial differentiation. *J Invest Dermatol* 1983; 81:109s–115s.

12. Battifora H: Recent progress in the immunohistochemisty of solid tumors. *Sem Diagn Pathol* 1984; 1:251–271.

13. Denk H, Krepler R, Artlieb U, et al: Proteins of intermediate filaments. An immunohistochemical and biochemical approach to the classification of soft tissue tumors. *Am J Pathol* 1983; 110:193–208.

14. Virtanen I, Lehto VP, Lehtonen E, et al: Expression of intermediate filaments in cultured cells. *J Cell Sci* 1981; 50:45–63.

15. Miettinen M, Lehto VP, Badley RA, Virtanen I: Alveolar rhabdomyosarcoma: demonstration of the muscle type of intermediate filament protein, desmin, as a diagnostic aid. *Am J Pathol* 1982; 108:246–251.

16. Clark HB: Immunohistochemistry of nervous system antigens: diagnostic applications in surgical neuropathology. *Sem Diagn Pathol* 1984; 1:309–316.

17. Moore BW: A soluble protein characteristic of the nervous system. *Biochem Biophsy Res Commun* 1965; 9:739–744.

18. Omary MB, Trowbridge IS, Battifora HA: Human homologue of murine T200 glycoprotein. *J Exp Med* 1980; 152:842–852.

19. Pizzolo G, Sloane J, Beverley P et al: Differential diagnosis of malignant lymphoma and nonlymphoid tumors using monoclonal anti-leukocyte antibody. *Cancer* 1980; 46:2640–2647.

20. Corson JM, Pinkus GS: Mesothelioma: profile of keratin proteins and carcinoembryonic antigen: an immunoperoxidase study of 20 cases and comparison with pulmonary adenocarcinomas. *Am J Pathol* 1982; 108:80–87.

21. Ng KH, Prathap K: Intestinal leiomyosarcoma simulating malignant ovarian neoplasm. *Int Surg* 1975; 60:237–238.

22. Raj-Kumar G: Leiomyosarcoma of probable ovarian or broad ligament origin. *Br J Obstet Gynecol* 1982; 89:327–329.

23. Honore LH, Robins RE, Taylor RH: Leiomyosarcoma of the right ovarian vein, a case report. *Angiology* 1977; 28:285–288.

Ureteropyeloscopy

Ureteropyeloscopy has expanded the reach of the endoscopic urologist into the ureter, renal pelvis, and even the intrarenal collecting system. Whereas these anatomic regions were previously accessible only by radiologic evaluation or open surgical exploration, they can now be examined using endoscopic techniques.

The indications for ureteropyeloscopy have grown in number as experience with the procedure has increased. As Khuri et al[1] indicate in this issue of the *Journal,* rigid ureteropyeloscopy can be employed for diagnostic and therapeutic purposes. Certainly the diagnosis of urothelial lesions in the upper urinary tract, and possibly their treatment, can be undertaken with ureteropyeloscopy. Similarly, other abnormalities within the ureter that are inaccessible to cystoscopic manipulation from the level of the bladder can be inspected and manipulated under direct vision.

The major indication and use for rigid ureteropyeloscopy has been the treatment of urinary calculi. In the authors' series, 43 of 56 procedures were performed to remove ureteral calculi. My own experience indicates a similar distribution. The results in removing ureteral calculi have been good but have varied with the location of the calculus. The greatest success is achieved when the calculus is located in the distal ureter. Khuri et al[1] reported an overall success rate of 83% for retrieval of all calculi, and they were able to remove 95% of those located in the distal ureter. The addition of ultrasonic lithotripsy to ureteropyeloscopy has enabled the removal of any calculus in the ureter, regardless of its size.[2]

The results with ureteropyeloscopy surpass those involving cystoscopic stone manipulation in the ability to remove mid and distal ureteral calculi. Most urologists have limited cystoscopic manipulation to distal ureteral calculi that are below the pelvic brim and measure 8 mm or less in diameter. Successful removal, even with these limitations, has ranged from 57% to 80%.[3]

The complications associated with ureteropyeloscopy have been minimal. Mild self-limited hematuria has been common, and some lateral abdominal discomfort may also be seen. Although I have not yet encountered complications that required surgical intervention, there have been sporadic reports of broken instruments or strictures at the ureterovesical junction that required surgical correction.

Ureteropyeloscopy appears to complement other techniques for urinary stone removal. Percutaneous endourologic techniques provide ready access to the kidney, with successful removal of renal calculi. In comparison, ureteropyeloscopy is more successful in the ureter, particularly the distal portion. Extracorporeal shock wave lithotripsy is most beneficial for removing renal calculi and, possibly, upper to mid ureteral stones. One of the complications of this treatment has been obstruction of the ureter by passing fragments; these fragments may be amenable to ureteropyeloscopic removal.

There is an exciting potential for the future development of these techniques. More powerful and rapid techniques for fragmentation of calculi would be welcome additions. Technical advances may provide more reliable and more useful flexible instruments which can be placed into all portions of the intrarenal collecting system.[4] As endoscopic instruments and techniques develop, the removal of intrarenal calculi under visual control may become a reality.

DEMETRIUS H. BAGLEY, MD
Department of Urology
Jefferson Medical College of
Thomas Jefferson University
Philadelphia, PA 19107

1. Khuri FJ, Peartree RJ, Ruotolo RA, Valvo JR: Rigid ureteropyeloscopy: diagnostic and therapeutic indications. *NY State J Med* 1985; 85:205–206.

2. Huffman JL, Bagley DH, Schoenberg HW, Lyon ES: Transurethral removal of large ureteral and renal pelvic calculi using ureteroscopic ultrasonic lithotripsy. *J Urol* 1983; 130:31–34.

3. Drach GW: Stone manipulation. Modern usage and occasional mishaps. *Urology* 1978; 12:286–289.

4. Bagley DH, Huffman JL, Lyon ES: Combined rigid and flexible ureteropyeloscopy. *J Urol* 1983; 130:243–244.

Transurethral endoscopy of the upper urinary tract

Advances in endoscopic instrumentation and techniques enable diagnostic and surgical manipulation of the ureter and renal pelvis via transurethral routes. Certainly the concept of upper urinary tract endoscopy is not new to the medical field. However, the small-caliber, extended-length rigid and flexible endoscopes that have recently been developed facilitate routine and safe access into the ureter and kidney. These instruments are used for diagnostic and surgical procedures, including transurethral removal of ureteral and renal pelvic calculi, transurethral biopsy, fulguration, and, occasionally, resection of urothelial tumors in the upper urinary tract. Most ureteropyeloscopic procedures have been performed with rigid endoscopes[1]; flexible ureteropyeloscopes have been difficult to manufacture at the small diameter needed to traverse the ureter and yet maintain an accessory channel and a deflecting mechanism.

The endoscopic techniques employed in transurethral ureteropyeloscopy are not new to the urologist. The methods of instrument insertion, stone manipulation, and endoscopic biopsy are similar to those employed in cystoscopic procedures, and they can be translated directly to the upper urinary tract. As with any endoscopic technique, skill, patience, and good judgement are essential for success.

It must be emphasized that transurethral ureteropyeloscopy is not 100% successful. Anatomic variations in the lower or upper urinary tract may preclude the insertion of a rigid ureteropyeloscope to the level of the renal pelvis. However, as reported by many authors, including Khuri et al[2] in this issue of the *Journal*, more than 90% of male and female patients can tolerate the procedure. Refinements in flexible instrumentation may allow a greater percentage of successful examinations.

The greatest impact of transurethral urteropyeloscopy has been on the non-surgical removal of calculi in the upper urinary tract. Transurethral removal of stones in the upper urinary tract, once thought to be "off limits," is now possible with ureteropyeloscopic methods.[3-5] Successful implementation of this method allows the patient rapid discharge from the hospital and a prompt return to full activity. Oversized calculi—those too large to be extracted safely down the ureter—are fragmented prior to removal using ultrasonic lithotripsy; this technique can also be applied to ureteropyeloscopy.

The success rates of ureteropyeloscopic stone removal vary according to the position of the stone in the ureter. Those in the lower ureter can be removed in more than 90% of patients, whereas those in the proximal ureter can be removed in approximately 50% of patients.[6] Extracorporeal shock wave lithotripsy (ESWL)[7] and percutaneous nephrolithotomy techniques[8] are good methods for removing renal and upper ureteral stones. However, lower ureteral stones remain prime candidates for ureteropyeloscopic removal, since stones in this location cannot be approached by ESWL and are subject to relatively low rates of successful percutaneous removal.[9]

Epithelial tumors in the upper urinary tract often pose a diagnostic challenge. Unlike tumors in the bladder, which are diagnosed using cystoscopic techniques, a similar endoscopic method for diagnosing tumors in the upper urinary tract did not exist. As noted by Khuri et al,[2] ureteropyeloscopy is an effective tool for obtaining a closed, transurethral biopsy and then planning the appropriate surgical treatment. These techniques may be especially useful in identifying patients who could benefit from a renal-sparing treatment. As with cystoscopic examinations of the bladder, routine surveillance of the upper tract can be performed. Low-grade, localized tumors of the upper tract might be treated by endoscopic methods; however, more information is required regarding the accuracy of diagnosis and patient follow-up before this can become an acceptable therapeutic option.[10]

The complications associated with transurethral ureteropyeloscopy are minor. In a review by Sosa et al[11] of 838 ureteropyeloscopic procedures reported at the Third International Society of Urologic Endoscopy, there was a complication rate of 4.5%, less than 10% of which required surgical exploration. The high rate of success without significant complications is attributable to dilation of the ureteral orifice and intramural ureter prior to instrument insertion, the ability to visualize the passage of the instrument, balloon dilation of narrow regions in the ureter, and the use of ultrasonic lithotripsy techniques when extracting oversized calculi. There are many methods available for ureteral dilation, but those using a dilator passed over a pre-positioned guide wire appear to be the most satisfactory. There are several types of dilators: olive-shaped metal bougies, teflon-coated fascial dilators, and balloon dilating catheters similar to those employed for vascular dilation. Thus far, there is no experimental or clinical evidence of long-term complications associated with ureteral dilation for ureteropyeloscopy.[12]

Transurethral ureteropyeloscopy has expanded the endoscopic capabilities of the urologist. It is a safe and reliable procedure that has opened the entire urinary tract to endoscopic visualization. In regard to non-surgical stone removal, this technique appears to complement ESWL and percutaneous nephrolithotomy. Although many preliminary studies regarding the use of the ureteropyeloscope have been encouraging, its ultimate role in the field of urology is yet to be determined.

JEFFRY L. HUFFMAN, MD
Urology Service
Memorial Sloan-Kettering
Cancer Center
1275 York Ave
New York, NY 10021

1. Perez-Castro Ellendt E, Martinez-Pinciro JA: Transurethral ureteroscopy—a current urological procedure. *Arch Esp Urol* 1980; 33:445-460.
2. Khuri FJ, Peartree RJ, Ruotolo RA, Valvo JR: Rigid ureteropyeloscopy. *NY State J Med* 1985; 85:205-206.
3. Das S: Transurethral ureteroscopy and stone manipulation under direct vision. *J Urol* 1981; 125:112-113.
4. Goodman TM: Ureteroscopy in the management of distal ureteral disease.

J Urol 1984; 132:250-253.
5. Ford TF, Watson GM, Wickham JEA: Transurethral ureteroscopic retrieval of ureteric stones. *Br J Urol* 1983; 55:626-628.
6. Huffman JL, Bagley DH, Lyon ES: Transurethral ureteroscopy and pyeloscopy, in *Urologic Endoscopy—A Manual and Atlas*. Boston, Little, Brown and Co, 1984, pp 185-206.
7. Chaussey C, Schmiedt E, Jocham D, Brendel W, Forssmann B, Walther V: First clinical experience with extracorporeally-induced destruction of kidney stones by shock waves. *J Urol* 1982; 127:417-420.
8. Segura JW, Patterson DE, LeRoy AJ, McGlough PF, Barrett DM: Percutaneous removal of kidney stones. *Mayo Clin Proc* 1982; 57:615-619.
9. Clayman RV, Surya V, Miller RP, et al: Percutaneous nephrolithotomy: extraction of renal and ureteral calculi from 100 patients. *J Urol* 1984; 131:868-871.
10. Huffman JL, Morse MJ, Bagley DH, Herr HW, Lyon ES, Whitmore WF Jr: Endoscopic diagnosis and treatment of upper tract urothelial tumors—a preliminary report. *Cancer* 1985; 55:1422-1428.
11. Sosa RE, Vaughan ED Jr, Huffman JL: Complications of ureteropyeloscopy, in Huffman JL, Bagley DH, Lyon ES (eds): *Transurethral Ureteropyeloscopy*. Philadelphia, W B Saunders, 1985.
12. Ford TF, Parkinson MC, Wickham JEA: Clinical and experimental evaluation of ureteric dilatation. *Br J Urol* 1984; 56:460-463.

Long-term survival with metastatic uterine sarcoma

Due to the existence of low-grade variants of most sarcomas of uterine origin, interpretation of clinical trials and case reports involving patients with these sarcomas is often difficult. The most common uterine sarcoma is the malignant mixed müllerian tumor, a neoplasm for which a low-grade variant has not been identified. For other types of uterine sarcomas, however, low-grade variants display a natural history different from the aggressive course normally exhibited by uterine sarcomas. Endometrial stromal sarcoma, for example, may appear as a low-grade variant, endolymphatic stromal myosis. Five-year survival for patients with stage III endolymphatic stromal myosis is 100%,[1] in contrast with no survivors among those patients with a high-grade stromal sarcoma.[2] In addition, the behavior of fibrosarcoma is different from its low-grade counterpart, the desmoid tumor.[3]

Patients with leiomyosarcomas that have spread beyond the uterus rarely attain long-term survival, unless the tumors are of a very low grade. Neither the criteria for making the diagnosis of leiomyosarcoma nor the nomenclature used for these malignant smooth muscle neoplasms is clear. Kempson and Bari[4] have suggested the use of a mitotic count to differentiate tumors into truly malignant leiomyosarcomas and a lower-grade variant possessing less than five mitotic figures per 10 high power field. They characterize these as "smooth muscle tumors of uncertain malignant potential." Silverberg[5] believes that the mitotic count has prognostic significance but poor diagnostic specificity. He does not demand a specific number of mitoses for the diagnosis of sarcoma and suggests that even though a relationship may exist between mitotic index and outcome, this method is fallible and does not correlate as well as histologic grade with outcome.

Other investigators have used different terminology to designate a lower-grade smooth muscle sarcoma that is associated with long-term survival. Such terms as "benign metastasizing leiomyoma,"[6] "leiomyomatosis peritonealis disseminata,"[7] and the syndrome of multiple leiomyosarcoma[8] appear in the gynecologic literature.

Results of clinical trials using cytotoxic chemotherapy for metastatic sarcoma should take into consideration this variation in biologic behavior. Patients with metastatic leiomyosarcoma treated with vincristine, actinomycin D and cytoxan have shown a significantly prolonged survival (median = 19.3 months) when compared to patients with mixed mesodermal sarcoma (median = 9.27 months).[9] However, if patients with low-grade leiomyosarcomas, as determined by the criteria of Silverberg,[5] are not counted, the survival between the two groups of patients is no longer statistically significant. The survival advantage attained by patients with leiomyosarcoma can probably be attributed to the indolent natural history of these low-grade smooth muscle neoplasms, rather than to an increased sensitivity of this tumor to chemotherapy.

The role of radiation therapy in these patients is also not clear. When given in traditional doses as a sole method of therapy, pelvic irradiation is not effective for either local control or cure.[10] When used in patients with early disease, no survival advantage can be demonstrated with adjunctive radiation therapy, although there may be improved local control in the pelvis. Patients with small-volume metastatic sarcoma treated with a combination of radiation therapy plus chemotherapy have been observed to have prolonged survival when compared to a control group of patients.[11]

In this issue of the *Journal*, Patanaphan et al[12] report on a patient with prolonged survival following radiation therapy for a metastatic lesion in the iliac bone. In addition, this patient underwent surgical excision of a metastatic focus in the right thigh. The authors conclude that a radical course of radiation therapy should be offered to patients who have metastatic bone involvement without other evidence of sarcoma. Certainly radiation therapy would be one form of treatment available to a clinician confronted with such a patient. However, until results of clinical trials are available that stratify patients by grade, it is difficult to know if the prolonged survival seen with these patients is due to therapy or the indolent natural history of a low-grade variant of uterine sarcoma.

EDWARD V. HANNIGAN, MD,
Associate Professor
Division of Gynecologic Oncology
The University of Texas Medical Branch
Galveston, TX 77550

1. Piver MS, Rutledge FN, Copeland L, Webster K, Blumenson L, Suh O: Uterine endolymphatic stromal myosis: a collaborative study. *Obstet Gynecol* 1984; 64:173-178.
2. Peters WA, Kumar NB, Fleming WP, Morley GW: Prognostic features of sarcomas and mixed tumors of the endometrium. *Obstet Gynecol* 1984; 63:550-556.
3. Reitamo JJ: The desmoid tumor. IV. Choice of treatment, results, and

complications. *Arch Surg* 1983; 118:1318-1322.

4. Kempson RL, Bari W: Uterine sarcomas: classification, diagnosis and prognosis. *Hum Pathol* 1970; 1:331-349.

5. Silverberg SG: Leiomyosarcoma of the uterus. *Obstet Gynecol* 1971; 38:613-627.

6. Bachman D, Wolff M: Pulmonary metastases from benign-appearing smooth muscle tumors of the uterus. *Am J Roentgenol* 1976; 127:441-446.

7. Pearce PH: Leiomyomatosis peritonealis disseminata. *Am J Obstet Gynecol* 1982; 144:133-134.

8. Wood WG, Burch TK, Thomas Jr CY: Multiple leiomyosarcomas. *South Med J* 1977; 70:381-383.

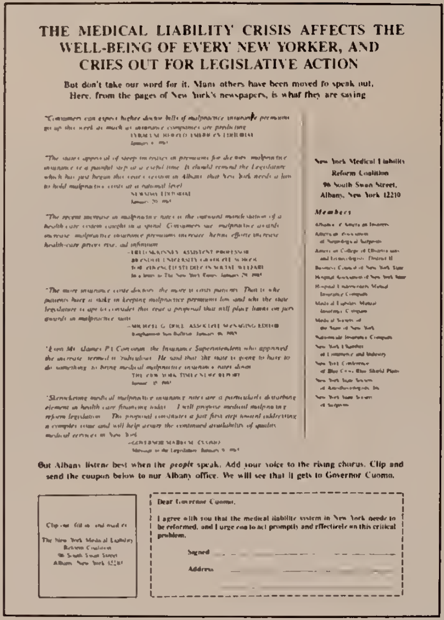
9. Hannigan EV, Freedman RS, Elder KW, Rutledge FN: Treatment of advanced uterine sarcoma with vincristine, actinomycin D, and cyclophosphamide. *Gynecol Oncol* 1983; 15:224-229.

10. Salazar OM, Bonfiglio TA, Patten SF, Keller BE, Feldstein M, Dunne ME, Rudolph JH: Uterine sarcomas: natural history, treatment and prognosis. *Cancer* 1978; 42:1152-1160.

11. Smith JP, Rutledge FN, Delclos L, Sutow W: Combined irradiation and chemotherapy for sarcomas of the pelvis in females. *Am J Roentgenol Radium Ther Nucl Med* 1975; 123:571-576.

12. Patanaphan V, Hafiz MA, Salazar OM: Metastatic leiomyosarcoma of iliac bone. *NY State J Med* 1985; 85:220-222.

MEDICAL GROUPS' ADS OPPOSE INSURANCE RATE HIKE



THE MEDICAL LIABILITY CRISIS AFFECTS THE WELL-BEING OF EVERY NEW YORKER, AND CRIES OUT FOR LEGISLATIVE ACTION

But don't take our word for it. Many others have been moved to speak out. Here, from the pages of New York's newspapers, is what they are saying

New York Medical Liability
Reform Coalition
96 South Swan Street,
Albany, New York 12210

Members

But Albany listens best when the *people* speak. Add your voice to the rising chorus. Clip and send the coupon below to our Albany office. We will see that it gets to Governor Cuomo.

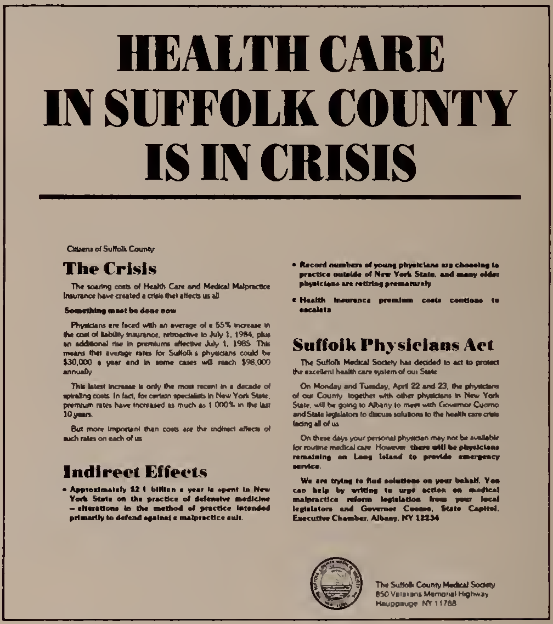
Clip out, fill in, and mail to
The New York Medical Liability
Reform Coalition
96 South Swan Street
Albany, New York 12210

Dear Governor Cuomo,

I agree with you that the medical liability system in New York needs to be reformed, and I urge you to act promptly and effectively on this critical problem.

Signed

Address



HEALTH CARE IN SUFFOLK COUNTY IS IN CRISIS

Citizens of Suffolk County

The Crisis

The soaring costs of Health Care and Medical Malpractice Insurance have created a crisis that affects us all.

Something must be done now

Physicians are faced with an average of a 55% increase in the cost of liability insurance, retroactive to July 1, 1984, plus an additional rise in premiums effective July 1, 1985. This means that average rates for Suffolk's physicians could be $30,000 a year and in some cases will reach $98,000 annually.

This latest increase is only the most recent in a decade of spiraling costs. In fact, for certain specialists in New York State, premium rates have increased as much as 1,000% in the last 10 years.

But more important than costs are the indirect affects of such rates on each of us.

Indirect Effects

- **Approximately $2.1 billion a year is spent in New York State on the practice of defensive medicine — alterations in the method of practice intended primarily to defend against a malpractice suit.**
- **Record numbers of young physicians are choosing to practice outside of New York State, and many older physicians are retiring prematurely.**
- **Health insurance premium costs continue to escalate.**

Suffolk Physicians Act

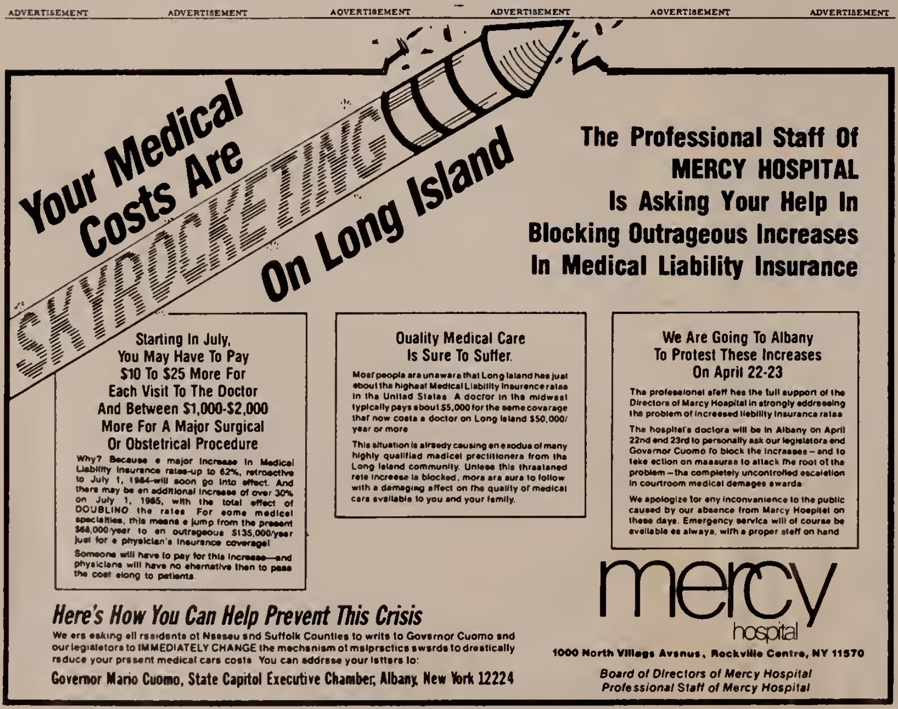
The Suffolk Medical Society has decided to act to protect the excellent health care system of our State.

On Monday and Tuesday, April 22 and 23, the physicians of our County, together with other physicians in New York State, will be going to Albany to meet with Governor Cuomo and State legislators to discuss solutions to the health care crisis facing all of us.

On these days your personal physician may not be available for routine medical care. However, **there will be physicians remaining on Long Island to provide emergency service.**

We are trying to find solutions on your behalf. You can help by writing to urge action on medical malpractice reform legislation from your local legislators and Governor Cuomo, State Capitol, Executive Chamber, Albany, NY 12234

The Suffolk County Medical Society
850 Veterans Memorial Highway
Hauppauge, NY 11788


ADVERTISEMENT ADVERTISEMENT ADVERTISEMENT ADVERTISEMENT ADVERTISEMENT ADVERTISEMENT
Your Medical Costs Are
SKYROCKETING
On Long Island

The Professional Staff Of MERCY HOSPITAL Is Asking Your Help In Blocking Outrageous Increases In Medical Liability Insurance

Starting In July, You May Have To Pay $10 To $25 More For Each Visit To The Doctor And Between $1,000-$2,000 More For A Major Surgical Or Obstetrical Procedure

Why? Because a major increase in Medical Liability Insurance rates—up to 62%, retroactive to July 1, 1984—will soon go into effect. And there may be an additional increase of over 30% on July 1, 1985, with the total effect of DOUBLING the rates. For some medical specialties, this means a jump from the present $68,000/year to an outrageous $135,000/year just for a physician's insurance coverage!

Someone will have to pay for this increase—and physicians will have no alternative than to pass the cost along to patients.

Quality Medical Care Is Sure To Suffer.

Most people are unaware that Long Island has just about the highest Medical Liability Insurance rates in the United States. A doctor in the midwest typically pays about $5,000 for the same coverage that now costs a doctor on Long Island $50,000/year or more.

This situation is already causing an exodus of many highly qualified medical practitioners from the Long Island community. Unless this threatened rate increase is blocked, more are sure to follow with a damaging affect on the quality of medical care available to you and your family.

We Are Going To Albany To Protest These Increases On April 22-23

The professional staff has the full support of the Directors of Mercy Hospital in strongly addressing the problem of increased liability insurance rates.

The hospital's doctors will be in Albany on April 22nd and 23rd to personally ask our legislators and Governor Cuomo to block the increases — and to take action on measures to attack the root of the problem — the completely uncontrolled escalation in courtroom medical damages awards.

We apologize for any inconvenience to the public caused by our absence from Mercy Hospital on these days. Emergency service will of course be available as always, with a proper staff on hand.

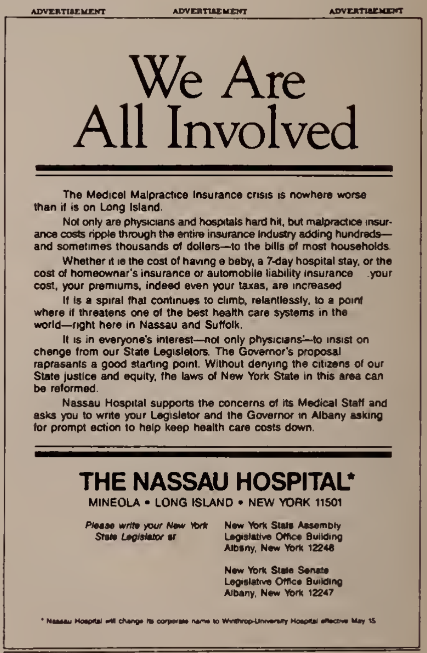
Here's How You Can Help Prevent This Crisis

We are asking all residents of Nassau and Suffolk Counties to write to Governor Cuomo and our legislators to IMMEDIATELY CHANGE the mechanism of malpractice awards to drastically reduce your present medical care costs. You can address your letters to:

Governor Mario Cuomo, State Capitol Executive Chamber, Albany, New York 12224

mercy hospital
1000 North Village Avenue, Rockville Centre, NY 11570

Board of Directors of Mercy Hospital
Professional Staff of Mercy Hospital



ADVERTISEMENT ADVERTISEMENT ADVERTISEMENT

We Are All Involved

The Medical Malpractice Insurance crisis is nowhere worse than it is on Long Island.

Not only are physicians and hospitals hard hit, but malpractice insurance costs ripple through the entire insurance industry adding hundreds—and sometimes thousands of dollars—to the bills of most households.

Whether it is the cost of having a baby, a 7-day hospital stay, or the cost of homeowner's insurance or automobile liability insurance ... your cost, your premiums, indeed even your taxes, are increased.

It is a spiral that continues to climb, relentlessly, to a point where it threatens one of the best health care systems in the world—right here in Nassau and Suffolk.

It is in everyone's interest—not only physicians'—to insist on change from our State Legislators. The Governor's proposal represents a good starting point. Without denying the citizens of our State justice and equity, the laws of New York State in this area can be reformed.

Nassau Hospital supports the concerns of its Medical Staff and asks you to write your Legislator and the Governor in Albany asking for prompt action to help keep health care costs down.

THE NASSAU HOSPITAL*
MINEOLA • LONG ISLAND • NEW YORK 11501

Please write your New York State Legislator at

New York State Assembly
Legislative Office Building
Albany, New York 12248

New York State Senate
Legislative Office Building
Albany, New York 12247

* Nassau Hospital will change its corporate name to Winthrop-University Hospital effective May 15.

Prior to an unprecedented physicians' rally organized by the Medical Society of the State of New York in Albany on April 23 to protest the extraordinary 55% increase in medical liability insurance premiums, the New York Medical Liability Reform Coalition purchased advertisements in newspapers throughout the state to appeal for legislative action. Long Island physicians, already hit with the highest insurance rates in the US, also sought public support.

LETTERS TO THE EDITOR

This section is designed to offer rapid publication (usually within 1-3 months) of new clinical, epidemiologic, and laboratory observations, as well as of comments on articles published in the Journal *or on any topic related to the role of the physician. Letters should be typed double-spaced and should include the professional affiliation, highest academic degree, address, and signature of each author. Preference is given to letters not exceeding 450 words. All letters are personally reviewed and acknowledged by the Editor. Address correspondence to Editor,* New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042.*

Neutropenia associated with procainamide

TO THE EDITOR: A report of neutropenia associated with sustained released procainamide (Procan SR®) appeared recently.[1] We report five additional cases with two fatalities.

Case Reports: **Case 1.** A 69-year old woman was treated with procainamide, theophylline, digoxin, furosemide, cimetidine, and thyroid replacement therapy following an acute inferior infarction complicated by ventricular tachycardia. Her complete blood cell count was normal. Twenty-eight days later she was admitted to the hospital with sepsis of undetermined etiology. Her leukocyte count was 500/mm^3 with only occasional segmented neutrophils and band forms found. The hematocrit was 30% and the platelet count was 420,000/mm^3. The result of an antinuclear antibody test was less than 1:20. Despite treatment with broad spectrum antibiotics, the patient died three days later. Postmortem bone marrow examination showed severe granulocytopenia.

Case 2. A 72-year-old man had coronary artery bypass grafting complicated by ventricular arrhythmia; he was treated with procainamide, digoxin, warfarin, propoxyphene, and flurazepam. His complete blood cell count was normal. Fifty-seven days later he was admitted with *Escherichia coli* septicemia. The leukocyte count was 2.2/mm^3 with 4% segmented neutrophils and no band forms. The hematocrit was 16% and the platelet count was 53,000/mm^3. An antinuclear antibody test was positive at 1:160, with a diffuse pattern. A bone marrow biopsy showed moderate hypocellularity, but was otherwise unremarkable. Despite antibiotic therapy, the patient died on the day after admission to the hospital.

Case 3. An 85-year-old woman was treated for frequent premature ventricular contractions with procainamide. Other medications included timolol, acetaminophen and fluorocortisone acetate. Her complete blood cell count was normal. Forty-one days later she was admitted to the hospital because of epistaxis and pseudomonas bacteriuria. A complete blood cell count showed a leukocyte count of 700/mm^3 with 5% segmented neutrophils and 4% band forms. The hematocrit was 30% and the platelet count was 4,000/mm^3. An antinuclear antibody test was positive at 1:80 with a diffuse pattern. A bone marrow biopsy revealed a slightly hypocellular bone marrow with markedly decreased granulopoiesis. The patient responded to treatment with ticarcillin and tobramycin. Upon discharge from the hospital, following the 14th hospital day, the leukocyte count was 3.7/mm^3 with 45% segmented neutrophils and 2% band forms. The hematocrit was 34%, and the platelet count was 470,000/mm^3.

Case 4. A 71-year-old woman had ventricular ectopy after porcine aortic valve replacement and was treated with procainamide, dipyridamole, low-dose aspirin, digoxin, furosemide, and potassium supplements. Her complete blood cell count was normal. She was admitted to the hospital with *E coli* septicemia six weeks after procainamide therapy was initiated. Her leukocyte count was 500/mm^3, with no segmented neutrophils or band forms. The hematocrit was 27%, and the platelet count was 325,000/mm^3. An antinuclear antibody test was positive at 1:640 with a diffuse pattern. After treatment with ticarcillin and tobramycin, the patient recovered. She was discharged from the hospital on the 16th hospital day with a leukocyte count of 5.2/mm^3, including 12% segmented neutrophils, seven band forms, 19 metamyelocytes, and seven myelocytes. The hematocrit was 31%, and the platelet count was 497,000/mm^3.

Case 5. A 71-year-old man with stage II bladder cancer had ventricular ectopy following acute myocardial infarction. During his hospital stay he received quinidine, cephalexin, flurazepam, lidocaine, and trimethoprim/sulfamethoxazole. Upon discharge from the hospital, his medications included procainamide, nifedipine, digoxin, furosemide, nitroglycerin, warfarin, and isosorbide dinitrate. His complete blood cell count was normal. Twenty-two days later he had an *E coli* urinary tract infection that was treated with trimethoprim/sulfamethoxazole; the leukocyte count was within normal limits. Seven days later procainamide and trimethoprim/sulfamethoxazole were stopped because of falling leukocyte counts, reaching a low value of 500/mm^3 with no neutrophils. Bone marrow aspiration showed severe granulocytopenia. An antinuclear antibody test was positive at 1:80 with a diffuse pattern. The leukocyte count returned to normal 17 days later.

Discussion: All of these patients had complicated cardiac disease and, therefore, had been treated with multiple drugs. Thus, drugs other than procainamide could be implicated as causing the severe granulocytopenia observed. Nevertheless, based on our experience as well as other recent reports, we no longer prescribe procainamide.

DAVID P. KIMMEL, MD
LOUIS V. ANTIGNANO, MD
S. SERGE BAROLD, MD
LASZLO BOROS, MD
Department of Medicine
The Genesee Hospital
224 Alexander St.
Rochester, NY 14607

JOHN P. OLSON, MD
Department of Medicine
Rochester General Hospital
1425 Portland Ave.
Rochester, NY 14621

---

[1] Ellrodt AG, Murata GH, Riedinger MS, Stewart ME, Mochizuki C, Gray R: Severe neutropenia associated with sustained-release procainamide. *Ann Intern Med* 1984; 100:197-201.

Symposium on Violence

TO THE EDITOR: I should like to compliment you in the highest terms on the recent issue of the *New York State Journal of Medicine* (February 1985). The article by Dr Abraham Blinderman and the commentary by Dr Bery Engebretsen express what must be the concern of every human being on earth who will permit himself or herself to reflect on the human aberration that is labeled war. The issues and the manner in which they were addressed in the *Journal,* I would hope would set a precedent for continued and repeated expression of opinions by the medical community on this issue.

The combined efforts of probably every physician in this country during a period of many years could not make up for the loss of productive human life snuffed out in one major battle, the Battle of Verdun. The medical fraternity should have the clearest insight into the counterfeit currency labeled "just war."

The issues are more vital than any other dealt with in any medical or surgical journal, bar none, in terms of the potential for preventing human misery and useless loss of life.

ANTHONY M. IMPARATO, MD
New York University Medical Center
530 First Avenue
New York, NY 10016

The Berwid case

TO THE EDITOR: Serious flaws mar Schwartz' (1985; 85:67–68) description of the Berwid murder. I was secretary of the Ad Hoc Committee to Defend the Pilgrim Doctors accused in the case.

A central omission was the decision by the New York State Office of Mental Health not to hold hearings on whether the doctors were guilty of misconduct, despite its highly publicized pledge immediately after the murder to hold disciplinary hearings within three weeks to dismiss the doctors and possibly to have their medical licenses revoked.

Despite the claim of the Office of Mental Health that Berwid was mentally irresponsible and that the Pilgrim doctors were therefore to blame, Berwid was ultimately tried for murder. Despite deliberate attempts to act crazy in the courtroom, some of which I observed, he was found guilty and was given the maximum sentence. This fact was also omitted from the account in the *Journal*.

It was one psychiatrist, not two, who was responsible for granting Berwid the off-grounds pass during which the murder occurred. The most likely explanation is that Berwid fooled him, and that most psychiatrists would also have been fooled under the same circumstances. The doctor may therefore have been "set up."

Berwid had been in the hospital for 18 months when given the pass, after having been transferred six weeks earlier from Mid-Hudson Psychiatric Center (a high security facility) to Pilgrim, thus implying that he was no longer dangerous. His behavior there was excellent, and he told the Pilgrim psychiatrist, who had not treated him earlier, that his threats many months before against his wife had been the product of an illness from which he had recovered. Mid-Hudson's experts on dangerousness had concurred. He also assured the psychiatrist, one of the most experienced and respected on the staff, that he would use only legal methods to get the children from his wife.

The second psychiatrist referred to in the *Journal*'s account was the first doctor's administrative supervisor. He had nothing to do with the clinical decision to issue the pass.

The State Commissioner of Mental Health told the press that the second psychiatrist had told the subordinate not to grant the pass. Had this been the case, or had the supervisor said it was the case, the subordinate could then have been brought up on charges of insubordination. But the supervisor would not lie, despite immense pressure on him, including anonymous threats against his life, which almost drove him to suicide.

The ward psychiatrist obviously erred in granting Berwid the pass, but there is no basis for Schwarz' assertion that he failed "to take proper steps in assessing the dangerousness of a patient." The statement erroneously implies medical misconduct rather than an honest mistake.

There was no obligation whatsoever under the "Tarasoff decision" for the doctors to notify anyone because they did not consider Berwid to be dangerous. The statement in the *New York Times* that Berwid had informed the doctors that he intended to kill his ex-wife, but that they had given him the pass anyway, was false.

The Berwid estate sued the State for responsibility for Mrs Berwid's death. I testified for the State. The estate won because staff members who had evidence of Berwid's possible dangerousness in the hospital had not conveyed it to the doctors, and because of administrative foul-ups, rather than because of possible physician error.

The most important lesson of this case is that criminals may feign insanity and then attempt to use that insanity

Utilization review mini quiz

This satirical quiz can be used to introduce attending and house staff physicians to concepts and principles of Utilization Review.

1. **Utilization Review refers to:**
   - a) The review of care to see if the patient required hospitalization
   - b) A committee checking quantity of food consumed in the cafeteria by physicians
   - c) A department which monitors use of Pampers in the newborn nursery
   - d) The review of a utility by the Nuclear Regulatory Commission
2. **Review coordinators perform the following tasks:**
   - a) Spy on physicians
   - b) Review cases according to criteria and report selected cases to physician advisors
   - c) Coordinate physicians' free time
   - d) Make medical decisions concerning your patients
   - e) Coordinate birth and death certificates to keep them equalized
3. **The role of physician advisor encompasses the following:**
   - a) Advises physicians concerning problems pertaining to their love lives
   - b) Writes discharge orders on all patients
   - c) Confers with physicians regarding problems relating to utilization, quality and risk and then backs down completely
   - d) Same as "C" but only backs down sometimes
   - e) Signs papers he or she hasn't read about patients he or she hasn't seen
4. **A "carve out" refers to:**
   - a) A sirloin steak dinner served at a Trustee barbecue
   - b) Custom woodworking on a nursing unit
   - c) A surgical procedure performed one day too late
   - d) A knife fight in the emergency room
   - e) A change in senior administration personnel
   - f) Denying payment for a day of care after the care has been rendered
5. **Alternate level of care:**
   - a) Refers to altered states of consciousness of some patients
   - b) A category of patients awaiting nursing home placement
   - c) When a floor of a hospital has a ramp leading to a slightly higher level
   - d) Plugging a cardiac pacemaker directly into alternate current
   - e) Turning a decubitus patient over every two hours

for exculpation. Years ago, Fredric Wertham, MD, described the "psychiatric hunting license" thus created. Sometimes, psychiatrists are blamed instead of the criminals.

The ploy failed with Berwid. It failed also with Alan Goodman, found guilty despite an insanity plea after killing several Arab worshippers on Jerusalem's Temple Mount on Easter Sunday, 1982. But it worked with John Hinckley, accused of attempting to assassinate President Reagan.

NATHANIEL S. LEHRMAN, MD
10 Nob Hill Gate
Roslyn, NY 11576

In reply: Dr Lehrman's letter addressed the Berwid case, a subject that warrants a paper in its own right. His statement that "criminals may feign insanity, and then attempt to use that insanity for exculpation" is, as a generalization, quite true. However, when he goes on to say, "The ploy failed with Berwid,"he demonstrates his misunderstanding of this case. Adam Berwid did just the opposite: He presented himself as "sane" for the purpose of being allowed to leave the hospital grounds, proceeded to kill his wife, and then refused to interpose the defense of lack of criminal responsibility as a result of mental disease or defect.

In regard to the issue of malpractice, it was not, as Dr Lehrman states, I who asserted that the ward psychiatrist had failed "to take proper steps in assessing the dangerousness of a patient." It was the court. Dr Lehrman's trial testimony notwithstanding, the court concluded that there had been "failures to properly examine Berwid, failures to adequately review his records, failures to obtain sufficient input from both hospital staff and others, failures to give adequate consideration to known information and observed signs and symptoms, and failures to record and report crucial observations and facts."

Space does not allow me to elaborate on these categories, but I would like to mention one: No attempt was ever made by the ward psychiatrist to have a meeting between Berwid and his former wife—the woman he repeatedly threatened to kill—under supervised conditions at the hospital before he was given his day pass. Under such a test situation, Berwid might have shown his murderous feelings and intentions, or he might have successfully concealed them. The point is that no attempt was ever made to assess his thoughts and feelings under circumstances that were still safe for Mrs Berwid. In the words of the court, such failures "to follow proper medical practice precluded any best judgment being made relative to Adam Berwid's temporary release." The case, it concluded, was a tragic example of an "avoidable mistake."

DANIEL W. SCHWARTZ, MD
Director
Forensic Psychiatry
Kings County Hospital Center
451 Clarkson Ave
Brooklyn, NY 11203

6. **Social workers are responsible for the following:**
    - a) Schedule social affairs for the hospital staff
    - b) Work as volunteers at the hospital center
    - c) Tracing the source of social diseases in the hospital
    - d) Helping to arrange appropriate discharge plans for patients
7. **A PRO is:**
    - a) A young woman who works in hotels
    - b) A PSRO with "S" carved out
    - c) A patient's religious official
    - d) A Peer Review Organization
    - e) An organization to encourage hospitals to transfer out patients who might die
8. **DRGs are:**
    - a) Patients addicted to prescription drugs
    - b) Yale University's only contribution to medical science
    - c) A way to turn used patients into instant cash
    - d) An opportunity for computers to really take over
    - e) A methodology of paying a hospital for the total care of a patient by diagnosis
9. **Peer review is:**
    - a) The concept that review of medical care is done by qualified physicians
    - b) A check of the ancestry of the British nobility
    - c) An endoscope with two eyepieces
    - d) A group of political physicians who review the work of practicing physicians
    - e) The organization which "a-peers" to the government to take the most carve outs
10. **Quality assurance is:**
    - a) Minding other people's business
    - b) Aiming for perfection if possible
    - c) Trying to find the cause of problems
    - d) Working with others to help the patient, the institution, and the physician
    - e) Required by the JCAH
    - f) All of the above

JOHN E. JACOBY, MD
CATHERINE GIORDANO, RN
Bronx-Lebanon Hospital Center
1650 Grand Concourse
Bronx, NY 10457

Esophageal ulceration associated with oral penicillin in Marfan's syndrome

TO THE EDITOR: There is a growing number of medications associated with esophageal erosion and ulceration.[1,2] Tetracycline derivatives have been implicated by more than one author.[1-5] Use of oral penicillin is ubiquitous. We recently observed a patient with esophageal ulceration due to an oral penicillin preparation.

Case Report: An 18-year-old woman with Marfan's syndrome and scoliosis received penicillin VK, 250 mg four times daily, for dental prophylaxis, beginning several days prior to the onset of symptoms. She took the tablets and adequate fluid with meals and at bedtime. On several different occasions the tablets seemed to stick in the esophagus, a sensation that was ignored and passed spontaneously. Five days before admission she experienced the abrupt onset of severe odynophagia, with both liquids and solids, in the lower retrosternal area. She denied any antecedant gastrointestinal symptoms. Physical examination was unremarkable. An esophagram showed extrinsic compression at the level of the aortic arch with a slight reduction of luminal caliber, and a similar compression at the level of the left bronchial crossing. The esophageal mucosa was unremarkable. A thoracic computed axial tomographic scan did not show an aortic aneurysm. One week following the onset of pain, esophagoscopy was performed and two irregularly shaped ulcers were seen 32 cm from the incisors. There was no evidence of stricture or hiatal hernia. Biopsies of the ulcers revealed squamous epithelium with acute inflammation. Brushings did not show evidence of herpes or fungi.

Discussion: The sensation of the tablets lodging in the retrosternal area and the onset of characteristic symptoms soon after beginning penicillin therapy suggest a cause and effect relationship between the medication and the esophageal ulcers. The region of the aortic arch has been suggested as a potential "hang up" for medication.[6] When medication is taken at bedtime, the subsequent recumbent position appears to delay esophageal transit.[7] Delayed passage of the tablets occurs more often in patients with esophageal motor disturbances.[8]

There have been no reports in the literature of esophageal abnormalities occurring with Marfan's syndrome, except for one coincidental case of achalasia.[9] However, it is possible that this patient's extrinsic compression due to scoliosis facilitated prolonged mucosal contact and subsequent ulceration. This is the second reported case of penicillin induced esophageal injury. However, in view of the frequency with which penicillin is used, this represents a rare complication. Adequate water following medication and maintenance of upright posture should be emphasized in all patients.

PERRY C. GOULD, MD
ROBERT S. BARTOLOMEO, MD
HOWARD M. SKLAREK, MD
Department of Medicine
Nassau Hospital
Mineola, NY 11501

1. Kirendall JW, Friedman AC, Oyewole MA, Fleisher D, Johnson LF: Pill-induced esophageal injury. *Dig Dis Sci* 1983; 28:174-182.
2. Mason SJ, O'Meara TF: Drug-induced esophagitis. *J Clin Gastroenterol* 1981; 3:115-120.
3. Collins FJ, Matthews HR, Baker SE, Strakova JM: Drug-induced oesophageal injury. *Br Med J* 1979; 1:1673-1676.
4. O'Meara TF: A new endoscopic finding of tetracycline-induced esophageal ulcers. *Gastrointest Endosc* 1980; 26:106-107.
5. Carlborg B, Densert O, Lindqvist C: Tetracycline induced esophageal ulcers. A clinical and experimental study. *Laryngoscope* 1983; 93:184-187.
6. Blackstone MO: *Endoscopic Interpretation. Normal and Pathologic Appearances of the Gastrointestinal Tract.* New York, Raven Press, 1984, p 30.
7. Hey H: Oesophageal transit of six commonly used tablets and capsules. *Br Med J* 1982; 285:1717-1719.
8. Evans KT, Roberts GM: Where do all the tablets go? *Lancet* 1976; 2:1237-1239.
9. Rapcport K, Bremner CG: Marfan's syndrome presenting with achalasia of the oesophagus. A case report. *S Afr Med J* 1981; 59:766-768.

Advances in the treatment of diabetes mellitus

TO THE EDITOR: My thanks to Dr Handelsman for his kind review of my book, *Diabetes: The Glucograf Method for Normalizing Blood Sugar* (1985; 85:49). In the article, he describes our use of small doses of ultralente insulin twice daily and small doses of regular insulin before meals. The preface to the new soft cover edition of the book (which was published in 1984, not 1981 as indicated in the review) advises the reader to substitute human lente insulin for the previously recommended beef-pork ultralente insulin. Furthermore, the preface advocates that the human lente be given at bedtime, not before supper. A significant number of patients using the ultralente insulin tended to run elevated blood sugars upon arising in the morning, even though their 3:00 am blood sugars were normal. This effect is well known in the field of diabetes and has been named the "Dawn Phenomenon." It appears to be correlated with the diurnal midnight peaking of growth hormone in the blood, followed six to nine hours later by increased hepatic clearance of circulating insulin. The use of human lente insulin seems to circumvent this phenomenon with resulting normal glycemia when the patient arises in the morning. It is interesting that we were not able to secure equivalent results with human NPH (neutral protamine Hagedorn) insulin or with animal lente insulin. This leads us to speculate that perhaps the more antigenic insulins are cleared more rapidly from the circulation by the liver. Human lente is the least antigenic of the long and intermediate acting insulins currently available in this country.

RICHARD K. BERNSTEIN, MD
516 W. Boston Post Road
Mamaroneck, NY 10543

AIDS: a disease of ancient Egypt?

TO THE EDITOR: We share Doctor Berken's[1] skepticism of the current dogma of newness and the ascription of a transmissible agent as the sole cause of acquired immune deficiency syndrome (AIDS).

As to the newness of AIDS, translations of the Papyrus Ebers from the ancient Egyptology literature repeatedly make reference to difficulties in the diagnosis of conditions under the names ĀĀĀ disease, uxedu-disease, and the uha-disease.[2] The interpretation of these diseases has been inconclusive among Egyptologists. The many remedies proposed for the ravages of ĀĀĀ—whether by itself or complicated by uxedu- or uha-disease—brand it as a scourge of first magnitude. ĀĀĀ is mentioned 50 times in four papyri (28 times in Papyrus Ebers, 12 times in Papyrus Berlin, 9 times in Papyrus Hearst, and once in the London papyrus).[3] The name ĀĀĀ is translated as "semen" or "poison."[4] Could this be documentation of AIDS, or more accurately its prototype, occurring in Egypt during the time of the pharaohs? Could this deadly and terrifying disease have caused or reenforced the ensuing homosexual taboo, decreasing the prevalence of anal intercourse and lowering the incidence of the disease and subsequent documentation until modern times?

As to etiology, we favor Dr Berken's theory of nontransmissible—hence, noninfectious—co-factors. AIDS occurs for the most part only in those who already suffer from immunosuppression.[5] Our thoughts in this regard evolved from earlier studies that demonstrated the immunosuppressive properties of secretions from the prostate and other male accessory sexual glands.[6,7] In brief, our hypothesis[5] suggests that access of immunocompetent cells to the allogeneic enzyme transglutaminase, which is abundant in semen and is present in antihemophiliac factor(s), is the possible common denominator responsible for immunosuppression in homosexuals and hemophiliacs.

Transglutaminases, as they occur in the blood plasma, are more familiarly known as factors XIII and XIIIa; factor XIII is present in varying amounts in factor VIII and other replacement therapy concentrates administered to patients with hemophilia. In most patients at risk for AIDS, the immunosuppressive factors could be acquired through oral and rectal deposition of semen, whereas in patients with hemophilia, they could be acquired through Factor VIII replacement therapy.

Therefore, as an alternative explanation to proponents of infectious transmission of AIDS, it is suggested that opportunistic infections and their sequelae, including Kaposi's sarcoma and other tumors prominent among AIDS patients, are not the principal offenders, but result from a combination of one's lifestyle and a physical state of immunodeficiency. Whereas, with hemophilia, infections and their sequelae are a consequence of immunosuppression resulting from infusion of antihemophiliac factors.

RICHARD J. ABLIN PHD
MAURICE J. GONDER, MD
Department of Urology
SUNY at Stony Brook
Stony Brook, NY 11794

RONALD S. IMMERMAN, MD
Cleveland Hts, OH 44106

1. Berken A: AIDS: Neither new nor transmissible? *NY State J Med* 1948; 84:440–441.
2. Bryan CP, Smith GE: The ĀĀĀ disease, in *Ancient Egyptian Medicine. The Papyrus Ebers.* Chicago, Ares Publishers Inc, 1924, pp 119–123.
3. Chalioungui P: Magic and medical science, in *Ancient Egypt.* Hodder and Stoughton, 1963.
4. VonDeines H, Westencorf W:*Wörterbuch der medizimischen Texte,* vol 7, part 1, Berlin, Akademie-Verlag, 1961, pp 129.
5. Ablin RJ: Acquired immune deficiency syndrome: toward a possible explanation of its etiology. *IRCS Med Sci* 1983; 11:766–767.
6. Ablin RJ: Immunologic properties of sex accessory tissue components, in Brandes D (ed): *Male Accessory Sex Organs. Structure and Function in Mammals.* New York, Academic Press, Inc, 1974, pp 433–467.
7. Ablin RJ, Bhatti RA, Bush IM, Guinan PD: Immunosuppression of cell- and serum-mediated tumour-associated immunity in prostatic cancer by human seminal plasma. *Eur J Cancer* 1980; 16:775–780.

Dr Stiles D. Ezell

TO THE EDITOR: I am writing in reference to a quotation by Dr Ezell—"Regulation and Licensing of Physicians in New York"—that was published in the *Journal* (1984; 84:607).

I do not wish to embarrass anyone but, as far as I remember, it was the same Dr Stiles D. Ezell who was convicted of having sold the New York State Board examination questions, before the examination was given, to a number of physicians in 1961.

PETER H. STERN, MD
The Burke Rehabilitation Center
785 Mamaroneck Avenue
White Plains, NY 10605

Dr Stern is correct. Dr Ezell pleaded guilty to unlawful acts in respect to medical licensing examinations and was convicted. His medical license was revoked by the Board of Regents in 1964 but was restored in 1969; he was also reinstated as a member of the Medical Society of the State of New York. We were unaware of these events when we selected the quotation and appreciate Dr Stern's having brought it to our attention.

—Editor

ERRATUM

On the contents page of the March issue of the *Journal,* the first author of the letter "Solitary nonparasitic liver cysts" (1985; 85:95–96) is Steven M. Keller, MD.

The American Medical Soccer Association has been established for the purpose of exchanging medical and general information concerning the most popular sport in the world. The association also hopes to sponsor competitive soccer events for its members. Information about membership may be obtained from Dr Noah S. Finkel, Northeastern Representative, American Medical Soccer Association, 205 E. Main St, Huntington, NY 11743.

Accidents in the skilled nursing facility

JACOB DIMANT, MD

ABSTRACT. Three hundred and ten accidents in a skilled nursing facility were reviewed. Accidents were not related to age, sex, the number or degree of acute or chronic illnesses, or medication use. Most patients were confused and disoriented. Accidents occurred most frequently at the bedside and peaked at hours of increased patient activity and staff shift changes. One third of all accidents were falls while walking, mostly attributed to factors related to the patients' ages and conditions. Thirteen percent were falls out of bed, and 5% were falls off a toilet or in the bathtub. One quarter were falls related to wheelchairs or chairs. Nine percent of accidents occurred while patients were being handled or transferred by a staff member. One third of accidents resulted in no injury, and 60% resulted in soft-tissue injury that required surgical intervention in 7% of injuries. The head and face area was involved in 33% of accidents. An additional 7% were serious injuries, including fractures and head trauma. Thus, 15% of accidents resulted in serious morbidity. Admission to the hospital was required in 4% of cases, and one patient died. A successful accident prevention program was instituted based on findings from this study.

(NY State J Med 1985; 85:202–205)

INTRODUCTION

Accidents are a major cause of illness and death among long-term care facility residents. Accidents frequently occur among the elderly because of a multitude of host factors (factors associated with the anatomic and psychologic changes that accompany aging). A particular group at risk are residents in skilled nursing facilities because many such residents have chronic and acute conditions and because of environmental factors unique to such institutions. Although host factors associated with accidents in the elderly are difficult to alter, environmental factors may be modified to help minimize risk. Accidents at the Prospect Park Nursing Home in Brooklyn, a skilled nursing facility, were studied to provide information about some of these factors. This information is important for the design of successful accident prevention programs in long-term care facilities.

METHODS

During 29 months, 310 accidents occurred involving 310 patients (216 women and 94 men). The accidents were investigated by the assistant director of nursing and the medical director and were discussed periodically at multidisciplinary patient care meetings. These meetings provided an understanding of accident causes and suggestions for possible preventive action. Accidents were analyzed for host factors including age, sex, ambulatory status, mental status, chronic illnesses, and the presence of precipitating acute illness. Particular attention was paid to the number and kind of medications victims were receiving and their potential role in precipitating an accident. Environmental factors surveyed included the location and time of the accident, the condition of equipment involved, staff action and presence at the time of occurrence, and a detailed description of the accident itself. Victims were examined by a physician immediately if indicated or within 24 hours. Appropriate diagnostic tests and care were administered, and the nature of the injury was recorded. This study focused on detailed analyses of patients who had accidents.

Because of a high turnover rate of patients in the facility, patients who had accidents were compared with all patients in the facility. However, the patient population was fairly homogeneous with regard to host factors such as illnesses, medication use, and ambulatory and mental status. Thus, it was not possible to define particular groups of residents who were at an increased risk of having an accident.

RESULTS

The age and sex of accident victims were comparable to the age and sex of all residents. The average number of diagnoses and number of medications (three per patient) were similar for accident victims and all other patients. The use of particular medications, including psychotropic and antihypertensive agents, was not judged to contribute directly to any accident. Of the 310 accident victims, 117 (38%) were ambulatory, 28 (9%) walked with assistance, 162 (52%) were confined to a wheelchair or geriatric chair, and three (1%) were confined to bed. This was similar to the distribution of all patients in the facility. The mental status of accident patients and all other patients was similar: 249 (80%) were disoriented and confused due to organic mental syndrome, 48 (16%) had a normal mental state, and 13 (4%) had occasional periods of confusion or disorientation. Acute illness was found to be the precipitating cause of accidents in six patients.

The majority of accidents occurred in patient's rooms (196, or 64%), while 44 (14%) occurred in dining rooms, 25 (8%) in bathrooms or toilets, 26 (8%) in public areas, seven (2%) outdoors, and 12 (4%) in unknown locations. The home has four nursing stations, each with an equal number of patients. The stations differ only in the location of dining rooms and some bathrooms. The number of accidents did not differ substantially among nursing stations with respect to accidents in patients' rooms, dining rooms, or public areas. However, in one nursing station there were 17 accidents in bathrooms, compared with one, four, and three in other stations. Accidents occurred during all hours residents were awake, with large

---

From the Prospect Park Nursing Home and the State University of New York-Downstate Medical Center, Brooklyn.

Address correspondence to Dr Dimant, 3245 Nostrand Ave, Brooklyn, NY 11229.

peaks corresponding to staff shift changes and smaller peaks corresponding to mealtimes and getting in and out of bed. Very few accidents occurred at night. During daytime hours, lighting in patients' rooms was adequate.

Accident types are summarized in Table I. The majority were falls (243, or 78%), mostly from a standing position. Forty-one accidents (13%) were falls out of bed (generally while patients were trying to climb over side rails). In many cases urine was found on the floor, indicating that patients may have been trying to get to the bathroom and either they did not call for help or staff members did not respond expeditiously. Falls involving wheelchairs accounted for 75 accidents (24%). Falls from toilets or in bathtubs accounted for 16 accidents (5%).

TABLE I. Accidents

Type	No. (%) of Patients Involved	
Fall	107	(36)
Fall while being handled	4	
Fall out of bed	35	(13)
Fall out of bed while being handled	6	
Fall out of wheelchair or geriatric chair	61	(24)
Fall out of chair while being handled	5	
Fall with wheelchair	9	
Fall off toilet or in bathtub	12	(5)
Fall off toilet or in bathtub while being handled	4	
Hit head on side rails of bed	9	(4)
Hit head while being transferred	3	
Soft tissue or head trauma	31	(13)
Caused by wheelchair parts	2	
While being transferred	5	
Burns	6	(2)
Hit by another patient	7	(2)
Miscellaneous	4	(1)

Head trauma from bed rails occurred in 12 accidents, three of them while the patient was in the process of transfer. Other soft-tissue or head trauma occurred in 38 instances (13%). There were six burns (usually minor) from spilled hot liquids. Seven accidents resulted from fights between patients, and there were four miscellaneous accidents.

It is particularly noteworthy that 27 accidents (9%) occurred while a patient was being handled by a staff member; and, in most cases, lack of appropriate transfer techniques or carelessness was involved. A few accidents, mostly falls from chairs, occurred while patients were left without appropriate restraint or supervision. The nature of injuries is shown in Table II. The majority of accidents (186) resulted in soft-tissue lacerations, abrasions, or hematomas. Of these, 22 required surgical intervention and suturing. Soft-tissue injuries involving the face and head occurred in 103 cases. Head trauma was severe enough to warrant a hospital stay in two additional patients, one of whom died of her injuries. A soft-tissue hematoma became infected in another patient who subsequently required admission to the hospital. Fractures resulted from 15 accidents (5%), including seven hip fractures. There were no detectable injuries in 97 accidents (32%). Thirteen patients (4%) had to be treated in the hospital, seven with hip fractures, two with other fractures or dislocations, two with severe head trauma (one of whom died), and one each with infected hematoma and gastrointestinal bleeding.

TABLE II. Injuries

Type	No. of Patients Involved
Soft tissue laceration, abrasion, hematomas, and others	186
Requiring sutures	22
Involving the head	103
Minor soft tissue burns	6
Serious head or scalp trauma	2
Hip fractures	7
Other fractures and dislocations	8
Cellulitis or hematoma	1
Epistaxis	2
Asphyxia due to food	1
No injury detected	97

A comprehensive accident prevention program, which addressed findings from this study, was instituted. The program included a discussion of findings, changes in staffing and supervision patterns, and intensive in-service education on transfer and handling techniques. Toward the latter part of the study, it was noted that more accidents were being reported and were reported more accurately. There was a reduction in the number of accidents, particularly those that occurred while handling and transferring patients. There was also a noticeable reduction in the number of accidents that occurred at the time of staff shift changes. A follow-up study is in progress to assess the effects of this in-service program.

DISCUSSION

Periodic analysis of accidents contributes to an understanding of the major host and environmental factors involved and helps in formulating an effective accident prevention program. A continuous analysis of accidents and a prevention program that utilizes accident data have been in effect at the Prospect Park Nursing Home since 1977. These have helped contain the rate of accidents to approximately 17 per 10,000 days. The rates in other institutions that care for the elderly were reported to be from 21 to 42.7 per 10,000 days. However, the population in these studies included different types of patients, such as those with acute illness who are more prone to accidents.[1,2]

An accident can be defined as an undesirable or unfortunate event, unintentionally caused and usually resulting in harm, injury, damage, or loss.[3] A distinction must be made between accidents and incidents; the latter are nonroutine, not necessarily unusual events of seemingly minor importance that do not result in substantial injury. It is important to educate staff members in the appropriate reporting of accidents and the distinction between accidents and incidents. Delay in reporting serious accidents may cause delay in medical care, whereas excessive reporting of routine events diverts the staff from performing more important tasks. For example, one patient was "found in bed with a fracture of the hip," according to one report. Obviously, a previous accident occurred that resulted in this injury and was not reported by the staff, presumably because of its seemingly minor importance.

Numerous host factors render the elderly prone to accidents. These include denial of limitations related to aging or misjudgment of capabilities; acute and chronic illness; arthritis; organic brain syndromes associated with confusion, disorientation, and loss of memory or concentration ability; decreased vision or hearing; impairment of balance; orthostatic hypotension; abnormalities of sensation, proprioception, and gait; muscle weakness; cardiovascular problems (particularly cardiac arrhythmias); cerebrovascular disease; "drop attacks" (sudden loss of muscle tone without loss of consciousness resulting in a fall); and medications, particularly tranquilizers.[4-8]

A correlation between accidents and acute or chronic illness was previously reported.[4-9] This study did not support a relationship between the number of chronic conditions and accidents, perhaps because the population was homogenous and most of the patients had a number of medical as well as organic mental conditions. Moreover, acute illness precipitated only six accidents (2%). This is in contrast to other reports suggesting that a greater number of accidents may be related to acute conditions, particularly acute changes in cardiac status[4] or cardiac arrhythmias.[5]

Contrary to the literature,[4,9] this study did not confirm a relationship between accidents and the number or type of medications used. Confusion and disorientation were not related to the use of medications. Psychotropic medications in this facility are used with great caution, and side effects are closely monitored to prevent lethargy. Although it is not always possible to assess the separate contribution of mental status change, chronic disease, or the use of medications in each accident, no accident was clearly related to the use of medications, and there was no evidence of a higher number of medications used by those patients who had accidents.

The lack of a relationship between accidents and acute or chronic disease and the use of medications suggests that in the long-term skilled nursing care facility, where all patients have multiple medical conditions and use multiple medications, other risk factors for accidents are more important. These include mental status, ambulatory status, and particularly environmental factors.

Most accidents occurred in patients who were confused and disoriented. A confused mental state has been shown to be associated with a higher risk of accidents in the elderly.[10] Ambulatory patients, most of whom were confused, were involved in 117 accidents compared with only 28 accidents in patients who walked with assistance or used assisting devices. Impairment of gait and posture in these patients, as well as their impaired mental status, contributed to falls.[6] Encouraging confused, ambulatory patients to use assisting devices or to request assistance might reduce the number of falls. The use of such devices has been found to decrease accident risk.[9]

Wheelchairs are a major site of accidents in long-term care facilities.[2,7,9] Wheelchair-related accidents included falls out of chairs due to a lack of or inappropriate placement of restraints, falls associated with transfer in and out of a chair (occasionally caused by an improperly secured chair or improperly applied brakes), fall of the entire chair, broken chairs, and cuts and bruises from sharp parts of the chairs. Wheelchairs are a major environmental risk factor, and staff members should be thoroughly familiar with appropriate techniques for handling and securing them, transferring patients to and from them, and using restraints while the patient is in a chair. In "The dangerous wheelchair," Hartigan[11] listed pressure ulcers, flexion deformities, and radial nerve paralysis as complications from the use of wheelchairs. It is apparent that accidents involving wheelchairs should be added to this list.

Environmental factors play an important role in accidents in long-term care facilities. These include architectural design and location of bathrooms, toilets, and dining areas; obstruction by furnishings and equipment; slippery and wet floors; broken or unfit equipment; and, most important, patterns of staffing and supervision. While the potential for correction of host factors in the skilled nursing facility population is limited, environmental factors can be controlled or changed in many instances. It is important to recognize factors common to all long-term care facilities, as well as those unique to each institution. Contributing factors can be recognized by careful analysis of all accidents that occur in the facility. In this study, analysis of accident location by station and specific area revealed a large number of accidents in the bathroom of one nursing station. The main bathroom used during the day for this floor is located behind the main desk, making it difficult to supervise. Findings of this study identified the problem, and education of the nursing staff resulted in increased awareness and supervision. A follow-up study indicated a reduction in the number of accidents at this site. This demonstrates the importance of architectural design in causing accidents, and the importance of this type of study for recognizing and correcting problems that can lead to accidents.

This study confirmed earlier reports that the most common location of accidents in long-term care facilities is at the bedside,[2,8,9] most likely because there is less supervision in this area. Patients usually spend most of their day in the dining and recreation areas, yet fewer accidents occurred at these sites, probably because of more supervision. Timing of accidents was similar to that found in other studies.[2,7,9] Peaks in the number of accidents corresponded to increases in patient activity such as getting in and out of bed, mealtime, and recreation time. In one study, accident peaks were traced to the time of shift change or staff breaks.[9] In this study, peaks that were related to staffing patterns occurred at times of staff shift changes, with the highest number of accidents between 3:00 pm and 4:00 pm, the hour after the change between morning and afternoon shifts. This could be traced to the patterns of staff activity at that time, particularly the manner of getting organized for the shift. Appropriate educational and supervisory action was taken to eliminate the problem, and this resulted in a reduction of peak accident occurrence at this time.

It is important to note that approximately 10% of accidents occurred while patients were handled or transferred by staff members. Investigation generally showed lack of appropriate training in transfer and handling techniques, thus identifying another major environmental risk factor. An in-service education program in patient handling, particularly transfer techniques, has been instituted to correct this problem.

The majority of falls occurred while patients were walking. In most of these instances, no particular contributing factor (such as wet floors or obstructive objects) was found, and falls were attributed primarily to host factors. However, falls out of bed usually occurred while patients tried to climb over bed rails. The presence of urine on the floor and patients' statements indicated that in most cases they had been trying to go to the bathroom. In this type of accident, lack of quick response by staff members to calls or failure to anticipate patients' needs was largely responsible. This type of accident could be prevented or reduced by appropriate staff education and supervision.

Most accidents resulted in soft-tissue trauma. Only 15% resulted in serious morbidity, 4% resulted in hospital stays for a major health event, and one patient died as a result of an accident. In a previous study of 614 accidents in patients who were admitted to the hospital, 40% resulted in slight injury, 11% in moderate injury, 4% in severe injury, and less than 1% of the patients died.[10] In Rodstein's study,[7] only 11.9% of falls required treatment, 3.2% resulted in a fracture (half were hip fractures), and 0.1% were fatal. Thus, accidents in long-term care facilities generally do not result in serious morbidity or mortality. However, the handling of accident victims, nursing and medical attention, and investigations use time and resources that could be better utilized.

Preliminary results suggest that an in-service accident prevention program utilizing data from this study resulted in fewer accidents, particularly those associated with staff handling of patients, shift changes, and the bathroom previously associated with a number of accidents. It also resulted in more accurate and timely reporting of accidents.

Accidents in long-term care facilities should be studied to identify high-risk patients as well as environmental risk factors unique to each facility. An accident prevention program, geared particularly to alteration of environmental risk factors, should be instituted.

Acknowledgments. H. Mattis, assistant DNS provided help in gathering data and investigating the accidents, and Dr Rose Jearolmen Dimant gave editorial assistance.

REFERENCES

1. Rodstein M: Accidents among the aged, in Reichel M (ed): *Clinical Aspects of Aging*. Baltimore, Williams and Williams, 1978, pp 499-513.
2. Berry G, Fisher RH, Lang S: Detrimental incidents, including falls, in an elderly institutional population. *J Am Geriatr Soc* 1981; 29:322-324.
3. *The Random House Dictionary of the English Language*, New York, Random House, 1971, p 9.
4. Rodstein M, Camus A: Interrelation of heart disease and accidents. *Geriatrics* 1983; 28;87-91.
5. Gordon M: Occult cardiac arrhythmias associated with falls and dizziness in the elderly: detection by Holter monitoring. *J Am Geriatr Soc* 1978; 26:418-423.
6. Overstall PW: Prevention of falls in the elderly. *J Am Geriatr Soc* 1980; 28:481-484.
7. Rodstein M: Accidents among the aged: incidence, causes and prevention. *J Chron Dis* 1964; 17:515-526.
8. Lipsitz LA: The drop attack: a common geriatric symptom. *J Am Geriatr Soc* 1983; 31:617-620.
9. Kalchthaler T, Bascon RA, Quintos V: Falls in the institutionalized elderly. *J Am Geriatr Soc* 1978; 26:424-428.
10. Parrish HM, Weil TP: Patient accidents occurring in hospitals: epidemiologic study of 614 accidents. *NY State J Med* 1958; 58:838-846.
11. Hartigan JD: The dangerous wheelchair. *J Am Geriatr Soc* 1982; 30:572-573.

Rigid ureteropyeloscopy

FUAD J. KHURI, MD, ROBERT J. PEARTREE, MD, RONALD A. RUOTOLO, MD, JOHN R. VALVO, MD

ABSTRACT. **Fifty-six patients underwent rigid ureteropyeloscopy at Rochester General Hospital during 1984. Eleven procedures were for diagnostic purposes and 45 for therapeutic purposes. Four procedures were performed to confirm the diagnosis of ureteral or renal pelvic transitional cell carcinoma, four for unilateral bleeding, and three for ureteral narrowing that proved to be due to a retroperitoneal tumor. Two ureteroscopic procedures were performed for insertion of a ureteral catheter. Forty-three were done for extraction of ureteral calculi. Thirty-six ureteral calculi were extracted for a total success rate of 84%. Ultrasonic disintegration was performed in 12 patients. No significant complications were encountered in this series. Ureterolithotomy can be avoided in most patients with ureteral calculi.**

(NY State J Med 1985; 85:205-206)

INTRODUCTION

Advances in optics and lens technology have enabled the development of instruments that permit the visualization and management of the entire urinary tract. Ureteral calculi that are too large to pass spontaneously or too difficult to be manipulated blindly may now be fragmented and removed using ultrasonic disintegration under direct vision.[1] Ureteral or renal pelvic filling defects can be visualized and biopsies can be performed.[2]

Rigid and flexible instruments are available for ureteropyeloscopy. Flexible ureteroscopes are expensive and fragile, and they give poor quality images; their main use is in combination with the rigid ureteroscope.[3] This report is confined to our experience with the rigid ureteroscope at Rochester General Hospital during the past year.

PATIENTS AND RESULTS

Fifty-six patients underwent ureteroscopy; 11 for diagnostic purposes and 45 for therapeutic purposes during the past year. Of the 11 procedures done for diagnostic purposes, four were performed to confirm the diagnosis of ureteral or renal pelvic carcinoma; four for unilateral bleeding; and three for ureteral narrowing that proved to be due to a retroperitoneal tumor. All four patients with proven urothelial carcinoma underwent nephroureterectomy, and the diagnosis was confirmed on biopsy. The four patients with unilateral hematuria were not helped by the ureteroscopy, and their management was not altered by the procedure.

Two ureteroscopic procedures were performed for insertion of ureteral catheters, which could not be inserted in the usual cystoscopic fashion. Forty-three ureteral calculi in 43 patients were managed with the ureteroscope. Thirty-six calculi were successfully extracted and seven procedures failed, for a success rate of 84% (Table I). Two failed procedures were due to dislodgement of an upper ureteral stone to an inaccessible place in the kidney; these stones were salvaged later by percutaneous ultrasonic nephrolithotripsy. Two

---

The authors are in a group practice in urology in Rochester, NY.
Address correspondence to Dr Khuri, 1445 Portland Ave, Suite 304, Rochester, NY 14621.

TABLE I. Rigid Ureteroscopy for Ureteral Calculi

Result	No. of Patients	%
Calculus retrieved	36	83
Calculus not retrieved; open ureterolithotomy required	2	5
Calculus not retrieved, but salvaged percutaneously	2	5
Calculus not retrieved, but passed spontaneously	2	5
Inability to insert ureteroscope	1	2
Totals	43	100

patients required open ureterolithotomy, for an open surgical rate of 5%. Two calculi passed spontaneously following the procedure. The calculi measured 7 mm to 15 mm in 40 patients; four patients had calculi that measured less than 7 mm. Table II shows the location of the stones in the ureter and the success rate of the treatment. Ultrasonic disintegration was needed to remove three of the six upper ureteral calculi, six of the 14 mid-ureteral calculi, and three of the 23 distal ureteral calculi. Operating time varied from 10 min to 90 min. The length of hospital stay varied between two and five days. Complications included false passage at the ureterovesical junction and renal colic. There was no apparent damage to ureteral integrity, sepsis, or significant bleeding. Forty-two stones were composed of calcium oxalate, and one was struvite.

Table II. Calculus Location and Success of Extraction by Rigid Ureteroscopy

Location in Ureter	No. of Patients	Stone Retrieved	%
Upper ureter	6	3	50
Mid-ureter	14	11	80
Distal ureter	23	22	95

DISCUSSION

Ureteroscopy permits endoscopic access to the ureter and pelvis; regions that previously could be visualized only during an open operation. Manipulation of ureteral calculi with ultrasonic disintegration offers distinct advantages over blind stone manipulation or open ureterolithotomy. The length of hospital stay is minimized, and patients return to work and resume normal activity immediately after discharge from the hospital.

Diagnostic and therapeutic indications of ureteropyeloscopy are growing with increased experience and the demonstration of ease, efficacy, and safety of upper tract renal endoscopy. Diagnostic indications include the following: visualization and biopsy of ureteral or renal pelvic filling defects to differentiate between blood clot, tumor, or uric acid stone; demonstration of ureteral stricture or extrinsic ureteral compression; and follow-up for low-grade urothelial ureteral tumors. Therapeutic indications include ureteral calculus removal with or without ultrasonic disintegration, biopsy and fulguration of early ureteral lesions, and insertion of a ureteral catheter when usual cystoscopic methods are unsuccessful.

The rigid ureteroscope has a working length of 41 cm and is available with sheaths of 11F to 12F. Telescopes with a viewing angle of 5 or 70 degrees are available.

Cystoscopy is peformed and dilation of the intramural ureter is carried out with ureteral dilators. Metal ureteral dilators as well as silastic ureteral dilators along a ureteral guidewire were used to dilate the ureterovesical junction. The ureteroscope is then introduced through the urethra under direct vision into the bladder and through the ureterovesical junction. The scope is advanced into the ureter as the lumen is visualized. A ureteral catheter may sometimes serve as a guide. Fluoroscopy may be helpful, but is not necessary.

Ureteroscopy for calculi represents the most frequent use of the procedure.[4-6] Once the calculus is clearly in view, a Pfister-Schwartz stone basket is introduced and the calculus is engaged. The entire unit of basket, calculus, and scope are withdrawn down the ureter. In those situations in which either the calculus is too large or the ureter is narrow, the calculus is disintegrated using ultrasound equipment. Fragmentation of the calculus is continued until all fragments are extracted easily.[1]

Identification of papillary urothelial tumors in the ureter and renal pelvis are followed by biopsy and electrocoagulation.

Like most procedures, ureteropyeloscopy has disadvantages: general anesthesia is required; the procedure may fail; calculus removal may not be 100%; and the procedure could result in ureteral perforation.

Ureteropyeloscopy adds significantly to the urologist's diagnostic and therapeutic capacities. The procedure requires patience and caution for safe performance, but can be satisfying to both patient and urologist.

REFERENCES

1. Huffman JL, Bagley DH, Schoenberg HW, Lyon ES: Transurethral removal of large ureteral and renal pelvic calculi using ureteroscopic ultrasonic lithotripsy. *J Urol* 1983; 130:31-34.
2. Goodman TM: Ureteroscopy with rigid instruments in the management of distal ureteral disease. *J Urol* 1984; 132:250-253.
3. Bagley DH, Huffman JL, Lyon ES: Combined rigid and flexible ureteropyeloscopy. *J Urol* 1983; 130:243-244.
4. Huffman JL, Bagley DH, Lyon ES: Treatment of distal ureteral calculi using rigid ureteroscope. *Urology* 1982; 20:574-577.
5. Ford TF, Watson GM, Wickham JEA: Transurethral ureteroscopic retrieval of ureteric stones. *Br J Urol* 1983; 55:626-628.
6. Lyon ES, Huffman JL, Bagley DH: Ureteroscopy and ureteropyeloscopy. *Urology* 1984; 23:29-36.

The poultice

John S. Haller, Jr

From the days of the prophet Isaiah, who once ordered a paste made of figs for King Hezekiah, the poultice or cataplasm, as it was sometimes called, was an essential element in effective therapeutics. Not until the latter decades of the 19th century did germ theory and knowledge of asepsis lead to its gradual extinction. By then, modern medical teaching had labored to replace old customs and therapeutic practices with a more scientific medicine. The healthy objectivity of the more schooled—both in the sciences and the arts—within the profession, marked the beginnings of a new era and a new authority. With asepsis, one age ended and another began. And with that knowledge, the artifacts of countless generations began to crumble.

In western medicine, especially in England and the United States, poultices were typically made of bread, linseed meal, oatmeal, cornmeal, arrowroot, carrots, turnips, or bran. After the bread poultice, the linseed or flaxseed meal poultice was the most popular. Poultices were prepared in a saucepan with boiling water and were stirred to the consistency of cooked cereal. The poultice must be smooth, free of lumps, soft without being sloppy, and of sufficient bulk to retain both warmth and moisture. The natural oil in the linseed meal poultice tended to keep it soft and pliable, and physicians sometimes added lard to the oatmeal poultice to produce a similar effect. If not too soft or too stiff, it adapted to any surface. After application, care was taken to maintain the right consistency lest the edges of the poultice become too dry or too hard. A proper poultice was spread over the affected part to about ⅜ of an inch deep, with the same thickness on the edges as in the middle, and the surface was smoothed with a knife or thin spatula. The cooled meal was then reheated and, if it became too stiff, a little water was added to make it more pliable.[1]

COMMON USES

Poultices were prepared for use in both hot and cold form. Cold poultices were prescribed for ulcers, gangrene, and after amputations. One such example was recounted by surgeon John Power[2] of England in 1770, of a woman in her late sixties to whom he administered bark, red wine, spirituous stupes, opiates, and volatile and cordial medicines for a gangrene that had spread from her toes to her ankle. As she became exhausted from the treatment, Power resorted to a poultice made from yeast, wheat flour, honey, and water. He mixed the ingredients into a thin paste, which he heated until fermentation began. He then let it cool before applying it to the affected foot. The treatment, which lasted from July through November, ended when the toes became loose and were removed, the putrid stench abated, new flesh appeared, and the patient was declared healed.

Physicians found the hot poultice just as useful for a variety of internal inflammations including pleuritis, pneumonia, bronchitis, enteritis, hepatitis, peritonitis, and cynache. In pleuritis, poultices were applied after general and local bleeding and blistering. After applying a large blistering plaster, physicians directed that a warm poultice be laid over the area and replaced every two hours. "I have seen cases that put on all the characters of peritonitis and enteritis," remarked physician William English of London in 1816, "where the stomach was so irritable, that medicines could not be retained for an instant, which did well by the application of a very large blistering plaster, succeeded in two hours by poultices kept constantly hot, after which, medicines could be administered effectually." [3]

For some purposes, physicians prepared a bag of flannel or oiled silk into which fit another piece of flannel filled with the hot poultice. While the outside bag was kept warm by holding it before a fire, the poultice itself was prepared with boiling water or milk. Once the outer covering was sealed, with the poultice inside, it could be applied to the skin without burning. Although doctors varied the temperature of the poultice with the nature of the disease, they preferred generally to make the poultice as hot as possible without causing the skin to be scalded.[4]

In treating bronchitis and pneumonia, physicians applied a poultice from the armpit to the pit of the stomach and all around the chest, back, sides, and front. To enhance the effect, the patient wore a flannel shirt over the poultice and lay under blankets. In typical cases, physicians worked with two poultices at a time—one applied to the patient and the other held ready for application. As the poultice cooled, the flannel holding it was opened, the meal scraped off, and the new hot poultice applied.

Although some physicians believed that poultices were effective only by virtue of the water or the feculent matter they contained, most thought the remedial effects were due both to the warmth and moisture as well as to the active ingredients of the poultice—linseed-meal, or more sophisticated bread poultices containing emollients, narcotics, and other medicines. The fermenting or yeast poultice remained the common application in cases of mortification or fetid sores. Chlorinated soda poultices proved effective for indolent sores, charcoal poultices (wood

---

Address correspondence to Mr Haller, Department of History, California State University at Long Beach, 1250 Bellflower Blvd, Long Beach, CA 90840.

charcoal in powder form) were popular for fetid ulcers and sloughing sores, and the soothing arrowroot poultice became a popular treatment for irritable sores.[5]

NEW INGREDIENTS AND FORMS

Physicians, as a rule, were very conscious of the cost of their medicines and, particularly in the late 18th and early 19th centuries, when sectarianism was rampant, were anxious to provide medical treatment that was as economical as it was wise. Exemplary of this attitude was the English surgeon Thomas Payne[6] of Brook Street, Grosvenor Square, who suggested a poultice that he hoped would provide a considerable savings to hospitals, infirmaries, dispensaries, and the British Navy during periods of wheat flour scarcity. His substitute, which he modestly called Mr. Payne's Poultice Powder, consisted of three parts pollard (bran), one part linseed flour, and four parts boiling water. The resulting pulp, he claimed, was as good as the bread and milk poultice and, in addition, kept longer without putrefaction. Moreover, the expense was not half of that made with bread and milk, and the ingredients were such "as not to encourage dishonesty in the servants of public institutions." He also recommended the ingredients as the basis of almost every other form of poultice—stale beer poultices, anodyne poultices (with decoction of poppies), and sub-astringent poultices (with the saturine water).

By the 1840s, substitutions for the meal poultice reflected the newer technology of the industrial revolution. First English physicians, and then European and American physicians, made use of the spongio-piline, a three-layered fabric composed of sponge and wool felt and coated on one surface with India rubber, as a substitute for the poultice and as a vehicle for applying lotions, liniments, and other topical applications. The coating of India rubber on one side of the spongio-piline prevented evaporation. When used as a cataplasm, physicians cut and pared the fabric to the required form and size. They then applied distilled vinegar, salt, powdered charcoal, warmed porter-dregs or strong beer, decoction of hemlock, evaporated liquor of boiled carrots, decoction of foxglove, the fermenting or yeast poultice, acetate of lead, and chloride of zinc.

Compared with standard pulp poultices, the spongio-piline was light, clean, sweet, compact, soft, economical, and comfortable to use. The material, physicians argued, saved time and energy that previously went into the making of bread and oatmeal poultices; it also saved valuable meal for use in public and charitable institutions, where the ingredients were made into gruel. Most important, the fabric was durable and could be reapplied as many as 100 times.[7]

The spongio-piline was not the only substitute for the poultice. Increasingly, French, German, and American physicians sought alternative forms of treatment, such as the Rigollot papers, which displaced the mustard-poultice, and the Lelievre cataplasm, which replaced the linseed meal poultice in the 1870s. Lelievre prepared the cataplasm by wetting two layers of wadding with a solution of *Fucus crispus* and then allowing them to dry under heat and pressure. This process produced a sheet with the consistency of cardboard, which physicians could then cut to any desired shape, soak in hot water (which swelled the tissues), and apply to the treated part. When one side was covered by an impermeable tissue (India rubber), the wadding held the medicament without drying. This economical poultice did not soften or crumble and was especially useful in military hospitals and ambulances because of its easy portability. When produced on a large scale, the Lelievre wadding was cheaper than linseed-meal poultices and preferable to the notorious biscuit-dust poultices used in the Navy.[8]

Despite the increased use of substitutes during the second half of the 19th century, the older poultices continued to be employed in the healing of wounds and sores. Occasionally critics blamed poultices for the formation of "proud flesh" (granulations which arise on a sore in the process of healing) or of concealing defects of treatment; nevertheless, they remained popular among physicians throughout the 1860s. During that period, poultices were frequently used for surface ulcers, superficial and subcutaneous inflammations, and suppurating bubos. Bread poultices and warm water dressings remained popular prescriptions for treating wounds created by amputation.[9]

A SOURCE OF INFECTION?

With the better understanding of asepsis and antiseptic measures following the work of Louis Pasteur (1822–1895) and Lord Lister (1827–1912), physicians began to reconsider the appropriateness of poultices, which they discovered to be the source of pyogenic bacteria. "The ordinary filthy poultice of flaxseed, slippery elm or bread and milk, has no longer a place among the resources of the aseptic surgeon," commented Rogers[10] in 1894. "The common poultice," he concluded, "is a hotbed for bacteria, and as such should be discarded." [10] However, instead of eliminating the poultice out of hand, some physicians compromised on a poultice of hot bichloride (solution of bichloride on gauze), which they placed on the part to be healed, thus applying continuous heat and moisture. For example, William Stewart Halsted (1852–1922), Professor of Surgery at the Johns Hopkins Hospital, prescribed a hot iodoform gauze soaked in vaseline or oil, which he applied to infected wounds. Others preferred to use sterilized gauzes treated with carbolic and boracic acid.[10]

Even with the impact of germ theory and asepsis, not all physicians chose to keep up with the newer medicine. One physician in 1892 insisted that the hot poultice was equal to bloodletting when used early in pneumonia or bronchitis. "This continued application of heat to the skin and subjacent tissues of the chest," he wrote, "will open the capillaries and fill them, like a sponge, with blood. It is in fact equal to bloodletting, without letting out the blood, and the relief given to the lungs, by reflex action and the withdrawal of the blood from them, is simply marvellous."[1] As late as 1900, Samuel E. Earp, MD, of Indiana, was still recommending poultices made of flaxseed meal, cornmeal, bread and milk, chamomile leaves, and hops to produce pus in high-grade inflammations. He recommended that the poultice be covered with sheet rubber, oiled silk, or an oiled newspaper to hold its warmth.[11]

ASEPSIS: A NEW ERA

Although isolated support for the poultice continued, prominent surgeons such as Roswell Park (1852–1914) of

Connecticut remained unconvinced as to the efficacy of either the original poultice or its aseptic successor. For Park, the author of a textbook on surgery, "the essence of successful treatment of suppuration is contained in adequate incision, efficient drainage, and frequent change of dressing, supplemented by irrigation." The moist dressing no longer served as an effective treatment in cases of suppuration.[10]

Park's views were by no means atypical of educated medical thinking in both Europe and America in the 1890s and early 1900s. The poultice had proven itself to generations of physicians and no doubt had earned a rightful reputation in the therapeutic regimen of an age when "laudable pus" was a sign of proper healing. Park and the newer generation of physicians who had benefited from the labors of Semmelweis, Pasteur, and Lister found little consolation in the claims of this old therapeutic giant and the resulting high mortality from septicemia, pyemia, erysipelas, tetanus, and hospital gangrene. The age of Listerism, surgical cleanliness, and general asepsis had arrived—and none too soon.

REFERENCES

1. Bonavia E: Comments on cataplasmata. *Provincial Med J* 1892; 11:123.
2. Power J: On the use of fermenting cataplasms in mortifications. *Medical Transactions*, Royal College of Physicians of London. 1785; 3:47–50.
3. English W: On the use of the poultice in internal inflammations. *Edinburgh Med Surg J* 1816; 22:303–305.
4. How to make a poultice. (editorial) *Practitioner* 1882; 29:279–281.
5. Marwick A: On the application of poultices. *Lancet* 1848; 2:346.
6. Payne T: A short address to the faculty, and others whom it may concern, recommending the use of a new poultice; with the view of saving wholly the consumption of bread and oatmeal. *London Medical Review and Magazine* 1799–1800; 2:568–570.
7. Kay WT: Markwick's patent epithems; comprising the substance of a report on their use, economy, and superiority over ordinary cataplasms, fomentations, etc., made to Sir William Burnett, Medical-Director-General of the Navy. *Medical Times* 1849; 19:56–57.
8. "A new poultice." *Medical Times and Gazette* 1874; 2:720.
9. Callender GW: Notes on the use of poultices. *Practitioner* 1869; 2:268–271.
10. Rogers JT: The use of poultices and wet dressings in surgery. *Northwestern Lancet* 1894; 14:224–225.
11. Earp SE: The use and abuse of poultices. *NY Med J* 1900; 71:163–164.

FROM THE LIBRARY

AN EXPERIMENTAL AND CLINICAL STUDY OF THE VALUE OF CLAY-MIXTURE POULTICES

One of the most interesting phenomena in the trend of our modern therapeutics is the present extensive use by physicians of the variously medicated clay mixtures as external applications in the treatment of pneumonia, bronchitis, pleurisy, rheumatism, synovitis, boils, etc., in place of the cotton jacket, mustard and flaxseed poultices, blisters, cupping, camphorated oils and hot stupes. By a clay mixture I mean any one of the various proprietary preparations which are said to have an antiphlogistic action in inflammations of superficial and deep structures. The essential composition of all such clay mixtures is a base consisting of silicate of alumina and magnesia combined with salicylic acid, peppermint, wintergreen, eucalyptus, iodin or ammonium iodid and iron carbonate. The glycerin is added to give the kaolin or white clay its thick, jelly-like consistency so as to allow of easy spreading, while the other substances are added for whatever antiseptic power and pleasant odor they may impart to the mixture.

I take it that these mixtures are used by physicians partly because they are widely advertised, but mainly because their use is based on a so-called theory or rationale of therapeutic action. . .

AUGUSTUS H. ROTH, AB, MD
(*JAMA* 1905; 44:1185)

Informed consent

The New York approach

James M. Morrissey, Esq

Every human being of adult years and sound mind has a right to determine what shall be done with his own body; and a surgeon who performs an operation without his patient's consent, commits an assault, for which he is liable in damages.[1]

In 1914, with this now-famous passage from *Schloendorff v New York Hospital,* Judge Benjamin Cardozo of the New York State Court of Appeals presaged the requirement that physicians, whenever possible, obtain the informed consent of their patients before providing treatment. The legal requirement of informed consent, however, is only about 30 years old, has proved to be a nettlesome issue for both the physician and patient and, not surprisingly, has been the topic of much legislative and judicial action. The purposes of this article are as follows: first, to discuss the theoretical underpinnings of the doctrine of informed consent; second, to discuss the definition of informed consent in New York State; and third, to set forth the exceptions to the requirement of informed consent.

Underlying Basis

The doctrine of informed consent is an offshoot and logical extension of the most fundamental and basic rights of free people: the inviolability of the human body and the right of a person to exercise complete dominion over his or her person. The doctrine of informed consent is not simply an arbitrary imposition of the values of the legal profession on the medical profession. Rather, the doctrine serves several important public purposes, including the right of persons to self-determination, the promotion of individual autonomy, and the avoidance of fraud and duress in the consent to medical treatment transaction. It also encourages self-scrutiny by physicians and rational decision-making by patients, and educates the public.[2]

The theory underlying the doctrine of informed consent grew from the right of a patient to sue a physician for failure to obtain consent before treating that patient—a common-law battery defined as the intentional offensive touching of a person without his or her consent. (Although the courts often refer to the basis of an action for treatment without informed consent as an assault, the correct term is battery.) The failure of a physician to obtain consent was classified as a battery, according to the following decision in the *Pratt v Davis* case:

> [U]nder a free government at least, the free citizen's first and greatest right, which underlies all the others—the right to the inviolability of his person, in other words, his right to himself—is the subject of universal acquiescence, and this right necessarily forbids a physician or surgeon, however skillful or eminent . . . to violate without permission the bodily integrity of his patient . . . and [to operate] on him without his consent or knowledge.[3]

Thus, the early consent cases dealt simply with the right to consent to treatment and the concomitant right to refuse treatment. They did not speak of consent as being "informed," as that term is understood today.

The facts before Cardozo in *Schloendorff* supported the battery theory. The patient was admitted to the hospital suffering from a stomach disorder. The treating physician discovered a lump, the character of which, the patient was informed, could not be determined without an ether examination. The patient consented to the ether examination but stated that an operation was not to take place. The ether examination took place and, while the patient was unconscious, a fibroid tumor was discovered. Notwithstanding the patient's instructions, the physician removed the tumor.

The theory that a physician commits a battery upon a patient when he renders treatment without informed consent was soon rejected by the courts: "the unauthorized touching, within the concept of medical care, lacked the traditional elements of wrongful intent and harmful aggression."[4] Simply put, the physician rarely, if ever, means to do harm.[5]

Usually the crux of a patient's claim was not that no consent was given, but rather that the consent was ineffective due to the physician's failure to make full and complete disclosure of the risks and benefits of treatment. Patients' claims changed from "I did not give consent" to "I gave consent, but the physician failed to provide me with sufficient information upon which to render a meaningful decision."

Furthermore, in some cases, full disclosure of the risks and benefits of treatment could be antithetical to the physical and mental well-being of the patient.[6] What is commonly referred to as "therapeutic privilege" means that in some situations a physician is legally permitted to withhold from a patient material facts about treatment when he deems that complete "risk-disclosure poses such a threat of detriment to the patient as to become unfeasible or contraindicated from a medical point of view."[7]

Address correspondence to Mr. Morrissey, Morrissey & Thrope, 12 W 37th St, New York, NY 10018.

Courts, generally loath to change established law, narrowed the battery theory's import, so that today it is virtually meaningless. As one court explained, in *Schloendorff* the facts before Cardozo were as follows:

> . . . the patient notified her physician that surgery was not to be performed under any circumstances without her consent. Her instructions were nevertheless disregarded and such violation was held by the court to have constituted a trespass upon her body and the basis for an action in assault [battery].[5]

However, the court noted, the following circumstance would present an entirely different case:

> . . . [t]he surgery in question was not undertaken without any consent by the patient. [In such cases the physicians do] not intend to inflict any injury or impermissible procedure upon the [patient]. [Where] the patient [gives] consent to surgery, but it was given based on claimed misrepresentation of fact . . . [s]uch failure constitutes an act of negligence or malpractice, rather than assault or battery.[8]

Thus, under current law, when a physician has obtained some type of consent from a patient, a lawsuit for treatment without first obtaining informed consent will be grounded in malpractice, not battery.

There are several distinctions between battery and malpractice. First, a lawsuit based on assault must be brought within one year of the incident, but an action based on malpractice may be brought within 2½ years of the incident.[9] Second, under the common law, the plantiff in a battery action would be required to show only that the physician provided treatment to which the plantiff did not give informed consent; initially, no expert medical testimony was required to prove that the physician was remiss, from a medical point of view, in failing to make adequate disclosures before treating the patient. In an action based on common-law malpractice however, the plantiff has to show that the physician failed to act in accordance with the standard of practice in the medical community regarding informed consent; expert medical testimony to that effect is required or the patient's claim cannot succeed.[10]

Also, in an action based on battery, a patient could be awarded damages without proving actual harm. In malpractice, the harm must be proven. Finally, the courts may award punitive damages as part of a battery lawsuit, but may not do so as part of a malpractice action. Punitive damages are damages designed to punish the offending party and to discourage others from embarking on the same course of conduct.

But the determination that malpractice standards would apply to informed consent cases did not end the controversy. Some courts subsequently ruled that the standard against which the physician would be judged should not be determined by accepted medical practice, but rather by what the reasonable layman would deem an adequate disclosure of information. The former is referred to as the "physician-centered" approach and the latter the "patient-centered" approach.

The physician-centered approach was severely criticized by the court in *Canterbury v Spence:*

> There are, in our view, formidable obstacles to acceptance of the notion that the physician's obligation to disclose is either germinated or limited by medical practice. To begin with, the reality of any discernible custom reflecting a professional consensus on communication of option and risk information to patients is open to serious doubt. We sense the danger that what in fact is no custom at all may be taken as an affirmative custom to maintain silence, and that physician-witnesses to the so-called custom may state merely their personal opinions as to what they or others would do under given conditions . . . Nor can we ignore the fact that to bind the disclosure obligation to medical usage is to arrogate the decision on revelation to the physician alone. Respect for the patient's right of self-determination on particular therapy demands a standard set by law for physicians rather than one which physicians may or may not impose upon themselves.[11]

THE NEW YORK APPROACH

Until 1975, New York was a patient-centered state and "a doctor [was] obliged to divulge to his patient the risks singly or in combination, tested by general considerations of reasonable disclosure under all circumstances, [that would] materially affect the patient's decision whether to proceed with treatment."[12]

Disclosure defined by the medical profession. This approach, however, was rejected by the New York state legislature in 1975 when it passed §2805-d of the Public Health Law entitled "Limitation of Medical Malpractice Action Based on Lack of Informed Consent," a statute generally viewed as more favorable to physicians. This law defines the lack of informed consent as follows:

> . . . the failure of the person providing the professional treatment or diagnosis to disclose to the patient such alternatives thereto and the reasonably foreseeable risks and benefits involved as a reasonable medical practitioner under similar circumstances would have disclosed, in a manner permitting the patient to make a knowledgeable evaluation.[13]

This duty and the scope of disclosure for the physician is now defined and established by the medical profession in that expert medical testimony is required to establish whether the physician failed to make adequate disclosure. Whereas the standard of disclosure refers to that which the "reasonable medical practitioner"—the ordinary physician—would disclose, a physician with special expertise or knowledge in a treatment area, for example a specialist or researcher, probably would be held to a more rigorous standard of disclosure.[14] Only "reasonably foreseeable risks" need be disclosed, as contrasted with the more stringent "all known" risks.

A question frequently arises as to who is responsible for assuring adequate disclosure when more than one physician is involved with the patient. The answer is not a simple one. The law in New York is that when a physician "refers his patient to another physician and retains a degree of participation, by way of control, consultation, or otherwise, his responsibility continues to properly advise his patients with respect to the treatment to be performed by the referred physician."[15]

Obviously, this answer begs the question of what is meant by a "degree of participation". *Graddy v New York Medical College*[16] is instructive on this point. In this case, two physicians, Street and Bell, were specialists in otolaryngology. They shared office space, which they occupied at different times of the day. They also shared a secretary, as well as professional equipment and supplies. They were not partners, and each had his own patients whom he billed as an individual practitioner. However, when one physician was absent, his patients would, with their consent, be treated by the other physician. The treating physician was paid half of the fee.

A patient had seen Street on several occasions for a problem, and Street had advised against surgery. Bell, however, saw the same patient for the same problem in Street's absence and recommended surgery. Bell performed the surgery and was found later to have committed malpractice. The issue presented to the court was whether Street could be held liable for the negligent acts of Bell. The court held that he could not. It stated that "[A] referral of a patient by one physician to another competent physician, absent partnership, employment, or agency . . . does not impose a liability on the referring physician."[17]

The general rule is that "Physicians employed together by the patient, and diagnosing or treating a case together, without withdrawal by, or discharge of, either, owe the same duty and are jointly liable for any negligence."[18] Thus, a family physician who participated in the diagnosis of a fracture was held jointly liable with the specialists who negligently applied the cast;[19] a hospital was held liable for the negligence of a nurse;[20] and a physician who went on vacation and turned over his office, equipment, staff, stationery, and patient files to a substitute was held liable for the negligent acts of the substitute.[21]

An entire surgical team, however, will not be charged to insure that a patient has given informed consent. Rather, the referring physician, if a degree of participation is retained, and the treating physicians, for example the surgeon and the anesthesiologist, must assure that consent to all phases of the procedure is informed. A consulting specialist retained to advise and make recommendations to the treating physician who then makes all final determinations probably does not have any obligations unless called in directly by the patient to render a second opinion.[22]

Prudent person. Section 2805-d erects an additional barrier that the patient must meet before legal recovery may be had. The patient must also show the following:

> . . . that a reasonably prudent person in the patient's position would not have undergone the treatment or diagnosis if he had been fully informed and that the lack of informed consent is a proximate cause of the injury or condition for which recovery is sought.[13]

The standard of a "reasonably prudent person" is an objective rather than a subjective standard, and liability for failure to obtain informed consent should not depend on the relative sophistication or lack of sophistication of the patient. The reader, however, is cautioned to examine the competency of each patient to render informed consent. If the patient appears to lack the mental capacity to consent to treatment, treatment should not proceed. A discussion of the treatment of incompetents is beyond the scope of this article; the physician may have to obtain substitute consent from a family member or may require judicial authorization to treat.

Also, a physician cannot be held liable if a patient decides, based on the negative results of the therapy, that he should not have consented to the treatment. As one court has stated,

> No one, least of all the patient, can answer [the question of whether consent to the procedure would have been forthcoming if adequately informed] and the physician is placed at the mercy of the patient's hindsight. Honest though he may be, the disastrous result of the therapy necessarily affects the patient's guess as to whether the risk, if divulged, would have been acceptable.[23]

Thus, liability will not result unless the patient can show that a reasonable person, fully informed, would not have consented to the procedure and that the lack of informed consent resulted in the injuries complained of.

In essence, New York State has melded the patient-centered and physician-centered approaches and requires a patient to meet both conditions to hold the physician liable for treatment without informed consent.

Exceptions. Lastly, §2805-d for the first time codifies the various exceptions to the requirement for informed consent, thus providing the physician with clearer guidance. Those exceptions are as follows:

> (a) the risk not disclosed is too commonly known to warrant disclosure; or
>
> (b) the patient assured the medical practitioner he would undergo the treatment, procedure or diagnosis regardless of the risk involved, or the patient assured the medical practitioner that he did not want to be informed of the matters to which he would be entitled to be informed; or
>
> (c) consent by or on behalf of the patient was not reasonably possible; or
>
> (d) the medical practitioner, after considering all of the attendant facts and circumstances, used reasonable discretion as to the manner and extent to which such alternatives or risks were disclosed to the patient because he reasonably believed that the manner and extent of such disclosure could reasonably be expected to adversely and substantially affect the patient's condition.

When a physician forgoes informed consent based on one of these exceptions, the decision should be well documented in the patient's chart.

CONCLUSION

In adopting an informed consent approach that looks to the medical profession to define acceptable standards of disclosure to patients, New York State has gone against the tide of the more patient-centered approaches adopted by other states. Law and Polan[24] commented on the statute:

> [I]n 1975 New York enacted a statute severely restricting patients' rights to be informed about proposed treatment. Courts in New York had previously adopted an informed-consent standard based on the informational needs of a reasonable patient. The new law returns to the medical profession the power to determine how much information must be provided to patients in obtaining their consent for treatment. . . . The New York legislature had before it information showing that actions for failure to obtain informed consent have *no* measurable effect on malpractice premiums. In the entire history of New York, there had been only *one* case in which money was paid solely on an informed consent theory [emphasis added].

Indeed, the New York approach undercuts the notion of informed consent for reasons that are not altogether clear. No doubt it represents a continuation of the highly paternalistic belief that physicians have only the best interests of their patients at heart, and the debate about informed consent is "much ado about nothing." Although physicians may have the patients' best interests at heart, this most emphatically does not provide any basis for restricting information to patients. As argued by Katz,[25] the paternalistic nature of the physician-patient relationship is at fundamental odds with the notion of personal autonomy, most notably because the values of the physician may not be the values of the patient. Thus, it may be a mistake to presume that the physician's well-intended opinion of what is best for a patient is in accord with the patient's privately held values.

This concept is often demonstrated when a patient refuses treatment necessary to preserve life. Whereas the physician may place primary value on his or her commitment to prolong life, the patient may place primary value on religious beliefs or "dying with dignity" and thus reject the treatment. Decisions on a particular mode of treatment also reflect value judgments. Although the surgeon may recommend a mastectomy to alleviate breast cancer, a young woman who seeks to "remain whole" may prefer to manage her illness by a lumpectomy and radiation therapy.[26]

It is beyond question that the values of the patient must win out. The physician must, therefore, assist the patient in clarifying his or her own personal values and in making treatment decisions accordingly, even if this is not mandated by the strict legal requirements of informed consent or if the patient's ultimate treatment decision is contrary to the physician's own judgment. It is important to view the requirement of informed consent as a law that advances important societal and individual interests. By understanding the purposes of informed consent, it is hoped that the physician will see it not as an unnecessary incursion on professional discretion but rather as the further performance of a high public calling.

REFERENCES

1. *Schloendorff v New York Hospital*, 211 NY 2d 125, 129–130 (1914).
2. Capron A: Informed consent in catastrophic research and treatment, 123 *University of Pennsylvania Law Review* 340, 364–376 (1976).
3. *Pratt v Davis*, 118 Ill. App. 161(1905); See also, *Shack v Holland*, 389 NYS 2d 988 (Sup Ct Kings Co 1976); *See also, Dries v Gregor*, 242 NYS 2nd 561 (1st Dept 1980); *Vargas v Rosal-Arcilles*, 438 NYS 2d 986 (Sup Ct Queens Co 1981); *Williams v Cordice*, 418 NYS 2d 995 (Sup Ct NY Co 1979).
4. *Shack v Holland*, supra, 389 NYS 2d at 991.
5. *Murriello v Crapotta* 382 NYS 2d 513, 514 (1st Dept 1976).
6. Prosser: *Handbook on the Law of Torts*, St. Paul, West Publishing Co, 3rd Edition 1964, p 107.
7. *Salgo v Stanford University Hospital*, 317 P 2d 170 (Ct App 1957).
8. *Williams v Cordice*, supra, 418 NYS 2d at 997.
9. *Civil Practice Laws and Rules* §§215(3) & 214-a; But see Article 2 of the CPLR for exceptions to this general requirement.
10. *Lipius v White*, 458 NYS 2d 928, 933 (2d Dept 1983); *Shack v Holland*, supra, 389 NYS 2d at 992.
11. *Canterbury v Spence*, supra, 464 F2d at 783–784 (1972); See also *Zeleznik v Jewish Chronic Disease Hospital*, 366 NYS 2d 163, 170; *Dries v Gregor*, supra.
12. *Fogal v Genesee Hospital*, 334 NYS 2d 552, 559 (4th Dept 1973).
13. *New York Public Health Law*, §2805-d.
14. *Toth v Community Hospital at Glen Cove*, 292 NYS 2d 440 (Ct App 1968).
15. *Prooth v Wallsh*, 432 NYS 2d 663, 665 (Sup Ct NY Co, 1980).
16. *Graddy v New York Medical College*, 243 NYS 2d 940 (1st Dept 1963).
17. Ibid. at 944.
18. 70 *Corpus Juris Secondum* §54-c.
19. *Morril v Komasinski*, 41 NW 2d 620 (Sup Ct 1950).
20. *Bing v Thunig* 2 NYS 2d 656 (Ct App 1957).
21. *Impastato v DeGirolamo*, 459 NYS 2d 512 (Sup Ct Kings Co 1983).
22. *Prooth v Wallsh*, supra, 432 NYS 2d at 665–666.
23. *Zeleznik v Jewish Chronic Disease Hospital*, supra, 366 NYS 2d at 171 (1972).
24. Law S, Polan S: *Pain and Profit: The Politics of Malpractice*. New York, Harper & Row, 1978, p 113.
25. Katz J: *The Silent World of Doctor and Patient*. New York, Free Press, 1984.
26. Ibid., pp 90–93.

ASSISTANCE FOR VICTIMS OF FAMINE

> *All deaths are hateful to miserable mortals, but the most pitiable death of all is to starve.*
>
> *Homer*
> The Odyssey, *XII, 341*
> (*translated by W.H.D. Rouse*)

Donations to the Ethiopian famine victims can be sent to Interaction, a coalition of more than 20 humanitarian and church relief groups:

Interaction
200 Park Ave S
New York, NY 10003
(212) 777-8210

The "Essays in Pediatrics" series will include articles that focus on clinical management of the diseases of children and adolescents and that discuss recent advances in basic science, as they relate to the needs of the practitioner. The series will emphasize changing trends, new directions in research, and current controversies in diagnosis and treatment.

Proposals for manuscripts for "Essays in Pediatrics" should be submitted to Carl Pochedly, MD, Director, Department of Pediatric Hematology/Oncology, Nassau County Medical Center, 2201 Hempstead Tpke, East Meadow, NY 11554.

Normal adolescent development

Helping teenagers cope with change

RICHARD E. KREIPE, MD

Although pediatricians are expected to care for patients under age 21,[1] many feel that they lack sufficient training to provide health care to adolescents.[2]. The purpose of this article is to review normal growth and development in the second decade of life. It is only from a secure knowledge base of normal adolescent processes and events that pediatricians can serve the special needs of their older patients.

This review will focus on pragmatic aspects of normal adolescent growth and development as they relate to the daily practice of pediatrics. Four main aspects of development are presented as they pertain to each of the three stages of adolescence: early (ages 10 to 14 years), middle (15 to 16 years), and late (17 to 21 years). The expected physical changes of puberty are described first; then the achievement of independence is discussed. The central task of establishing an identity is explored, and finally, cognitive changes (thinking styles) during adolescence are described. Each of these issues can influence healthy as well as unhealthy adolescents.

NORMAL PHYSICAL CHANGES DURING PUBERTY

Adolescence is the general term used to describe the passage from childhood to adulthood; puberty refers more specifically to the biologic changes that lead to reproductive capacity. For both girls and boys, pubertal events occur in a predictable sequence, but when and how quickly these changes begin are variable.[3] The primary changes themselves, such as menstruation or nocturnal emission, may be of concern to the adolescent. There also may be questions about secondary changes such as body odor or mood swings.

The integration of these changes into the adolescent's evolving self-concept is an important part of early adolescence. The pediatrician can play a key role in this process by providing reassurance of normalcy or early treatment of a pathologic condition. One should not merely give reassurance. Effective, safe treatment is available for a variety of conditions that can accompany puberty. Problems such as acne or dysmenorrhea deserve attention, because they can be serious from the adolescent's point of view.

Girls. Soon after her hips begin to widen, a girl will experience budding of her breasts and the appearance of dark straight pubic hair. These changes begin at about the age of 11, but development beginning at 8½ years is not considered precocious, and lack of development by age 13 is not considered delayed. Breast changes will proceed from the Tanner stage II form (early pubertal) to the Tanner stage V form (adult) during about four years, but these changes can occur during a period as short as 1½ years or as long as nine years. The progression of pubic hair from Tanner stage II to Tanner stage V takes about 2½ years, but can range from 1½ to 3½ years.

About one year after the initiation of breast development, during Tanner stage III, girls grow in height at their greatest rate. The peak of this growth spurt precedes the onset of menstruation. Thus, menarche is a late pubertal event, usually occurring six months after the growth spurt, at or just before Tanner stage IV.

Boys. Boys also experience a regular sequence of physical changes during puberty but lack any marker as obvious as breast development or as distinctive as menstruation. From ages 11 to 13, the average boy is shorter than the average girl, so it often is assumed that boys begin puberty two years later than girls. This is not so. Boys and girls begin puberty at a similar age. However, the growth spurt is a late event in the pubertal sequence of boys, in contrast to the early growth spurt in girls.

Testicular enlargement indicates a transition from Tanner stage I to stage II and heralds visible evidence of pu-

---

Address correspondence to Dr Kreipe, Division of General Pediatrics and Adolescent Medicine, University of Rochester Medical Center, Box 690, 601 Elmwood Ave, Rochester, NY 14642.

berty. It begins at about 11½ years of age, only a few months after the age girls' breasts begin budding. Within a year of testicular enlargement, penile enlargement begins, marking Tanner stage III. This is usually preceded by the appearance of pubic hair and followed by the appearance of axillary hair. Growth of the testes can begin as early as 9½ years or as late as 13½ years and can end at any time between the ages of 13½ and 17 years. An increase in penile length can begin at 10½ to 14½ years and can end at 12½ to 16½ years. The growth spurt, as previously noted, is a late occurrence in boys but can begin from ages 10½ to 16 years and can end from ages 13½ to 17½ years.

Given the range of ages during which development may occur, a pediatrician could treat a group of normal seventh graders whose pubertal development includes Tanner stages I through V. Because many conditions such as fluctuations in hemoglobin concentration in boys, worsening of scoliosis, acne, and gynecomastia are related to biologic maturation, staging of pubertal development can become the basis of preventive health care for adolescents, similar to that used with younger patients. Therefore, the assessment and recording of the Tanner stage should become a standard part of health care in children in the second decade of life.[4]

ACHIEVEMENT OF INDEPENDENCE

Because conflicts with authority, rebellion against rules, and challenges of autonomy are characteristic teenage behaviors, adults often assume that adolescents experience a great deal of psychological turmoil in the attainment of independence. Empirical evidence is to the contrary; most people pass from childhood to adulthood with little "storm and stress," especially if they have a consistent, supportive home environment.[5] In the process of achieving independence, adolescents may defy every limit established by their parents; they may even come in contact with police or be expelled from school. Such behavior, however, is the exception rather than the rule.

The essence of independence-seeking activity is a desire for self-determination and individualism; it is expressed through a range of behavior, from impulsive risk-taking to responsible adultlike actions.[6] Thus, activity related to the achievement of independence takes many forms; some problematic, some exemplary.

Problematic examples of independence-seeking activity can vary. For example, a 14-year-old learning-disabled boy may refuse to see a physician for evaluation of his school problems. A 15-year-old girl may choose to leave home by becoming pregnant; her 18-year-old boyfriend may not comply with gonorrhea treatment. A 16-year-old with hemophilia may regularly "forget" to administer his clotting factors and play football anyway. Parents may be concerned about their 15-year-old son flaunting family rules or about their 13-year-old daughter smoking marijuana after school.

But not all independence-seeking activity is negative or risk-taking. For example, a 17-year-old mature girl may seek confidential help with contraception. A 16-year-old boy may work 12 hours per week after school and require a Saturday morning appointment. A 15-year-old girl may ask a friend for a ride home from a party because she judges her boyfriend to be too drunk to drive.

What constitutes normal independence-seeking behavior is difficult and often dangerous to judge. First, one's culture, environment, family, and socioeconomic status play a role in determining any behavior. Streetwise behavior appropriate to the south Bronx may be out of place in Watertown. Second, many people consider a certain behavior to be normal merely because of its common occurrence. Adolescents themselves may use this rationale to justify sexual activity or drinking alcohol by saying, "everybody's doing it." Third, independence-seeking behavior is sometimes considered normal if there are no obvious harmful consequences. Moderate use of marijuana or the effective use of foam and condoms for birth control are examples. Finally, some kinds of behavior are normal because without them independence cannot be attained. Getting a job, accepting responsibility for oneself, and making decisions based on weighing risks and benefits are typical activities in this category.

There can be no simplistic distinction between what is normal and abnormal in exploratory behavior. Furthermore, such a dichotomous approach may lead the clinician to consider "normal" problem behavior as "something they'll outgrow" or "just a phase they're passing through." These attitudes breed complacency.

It is better to judge a particular behavior in the context of how the youth is developing at school, in the family, and with friends, and then determine whether it represents an isolated, episodic event or whether it fits into a more persistent pattern of behavior. Even isolated events, such as the first-and-only episode of driving while drunk, can be a problem, but the health care provider needs to be especially concerned about global, chronic, repetitive, or dangerous behavior.

IDENTITY

The establishment of an identity is an essential task of adolescence. In the process, there is a consolidation of one's ego, one's self-concept, and one's role in life. According to Erikson's theory of development, if an adolescent fails to attain a stable role or identity, role confusion results.[7]

Adolescents who have difficulty establishing an identity do not know who they are, do not know where they are going in life, and have trouble establishing long-lasting relationships. Children normally enter adolescence with an unformed sense of identity, but if they enter adulthood without this sense, their further development is impeded.

For the early adolescent, identity issues focus on pubertal changes.[8] Puberty results in somatic and psychologic changes, both quantitative and qualitative, that lead the youth to ask, "Am I okay? Am I normal?" Concerns of boys include voice changes, gynecomastia, nocturnal emissions, and being too thin or too short, whereas girls are often concerned about breast asymmetry, physiologic leukorrhea, menstruation, and being too fat or too tall. Acne, body odor, and overall appearance are of concern to both sexes.

When examining the early pubertal patient, the pediatrician should offer privacy and reassurance. The physical examination provides a natural setting in which to discuss the adolescent's concerns without parents present. Prolonged silences during a physical examination should be avoided. Instead, use the examination to teach the youth about his or her body and to confirm findings that show normal development. Encourage questions verbally, as well as through attitude and patience.

A normal variant should be mentioned, because the adolescent probably is already aware of it. Such a comment might be as follows: "I notice that your breasts are different in size. Some girls your age get worried when that happens. It's common and normal, and they should even out before you're done growing. Were you worried about that, or do you have any questions about it?" This approach teaches the girl something about herself, reassures her that she is normal and that other young girls experience similar changes, and lets her know what to expect in the future. It also invites her to ask questions.

Although children in middle and late adolescence still need reassurance of normalcy, their self-reflective concerns can be summarized by the question, "Who am I?" For the middle adolescent of 15 or 16 years, the answer is characteristically in flux; for the late adolescent 17 to 21 years of age, there should be a consolidation of the self-concept. By the late teenage years, the identity should be sufficiently defined to permit decisions regarding one's future education, vocation, and sexual behavior. Yet the identity is not so rigidly defined as to preclude continued growth and change.

In the process of determining who they are, adolescents may be labeled or may experience other difficulties. Juvenile delinquents often have a negative identity, in which being "bad" has its rewards. Adolescent mothers often see themselves in shallow terms in which education, marriage, or a career have no relevance. Drug abusers may spend most of their waking hours obtaining, using, or thinking about drugs, and are readily identifiable by peers as "druggies" or "burn-outs." The progeny of physicians and other professionals may experience difficulties, since expectations are often high in such families, and adolescents may adopt a forced identity as they believe that they are to follow unwaveringly in their parents' footsteps.

Remedies for such situations are not simple, and one may believe that it is not the pediatrician's role to deal with these "psychiatric" issues. However, it is important for the physician to acknowledge that the attainment of a stable identity is critical for adolescents, and that many disparate kinds of behavior can be understood in light of identity development. Furthermore, as a professional and objective observer of adolescents, the pediatrician who is willing to take the time is in an ideal position to provide reflective, nondirective counseling in such matters.

COGNITIVE CHANGES

Although intellect does not change dramatically during adolescence, the mode of thinking does change. Inhelder and Piaget[9] proposed the terms "concrete operational thinking," to describe the way children think, and "formal operational thinking," to describe the way adults think. The transition between these two cognitive styles occurs during adolescence.

Concrete operations limit one's thinking to literal, here-and-now interpretations of questions. A 12-year-old boy, for example, may respond to the opening question, "What brings you to the office today?" by saying "My mother." Formal operational thinking, on the other hand, enables one to understand figurative speech. It enables one to think about abstractions such as future options or to ask "what if?" and to imagine different, nonexisting possibilities.

In junior and senior high school, skills in formal operational thinking often are needed for academic functioning. Algebra requires the student to understand the abstraction, "let x=10." A history teacher may ask the student to hypothesize "What would have happened if Lee had won at Gettysburg?" An English assignment may require the student to interpret the meaning of a poem.

In the clinical setting, it is important to make an assessment of the cognitive or thinking style of the adolescent. Interviewing a boy who uses concrete operational thinking is laborious and frustrating when he does not respond. Shifting the topic to concrete things such as his dirt bike or favorite baseball team will often lure him into a livelier conversation. Likewise, when asking a girl with headaches about the relative intensity of her pain, one could ask the concrete question, "How bad is it, on a scale of 1 to 10?"

Formal operational thinking allows the adolescent to understand the need for preventive health care, and to consider health issues such as the effective use of birth control methods.[10] Moreover, the adolescent with skills in formal operational thinking is better able to discuss abstractions such as feelings or emotions.

Three clinical concepts related to adolescent cognitive development include egocentrism, personal fable, and imaginary audience.[11] Egocentrism is the normal self-centered focus of the adolescent's attention. Often viewed by parents as selfishness or narcissism, egocentrism is a reflection of the adolescent who is appropriately engaged in self-study, self-exploration, and self-determination. Moreover, adolescent egocentrism can be used to the physician's advantage. The physician can often engage the attention of an adolescent by showing an interest in him or her as a person.

Personal fable refers to a belief that one is in some way special, invulnerable, and invincible; it seems to be an especially powerful force in adolescents. A girl may be sexually active and not use birth control, believing that pregnancy "can't happen to me." A boy may drink and drive, believing that his reactions are not impaired. Both of these are dangerous examples of the personal fable in action. However, an adolescent's personal fable can also have salutary effects. A girl with cystic fibrosis may fight back from a life-threatening pulmonary hemorrhage, believing that "it's not yet my time to die." A boy with a relapse of leukemia may cooperate with therapy, believing that a cure awaits him.

Normal adolescents are egocentric; they think about themselves a great deal. Adolescents often feel as if they are on stage when in the presence of others. The term, "imaginary audience" aptly describes this normal phenomenon in which early and middle adolescents believe that they are the center of people's attention, and that all eyes and thoughts are focused on them.

In clinical practice, the imaginary audience concept becomes most evident during pauses in discussions or during physical examination, when young adolescents often become noticeably anxious. Long silences and cryptic mutterings on the part of the physician may be especially disconcerting. Rather than remain silent, use this time to get to know the youth.

CONCLUSIONS

Understanding the changes associated with adolescent development will enable the pediatrician to interact more effectively with adolescents and their parents. The physical changes of puberty often initiate subsequent psychological changes. Early concerns about identity are focused on physical changes, so that the normal early adolescent, who thinks in concrete terms, requires reassurance about normal growth and development.

As the child moves into middle adolescence, independence and the more abstract identity question of "who am I?" become preeminent. Mobility, physical maturity, and autonomy make this phase of development especially difficult because middle adolescents are neither children nor adults. However, a sincere concern for them as persons first and as patients second; a willingness to listen; and a trusting, confidential relationship that places an appropriate degree of responsibility on the adolescent provide a foundation for ongoing health care.

In many ways, the late adolescent appears to be an adult. It is important to recognize that even though an 18-year-old male may be Tanner stage V, working 40 hours per week, and living away from his family, he is not necessarily an adult man. He may still have anxiety about being away from his family or have questions about who he is and what he is all about, or he may still only think in concrete terms. "Allowing" him to be an adolescent may be one of the most important services the pediatrician can provide when managing the health care of such a person.

The role of the pediatrician is multifaceted. The challenges of adolescent medicine can be met by pediatricians who are willing to learn more about the changes of adolescence and to provide confidential health care to a developing teenage population.

REFERENCES

1. Burnett RD, Willian MK, Olmstead RW: Pediatric manpower requirements. *Pediatrics* 1978; 61:438.
2. Task Force on Pediatric Education: *The Future of Pediatric Education: A Report.* Evanston American Academy of Pediatrics, 1978.
3. Tanner JM: *Fetus into Man: Physical Growth From Conception to Maturity.* Cambridge, Harvard University Press, 1978.
4. Hoffman AD, Greydanus DE: *Adolescent Medicine.* Menlo Park, Addison-Wesley Books, 1983.
5. Offer D: *The Psychological World of the Teenager: A Study of Normal Adolescent Boys.* New York, Basic Books, 1969.
6. Aten MJ, McAnarney ER: *A Behavioral Approach to the Care of Adolescents.* St Louis, CV Mosby Co, 1981.
7. Erikson EH: *Identity: Youth and Crisis.* New York, Morton Publishers, 1968.
8. Coleman JC: Current contradictions in adolescent theory. *J Youth Adolescence* 1978; 7:1.
9. Inhelder B, Piaget J: *The Growth of Logical Thinking from Childhood to Adolescence.* New York, Basic Books, 1958.
10. Elkind D: Egocentrism in adolescence. *Child Dev* 1967; 30:1025–1034.
11. Elkind D: Understand the young adolescent. *Adolescence* 1978; 13:127.

FROM THE LIBRARY

THE PROBLEM CHILD

It is believed that many problems of childhood can be helped by a concerted effort on the part of the parents, physicians, the churches, the schools and psychiatric clinics. It is most essential that children have a proper religious training in order to instill in them a wholesome respect for the laws of God and Man. Men of affairs, men of influence in the community should show that they are not above religious influence by taking an active interest in churches and Sunday schools. If the boy and girl feel that father and mother can get along without religion they cannot see why it is necessary for their own welfare.

Such a wonderful organization as the Rotary Club, composed of business and professional men, is paying particular attention to boys' work movements and endorse heartily the Boy Scouts of America, realizing that healthy sublimations of this sort make for better citizens in later life. An organization like the Boy Scouts directs the gang instinct which is a normal boyhood reaction into healthy forms of activities, thus neutralizing the unhealthy sublimation which makes the gangster and the gunman, as the result of whom lawlessness is today so rampant in our large cities.

Similar organizations among the girls make for normal women in later life and prevent prostitution and illegitimate pregnancies.

The mental and physical examination of all first offenders in order to discover just what caused this first misstep in their lives would in my opinion prevent the development of many chronic offenders.

The grading by group and individual psychological tests of children on their entrance into our primary schools would solve many of the problems which arise there. In this way superior children will be kept sufficiently occupied to prevent their getting into mischief and the feeble-minded and dull children will receive the attention which they sadly need and which is impossible under the present school system.

It is realized that all this is an ambitious program, but it is one which, if properly carried out, will prevent many of our modern problems in criminology and amply repay us from an economic standpoint.

SYLVESTER R. LEAHY, MD
(*NY State J Med* 1923; 23:27)

Leiomyosarcoma of the ovary

SUDHAKAR A. REDDY, MD, TUNG PUI POON, MD, GITA RAMASWAMY, MD,
VICTOR TCHERTKOFF, MD

Primary, pure leiomyosarcoma of the ovary is a rare lesion that may histologically mimic mixed müllerian tumors with a predominant sarcomatous component, cellular fibrosarcomas, fibrothecomas, and stromal cell sarcomas. Electron microscopy and cytoplasmic intermediate filament typing by immunoperoxidase techniques are helpful aids in the confirmation of the diagnosis. We present a case of primary, bilateral leiomyosarcoma of the ovary with ultrastructural confirmation.

CASE REPORT

A 48-year-old gravida 10, para 6 obese woman was admitted to the hospital in November 1982 because of dizziness and a dull pain in the left lower quadrant of the abdomen. She had undergone a cesarean section and bilateral tubal ligation five years prior to admission. At that time the ovaries were reported as being unremarkable.

On physical examination a vague rounded mass, slightly tender and firm, was palpable in the left lower quadrant of the abdomen. On bimanual pelvic examination the uterus was felt to be enlarged to a size common for 16 weeks of pregnancy. Large, irregular, and firm masses were noted on either side of the uterus.

Roentgenographic studies of the chest, abdomen, and pelvis were within normal limits. An intravenous pyelogram revealed lateral deviation of the ureter with mild dilation. The results of serum chemistry studies and a hemogram were noncontributory.

She underwent an exploratory laparotomy which revealed multiple adhesions between the greater omentum and the bilateral ovarian tumors. The uterus was unremarkable. The left fallopian tube was intermittently involved by serosal tumor implants. The omentum was studded with numerous, small, pink-grey nodules that measured up to 4 cm in diameter. A total abdominal hysterectomy with bilateral salpingo-oophorectomy and omentectomy were performed.

Representative sections from the ovarian masses and from the omental nodules were examined, and a diagnosis of leiomyosarcoma was made.

The uterus with cervix was unremarkable. Tissue from the right ovarian tumor was fragmented, measuring 10 × 8 × 6 cm in aggregate dimensions. The left ovarian tumor measured 14 × 12 × 10 cm in aggregate dimensions. The tumors were firm, irregular masses, and were tan-grey in appearance with areas of hemorrhage and cystic changes.

Tissue for light microscopy was fixed in 10% buffered formalin. Sections were stained with hematoxylin and eosin, and sections from selected blocks were stained with Masson trichrome, phosphotungstic acid hematoxylin, and reticulin stains.

The tumors in both ovaries appeared similar in morphology and were composed of slender to plump elongated spindle cells arranged in fascicles of varying sizes. The nuclei were elongated with blunt ends and were hyperchromatic, with numerous and bizarre mitotic figures (more than 10 mitoses per 10 high power fields) (Figs 1,2). Tumor giant cells were occasionally seen. The nuclei were aligned together to form a pattern that resembled palisades. The cytoplasm was abundant and demonstrated a deep red staining with Masson trichome. With the phosphotungstic acid hematoxylin stain, longitudinal purple myofibrils were demonstrated. There was a delicate reticulin network between the cells. Necrosis, hemorrhage, and hyalinization were present within the tumor.

For electron microscopy, fresh tissue from the neoplasm was fixed in buffered glutaraldehyde. The specimen was post fixed in 2% osmium tetroxide, dehydrated through graded concentrations of ethanol and then embedded in epon. Thin sections were stained with uranyl acetate and lead citrate and were examined with an RCA electron microscope.

The tumor revealed deeply clefted nuclei and abundant cytoplasm containing myofibrils joined by dense bodies. Pinocytic vesicles and a basal lamina enveloping the cell membrane were present. The features confirmed the smooth muscle origin of the tu-

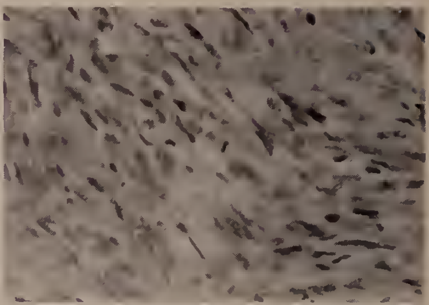


FIGURE 1. Leiomyosarcoma with numerous atypical mitotic figures. (hematoxylin eosin stain, original magnification × 125)

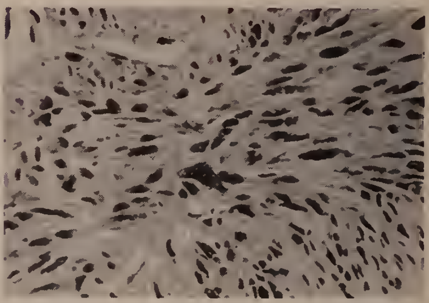


FIGURE 2. Leiomyosarcoma with large bizarre tumor giant cells. (hematoxylin and eosin stain, original magnification × 250)

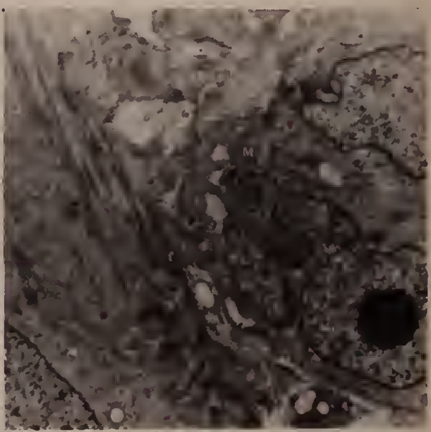


FIGURE 3. Electron dense myofibrils are found at the periphery of the tumor cell (Nuc, nucleus; f, myofibrils; M, mitochondria). (uranyl-acetate and lead citrate stain, original magnification × 11,000)

From the Department of Pathology, New York Medical College, Metropolitan Hospital Center, New York.

Address correspondence to Dr Ramaswamy, Assistant Professor, Department of Pathology, Metropolitan Hospital Center, 1901 First Ave, New York, NY 10029.

mor (Fig 3).

The patient's recovery was uneventful, and she was discharged from the hospital on the 17th hospital day. She was to be given external radiation therapy to the lower abdomen and pelvis as an outpatient. However, the patient did not keep her appointments and did not receive radiotherapy. She returned to the Gynaecology Oncology clinic about 16 months after being released from the hospital. At that time there were no physical signs of recurrence of the tumor. She was to return six months later, but did not keep the appointment.

Primary Leiomyosarcomas of the Ovary

No	Age (yr)	Side of Lesion	Associated Ovarian Lesion	Follow-Up	Reference
1	84	Right	None	Died of pulmonary embolization, pulmonary edema and abdominal tumor	2
2	61	Right	None	Died of peritonitis and abdominal metastasis	2
3	44	Bilateral	None	Died of peritonitis and pulmonary metastasis	2
4	48	Not specified	Malignant teratoma	No follow-up	3
5	70	Right	Dermoid cyst	Died of abdominal metastasis	4
6	70	Right	Serous cystadenoma	Alive six months after surgery	5, 10
7	60	Right	None	Died of pulmonary embolism, pulmonary and abdominal metastases	7
8	14	Right	Malignant teratoma	Died in six months	1
9	56	Right	Benign cystic teratoma	No follow-up	1
10	66	Not specified	None	Died in one month	1
11	61	Left	None	Died in two years with abdominal metastases	1
12	67	Right	None	Died 16 months after surgery with abdominal metastases	1
13	17	Right	None	No follow-up	8
14	74	Right	None	Died four months after surgery with abdominal metastases	9
15	56	Left	Serous cyst	Alive seven months after surgery	6
16	48	Bilateral	None	Alive 16 months after surgery	Present case

DISCUSSION

Primary ovarian sarcomas are rare tumors and constitute about 1% of ovarian neoplasms.[1] Primary ovarian leiomyosarcomas account for a small subset of ovarian sarcomas. In their review of 2,600 cases of primary ovarian tumors, Azoury and Woodruff[1] culled 43 cases of ovarian sarcomas, of which five were leiomyosarcomas. In two of the five patients with leiomyosarcoma the tumor arose in a teratoma. The remaining three cases were pure leiomyosarcomas.

A total of 15 patients with primary ovarian leiomyosarcomas have been described in the literature (Table).[1–10] The age range of these patients was 14–84 years. In most instances the patients were more than 40 years old. In only two of the 15 cases did the disease appear before 20 years of age. The most common initial symptoms were abdominal swelling with pain. In nine of the 15 patients the right ovary was involved. In three of the patients the left ovary was involved, and in two patients the side was not stated. In only one of the 15 patients was there bilateral ovarian involvement.

In four of the 15 patients the tumor arose in either a benign or a malignant teratoma.[1,3,4] In two instances the tumor was associated with a serous ovarian tumor.[5,6] Istre[2] reported the only case with concomitant, bilateral tumors.

The diagnosis of the leiomyosarcoma is based on the light microscopic findings of a spindle cell tumor in which the cells are elongated. The cells contain central "cigar-shaped" blunt-ended nuclei. Mitotic figures may be bizarre and are present in numbers exceeding 10 per 10 high power fields.[11] Low-grade leiomyosarcomas have fewer mitotic figures—5 to 9 per 10 high power fields.[11]

Characteristic ultrastructural findings include myofilaments joined by dense bodies in the cytoplasm, pinocytic vesicles, and intercellular connections. The basal lamina invests the entire cell membrane.[12]

In the last few years, cytoplasmic intermediate filament typing has been used to distinguish different cell types, to classify tumors, and to improve the accuracy of diagnosis in surgical pathology.[13] There are five distinct classes of intermediate filaments:

- the cytokeratins—characteristic of true epithelial cells;
- neurofilaments—characteristic of many but not all neurons;
- glial fibrillary acidic protein—filaments found in various glial cells such as astrocytes and Bergmann glial cells;
- desmin filaments—characteristic of most myogenic cells; and
- vimentin filaments—typical of various nonepithelial cells, in particular those loosely termed as being of mesenchymal origin.

There are also certain types that seem to lack intermediate filaments.

Immunoperoxidase techniques for detecting the cytoplasmic intermediate filaments are useful in the typing of anaplastic spindle cell tumors, especially when ultrastructural confirmation cannot be obtained. Leiomyosarcomas are generally found to be positive for desmin or vimentin, or both.[13]

Spindle cell neoplasms such as fibrosarcomas, fibrothecomas, and mixed müllerian sarcomas of the ovaries should be considered in the differential diagnoses. Ovarian leiomyosarcomas can be differentiated from the more frequent mixed müllerian sarcomas of the ovary, in that the latter are characterized by the presence of malignant, bizarre epithelial elements mixed with a sarcomatous stroma.[14] Epithelial elements are absent in pure leiomyosarcoma. Stromal cell sarcoma and fibrosarcoma may resemble leiomyosarcoma, however, cytoplasmic myofibrils are absent in these tumors.[12]

There are various histogenetic mechanisms proposed in the development of ovarian leiomyosarcoma. Balazs and Laszlo[7] draw attention to wolffian duct remnants and vessel walls as sources of smooth muscle neoplasms of the ovary. This may be the mechanism in cases of pure non-teratoid leiomyosarcoma. When leiomyosarcomas occur in association with teratomas, development from totipotential ovarian mesenchyme is the suggested histogenetic mechanism.[14]

The prognosis for ovarian leiomyosarcoma is poor, with most patients dying of recurrence within two years of initial presentation. Although three patients received radiotherapy or chemotherapy, or both after surgery, their follow-up was too short to evaluate the efficacy of this treatment.

Acknowledgment. Dr William B. Ober, Director Emeritus, Department of Pathology, Hackensack Medical Center, and Visiting Professor, University of Medicine and Dentistry, New Jersey, provided consultation and confirmation of the diagnosis.

REFERENCES

1. Azoury RS, Woodruff JD: Primary ovarian sarcomas: report of 43 cases from the Emil Novak Ovarian Tumor Registry. *Obstet Gynecol* 1971; 37:920-941.
2. Istre B: Sarcoma of the ovary. *J Oslo City Hosp* 1951 1:231-237.
3. Stout AP (moderator): *Tumor Seminars in Pathology.* St. Louis, The CV Mosby Co, 1955, pp 125-127.
4. Castleman B: Case 44202, in Case records of the Massachusetts General Hospital: weekly clinicopathologic exercise. *N Engl J Med* 1958; 258:1010-1013.
5. von Numers C, Mikkonen R: Leiomyosarcoma arising in serous cystadenoma of ovary. *Ann Chir Gynaecol* 1960; 49:240-244.
6. Walts AE, Lichtenstein I: Primary leiomyosarcoma associated with serous cystadenocarcinoma of the ovary. *Gynecol Oncol* 1977; 5:81-86.
7. Balazs M, Laszlo J: Das Leiomyosarkom des ovar. *Zentralbl Bynak* 1965; 87:633-638.
8. Connor EJ, Aydinel OH, Lee JS, Homayouni M: Leiomyosarcoma of the ovary, in de Watterville H (ed): *Diagnosis and Treatment of Ovarian Neoplastic Alterations.* Amsterdam, Excerpta Medica, 1975, pp 47-50.
9. Bettendorf U, Zimmerman H: Leiomyosarkom des Ovarium. *Med Welt* 1975; 26:1429-1430.
10. Nieminen U, von Numers C, Purola E: Primary sarcoma of the ovary. *Acta Obstet Gynecol Scand* 1969; 48:423-432.
11. Enzinger FM, Weiss SW: in, *Soft Tissue Tumors*, St. Louis, The CV Mosby Company, 1983, pp 298-315.
12. Ferenczy A.: Diagnostic electron microscopy in gynaecologic pathology. *Pathol Annu* 1979; 14 (pt 1):358-381.
13. Osborn M, Weber K: Tumor diagnosis by intermediate filament typing: a novel tool for surgical pathology. *Lab Invest* 1983 48:372-394.
14. Scully RE: Tumors of the ovary and maldeveloped gonads. *AFIP Fascicle* 1982; 16:311-315.

See also pp 191-192.

Metastatic leiomyosarcoma of iliac bone

VINITA PATANAPHAN, MD, MOHAMMAD ABDUL HAFIZ, MD,
OMAR M. SALAZAR, MD

The five-year survival rate for patients with uterine leiomyosarcoma who have been treated by surgery has been reported to be between 25% and 75%.[1-5] Distant metastases are common[3,5,7] and are associated with a poor prognosis. The lung is the most common site of distant metastases.[3,5-7] These occur in 50% to 80% of cases. Bone metastases are rare.[3,5-7]

Cytotoxic chemotherapy has been given to patients with metastatic leiomyosarcoma, with a poor response rate; however, there was some improvement in survival rates for complete responders.[8] Radiation therapy is usually given as an adjuvant to surgery to improve local control.[5,6,9] Leiomyosarcoma is regarded by some to be radioresistant.[2,10] Because of the sarcoma's propensity to early dissemination, radiation is not regarded as a primary course of therapy.

We report on a patient in whom soft tissue disease developed two years after excision of a vaginal leiomyosarcoma, with involvement of the iliac bone five years later. The latter condition was treated with radiation alone. The response was excellent, and the patient has survived this treatment without complications for more than five years.

CASE REPORT

In 1973, a 31-year-old woman was found to have a small cyst protruding from the vaginal wall; it was removed, and pathologic findings showed it to be a well differentiated leiomyosarcoma. Because of the possibility of a primary uterine lesion, abdominal hysterectomy was subsequently performed. No additional evidence of sarcoma was found in the uterine specimen.

Two years later, a 3 × 3-cm mass was discovered on the medial side of her right thigh; the mass was removed by wide excision. The same pathologic findings of leiomyosarcoma were found. A metastatic survey revealed no other metastases.

In January 1978, the patient discovered a lump in the right buttock about 2-3 inches below the right iliac crest. This was excised and found to be a lipoma. Because of pain around the surgical scar, a pelvic roentgenogram was taken, which revealed a large lytic lesion in the right iliac crest. Needle biopsy of this lesion revealed a leiomyosarcoma.

The gross appearance of the vaginal and thigh masses were similar, measuring 3 × 2 cm and 3 × 3 cm, respectively. Both had a lobulated, firm, pale-tan, and rounded appearance without any visible capsule. The needle core from the bone lesion had a pale-tan appearance. Histologic analysis of the three masses yielded similar results. They were composed of criss-crossing bundles of smooth muscle spindle fibers. The nuclei were generally vesicular; however, there were areas where nuclear atypia, such as hyperchromasia and enlargement, were present. Mitotic figures, the most important indicator for behavior in soft tissue sarcoma, were present throughout the tumors. Their number varied from area to area in all three specimens. Most regions had less than 5 mitoses per 10 high power field (HPF)—generally between 2 or 3—but other areas had between 10 and 15 mitoses per 10 HPF (Figs 1, 2).

Cytotoxic therapy was begun. The patient received two courses of cyclophosphamide, vincristine, and dacarbazine, but this was discontinued because of severe nausea and vomiting. Because of her reaction to chemotherapy, no further cytotoxic therapy has been given. She was referred to the Department of Radiation Oncology, where she received radiation—a total dose of 6,500 rad in 30 treatments—to the lesion in the right iliac crest. The first 5,000 rad/5 weeks was given with 4 MeV photons from a linear accelerator through 12 × 17-cm anterior-pos-

---

From the Departments of Radiation Oncology (Drs Patanaphan and Salazar) and Pathology (Hafiz), University of Maryland Hospital, Baltimore.

Address correspondence to Dr Patanaphan, Assistant Professor, Department of Radiation Oncology, University of Maryland Hospital, 22 S Greene Street, Baltimore, MD 21201.

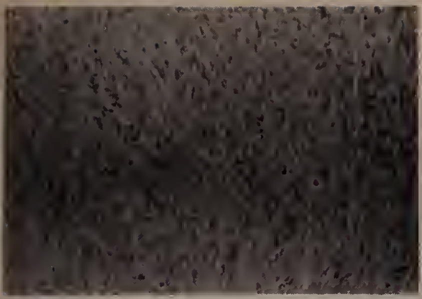


FIGURE 1. An area of the vaginal tumor showing criss-crossing spindle cells of leiomyosarcoma with some mitotic figures. (hematoxylin and eosin stain, original magnification ×40).

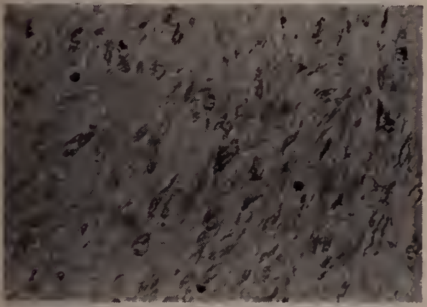


FIGURE 2. Higher magnification of the leiomyosarcoma showing enlarged nuclei and two mitotic figures. (hematoxylin and eosin stain, original magnification ×100).

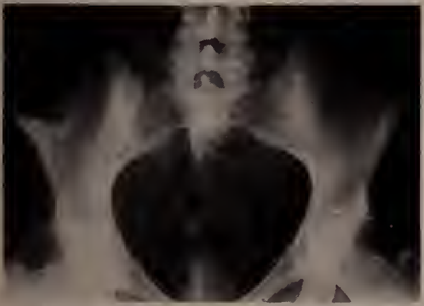


FIGURE 3. Roentgenogram of pelvis prior to radiation treatment showing osteolytic lesion involving the right ilium.

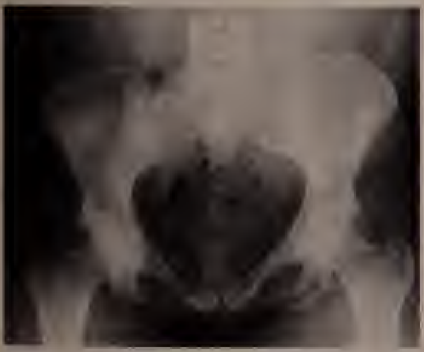


FIGURE 4. Roentgenogram of pelvis showing bony repair with increased sclerosis and a marked decrease in lucency.

terior portals. An additional 1,500 rad was given in five treatments with a 35 MeV electron beam, through a posterior 12 × 13-cm port.

The patient has been followed by both radiation oncologists and medical oncologists. Serial films of the pelvis have shown a decrease in the size of the lucent component of the lesion and an increase in the sclerotic component, indicative of bony repair (Figs 3, 4). Serial chest films, an intravenous pyelogram, and a small bowel series survey have shown no evidence of metastases. The patient has remained free of disease for more than five years following the last treatment and 10 years after the initial diagnosis. There have been no late sequelae from the pelvic irradiation.

DISCUSSION

Bone metastases from leiomyosarcoma are rare and are usually associated with widespread metastatic disease and a poor chance of survival.[5,7] There have been few reports in the literature concerning bone metastases from uterine leiomyosarcoma, with the exception of those that involve ribs and vertebrae.[11] Most of the lesions were osteolytic and occurred in long bones such as the femur and humerus, and a few were osteoblastic lesions of the lumbar vertebrae. Interestingly, there were long-term survivors among these patients.[11]

There is an entity called "multiple leiomyosarcoma" which occurs in soft tissue, including the genital organs, lower urinary tract, and respiratory tract; it is associated with excellent survival rates after only conservative surgical treatment.[12-14] Primary leiomyosarcoma of bone is a rare entity.[15,16] The majority of patients have osteolytic lesions of long bones, more commonly at the distal end of the femur; almost all of the patients have undergone amputation.

The issue in this patient was whether she had a primary uterine leiomyosarcoma with vaginal, soft tissue, and bone metastases; multiple leiomyosarcoma; or a primary bone lesion that was a separate entity from the vaginal tumor. The absence of a primary leiomyosarcoma in the uterus, and the fact that this patient already had two other sites of disease (vagina and soft tissue of the thigh) would place this tumor in the category of multiple leiomyosarcoma. The long-term survival of the patient without late signs of distant metastases suggests that multiple leiomyosarcoma is probably the proper diagnosis. If one considers this patient as having metastatic bone disease, palliative treatment with radiation or chemotherapy would have yielded a poor chance of tumor control or cure.

Radiation treatments, when given together with conservative surgery (wide excision), have been shown to be effective in achieving local control of soft tissue sarcomas.[17] This treatment has been considered as an alternative to radical surgery, which requires removal of part of a limb or the whole limb. Radiation alone has been reported to yield less favorable results[18] and has been described as not being effective against these tumors. This patient's management demonstrates that bony involvement of leiomyosarcoma can respond well to radiation when given in high doses (6,500 rad).

Radiation has been used for leiomyosarcoma of the bone only as a palliative treatment, usually after radical surgery. This case suggests that radiation alone could be given to metastatic leiomyosarcoma in areas where surgery is not possible. A radical course of radiation should be considered for patients who have metastatic bone involvement and no evidence of disease elsewhere.

Acknowledgment. Roberta A. Strohl, RN, MN, Linda J. Owings, and Linda Alston assisted in the preparation of this manuscript.

REFERENCES

1. Salazar OM, Bonfiglio TA, Patten SF, Keller BE, Feldstein M, Dunne ME, Rudolph J: Uterine sarcomas; natural history, treatment and prognosis. *Cancer* 1978; 42:1152-1160.
2. Vongtama V, Karlen JR, Piver SM, Tsukada Y, Moore RH: Treatment, results and prognostic factors in stage I and II sarcomas of the corpus uteri. *Am J Roentgenol* 1976; 126:139-147.
3. Vardi JR, Tovell HM: Leiomyosarcoma of the uterus: cliniocopathologic study. *Obstet Gynecol* 1980; 56:428-434.
4. Dinh TV, Woodruff JD: Leiomyosarcoma of the uterus, *Am J Obstet Gynecol* 1982; 144:817-823.
5. Mantravadi RV, Bardawil WA, Lochman DJ, Liebner EJ, Townsend DE, Chao JH, Chaudhuri B: Uterine sarcomas: an analysis of 69 patients. *Int J Radiat Oncol Biol Phys* 1981; 7:917-922.
6. Salazar OM, Bonfiglio TA, Patten SF, Keller BE, Feldstein ML, Dunne ME, Rudolph JH: Uterine sarcomas: analysis of failures with special emphasis on the use of adjuvant radiation therapy. *Cancer* 1978; 42:1161-1170.
7. Hannigan EV, Gomez LG: Uterine leiomyosarcoma: a review of prognostic clinical and pathologic features. *Am J Obstet Gynecol* 1979; 134:557-564.
8. Hannigan EV, Freedman RS, Elder KW, Rutledge FN: Treatment of advanced uterine sarcoma with vincristine, actinomycin D, and cyclophosphamide. *Gynecol Oncol* 1983; 15:224-229.
9. Salazar OM, Dunne ME: The role of radiation therapy in the management of uterine sarcomas. *Int J Radiat Oncol Biol Phys* 1980; 6:899-902.
10. Edwards CL: *Undifferentiated Tumors in Cancer of the Uterus and Ovary*. Chicago, Year Book Medical Publishers, Inc, 1969, pp 84-94.
11. Drake ET, Dobben GD: Leiomyosarcoma of the uterus with unusual metastases. *JAMA* 1959; 170:1294-1298.
12. Wood WG, Burch TK, Thomas Jr CY: Multiple leiomyosarcomas. *South Med J* 1977; 70:381-383.

13. Young RA: Leiomyosarcomatosis of probable uterine origin with long survival—a case report. *Postgrad Med J* 1977; 53:281-285.
14. Friedell GH: Long survival with metastatic uterine leiomyosarcoma. *Am J Obstet Gynecol* 1962; 83:1579-1581.
15. Sanerkin NG: Primary leiomyosarcoma of the bone and its comparison with fibrosarcoma. *Cancer* 1979; 44:1375-1387.
16. Angervall L, Berlin O, Kindblom LG, Stener B: Primary leiomyosarcoma of bone: a study of five cases. *Cancer* 1980; 46:1270-1279.
17. Suit HD, Russell WO, Martin RG: Sarcoma of soft tissue: clinical and histopathologic parameters and response to treatment. *Cancer* 1975; 35:1478-1483.
18. Fletcher GH: *Textbook of Radiotherapy*, 3rd ed. Philadelphia, Lea and Febiger, 1980, p. 924.

See also pp 191-192.

Recurrent chronic epididymitis as an initial symptom of colovesical fistula

CIRIL J. GODEC, MD, DAN RICE, MD, STEVEN YOUNGER, MD

Fistula formation can occur in any segment of the gastrointestinal tract, from the duodenum to the rectum, and in the urinary tract, from the renal pelvis to the urethra. Urologic symptoms are usually the most prominent, although the disorder mainly affects the gastrointestinal tract. In some cases, gastrointestinal symptoms dominate the clinical picture.

CASE REPORT

A 45-year-old man was initially seen in September 1982 with a complaint of left scrotal pain, a temperature of 38°C, and chills that lasted 36 hours. On physical examination, swelling of the left epididymis was found and the diagnosis of epididymitis was confirmed. Urinalysis revealed more than 50 white blood cells and zero to two red blood cells per high-power field. The patient was treated with oral antibiotic agents. He returned four days later with worsening symptoms, chills, and nausea. His temperature rose to 39°C. The swelling of the left epididymis increased, but there was no skin fixation. Urine culture documented greater than 10×10^3 colony-forming units of *Escherichia coli* per milliliter. Urinalysis showed more than 50 white blood cells and 3+ bacteria. A testicular radioisotope scan documented increased vascularity in the left epididymis. The patient was treated with intravenous antibiotic therapy; he responded well. Before discharge, an intravenous pyelogram was normal, and cystoscopic examination revealed no abnormal findings.

Three months later the patient complained of right scrotal pain. The diagnosis of right epididymitis was established, and the patient responded well to oral antibiotics. For the next three months, he was seen frequently at the urology clinic for recurrent epididymitis. Nine months after the initial visit, bilateral scrotal swelling developed. The patient reported that he had passed chunks of tissue and occasional gas through the urethra. Cytologic studies of a urine sample revealed atypical cells without malignancy. A urine culture contained more than 100×10^3 colony-forming units of mixed organisms per milliliter.

A tentative diagnosis of colovesical fistula was made. An intravenous pyelogram and a cystogram were unremarkable. An ulcerative lesion on the left anterior wall of the bladder was seen on cystoscopy. A barium enema examination demonstrated multiple diverticuli of the sigmoid colon and the presence of barium and a small amount of air in the bladder (Figs 1, 2). An oblique spot-film of the barium enema examination revealed the fistulous tract between the colon and bladder (Fig 3). Proctosigmoidoscopy showed an ulcerative lesion at 30 cm and multiple diverticuli. A biopsy specimen taken from around the ulcerative lesion within the sigmoid colon confirmed an ulcerative benign lesion.

Segmental resection of the sigmoid colon and primary anastomosis, partial resection of the bladder, and partial left epididymectomy were performed. The resected tissue contained a fistulous tract covered with both transitional columnar epithelial cells, with an abundance of goblet cells (Fig 4). The epididymal tissue showed chronic inflammatory changes. Eight months after surgery, the patient had normal micturition and bowel movements and a sterile urine culture.

DISCUSSION

Diverticulitis is the most frequent cause of enterourologic fistula formation. It is responsible for more than 50% of cases. In patients with diverticulitis, colovesical fistula is reported in 2% to 22.8%, with an average incidence of 10%.[1] Malignancy as the cause of the fistula is found in approximately one of four patients, with 75% caused by cancer of the rectosigmoid and the remaining 25% caused by cancer of the cervix or other pelvic organs.[2] The incidence of colovesical fistula in patients with Crohn's disease is 1.6%.[3] Only a small number of cases are due to radiation, trauma, iatrogenic injury, or congenital causes.

In older patients with chronic bladder irritation and chronic urinary tract infection, the possibility of diverticular disease of the bowel should be evaluated. Mayo and Blunt[4] reported that 22.8% of patients who required surgery for diverticulitis had a colovesical fistula. In the largest study, Pugh[5] found a colovesical fistula in 4% of more than 1,000 patients with diverticulitis.

The ratio of colovesical fistula between men and women is 3:1. Perhaps the interposition of the adnexa and uterus between the bladder and bowel decreases the incidence in women. The higher incidence of diverticulitis, as well as the proximity of the bladder to the bowel in the male pelvis, are probably responsible for a higher rate of this disease among men. In women with diverticulitis, colovaginal fistula is 7.5 times more prevalent than colovesical fistula.[6]

Enterovesical fistula occurs in one of 3,000 patients admitted to the hospital for treatment.[5] Most patients have a documented urinary tract infection (95% when initially examined) that is resistant to treatment or that recurs frequently. Urinary tract infection is usually caused by a single pathogen rather than by a mixture of species.

The average duration of symptoms before diagnosis has been reported to be four months[2]; in one case the delay was five years.[7] Diagnosis of colovesical fis-

---

From the Departments of Urology, Long Island College Hospital and the SUNY-Downstate Medical Center, Brooklyn, NY.

Address correspondence to Dr Godec, Director, Department of Urology, Long Island College Hospital, Brooklyn, NY 11201.

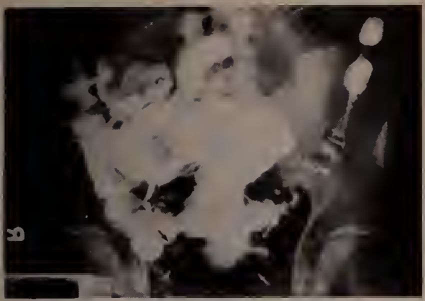


FIGURE 1. Anteroposterior barium enema examination shows barium and gas in the bladder (arrows surround the bladder).

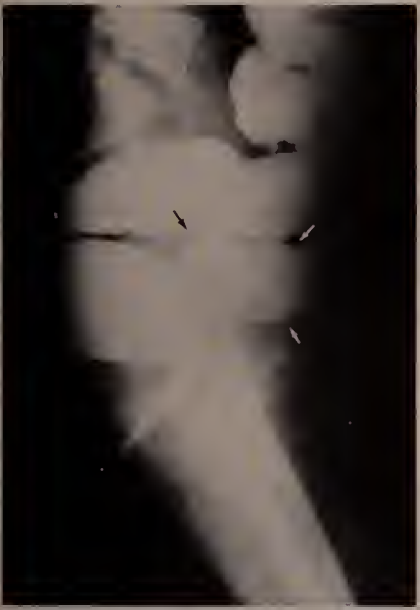


FIGURE 2. Lateral barium enema examination shows bladder almost completely filled with barium (arrows surround the bladder).

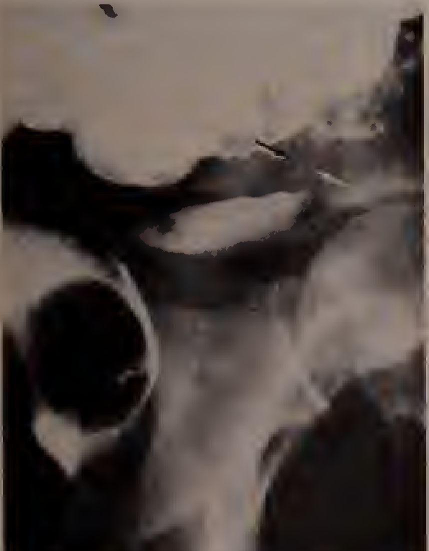


FIGURE 3. Oblique barium enema examination shows fistulous tract (arrows).

tula is usually delayed because of the nonspecific symptoms of suprapubic discomfort, dysuria, and urinary frequency and urgency. Hematuria is unusual, as are chills and fever. Pneumaturia is the most conclusive symptom, especially if the patient is not diabetic and bladder endoscopy has not been performed before the appearance of gas in the bladder. Fecaluria, occurring in about 50% of patients, is less common than pneumaturia, because the passage of fecal particles through a narrow lumen is more difficult than the passage of air. Lower abdominal pain, especially over the left lower quadrant, changed bowel habits, diarrhea, and constipation are the most frequent symptoms involving the gastrointestinal tract.

Although the major underlying disorder usually involves the gastrointestinal tract, enteric symptoms are common. The flow of material is usually from bowel to urinary tract, owing to the pressure gradient between the enteric and urinary systems.[8]

There is no single definitive diagnostic test; cystoscopy is the most helpful test. The usual finding is bullous edema and ulceration, with granulation tissue or ulceration on the dome of the bladder. This area of the bladder must be scrutinized because it is the least accessible to cystoscopy and the most likely to be overlooked. If a lesion is seen, it is advisable to perform a biopsy to check for malignancy. It is better to convert an occult fistula into an overt one than to miss the diagnosis of fistula or, possibly, cancer.

Colonoscopy (which has contributed to a diagnosis in 7% of cases) is less informative than cystoscopy (57%).[7] A combination of cystoscopy and roentgenographic studies appears to be most useful in making a diagnosis. A plain abdominal film can document an air bubble in the bladder, which can be mistaken for air in the intestinal tract. In this patient, bullous edema in the bladder was reduced through aggressive drug therapy, and, as a result, diagnosis by barium enema examination was possible. A colovesical fistula may be only intermittently patent, which can explain an inconsistent pathologic pattern.

Diverticulitis is usually diagnosed by the presence of pain in the left lower quadrant, accompanied by some degree of peritoneal irritation. If there is abscess formation caused by diverticulitis perforation, a palpable abdominal or pelvic mass may be present. Krompier et al[9] reported that 50% of their patients had local signs of peritonitis, and one third had a palpable abdominal or pelvic mass.

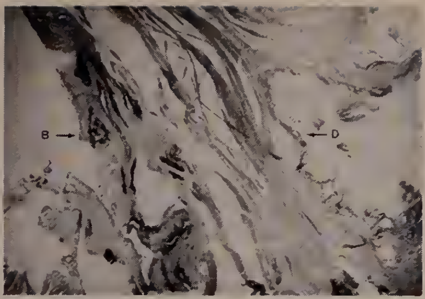


FIGURE 4. Section demonstrating diverticulum of the bowel penetrating the bladder. D indicates bowel mucosa of diverticulum; B, epithelium of the bladder (hematoxylin and eosin stain, original magnification × 40).

An inflamed bowel diverticulum within a segment of colon will adhere to the bladder wall. The diverticular or peridiverticular abscess eventually erodes the layers of bladder wall. A fistulous tract then forms between the diverticulum and the bladder mucosa. There is little damage to the kidney, even in cases in which the diagnosis is delayed for several years, as long as there is no subvesical obstruction. An experimental model using dogs documented an unusually high degree of tolerance to colovesical fistula.[10]

Chronic recurrent epididymitis is an unusual complication of colovesical fistula. This report demonstrates that if a patient with recurrent epididymitis does not respond to appropriate medication, an aggressive diagnostic evaluation is needed that should consider the possibility of colovesical fistula. Chronic epididymitis occurred, very likely, because of the retrograde flow of infected urine into the vas deferens. Left partial epididymectomy was performed in this patient because of persistent left epididymal pain and induration of the epididymis. One would expect that recurrent epididymitis would occur more frequently in patients with colovesical fistula but would not be reported because other urologic or gastrointestinal symptoms dominate the clinical picture.

Colovesical fistula should not be treated with conservative methods. Removal of the fistula alone, without resection of the diseased segment of colon, is associated with a high rate of recurrence. A diverting procedure, including a diverting colostomy, usually does not result in spontaneous closure of the fistula.[11] A one-stage procedure including segmental resection of the bowel, frequently without partial cystectomy, has been recommended. In this patient, partial bladder resection was performed because the perivesical inflammatory process involved a substantial segment of the bladder wall. Some authors[12,13] report that interposition of the omentum between the bladder and bowel may improve surgical results. There are some instances in which a one-stage operative procedure is not advised—with an inflammatory process, with or without an advanced unresectable malignant lesion, or in high-risk patients. An end colostomy and mucus fistula are advisable under these circumstances.

Acknowledgment. Jim Latteier provided assistance in preparing this manuscript.

REFERENCES

1. Carpenter WS, Allaben RD, Kambouris AA: Fistula complicating diverticulitis of the colon. *Surg Gynecol Obstet* 1972; 134:625-628.
2. Couris GD, Block MD: Intestinovesical fistula. *Surgery* 1963; 54:736-742.
3. Van Pater WN, Bargan JA, Feldman MB: Regional enteritis. *Gasteroenterology* 1954; 26:347-350.
4. Mayo CW, Blunt CP: The surgical management of the complication of diverticulitis of the large intestine: analysis of 202 cases. *Surg Clin North Am* 1950; 30:1005-1012.
5. Pugh JI: On the pathology and behaviour of acquired non-traumatic vesico-intestinal fistula. *Br J Surg* 1964; 51:664-657.
6. Hafner CD, Ponka JL, Brush BE: Genitourinary manifestations of diverticulitis of the colon: a study of 500 cases. *JAMA* 1962; 179:76-78.
7. King MR, Beart RW, McIlrath DC: Colovesical and rectovesical fistulas. *Arch Surg* 1982; 117:680-683.
8. Godec CJ, Cass AS: Comparison of pressure measurements in the lower urinary and lower fecal pathways. *J Urol* 1980; 123:58-60.
9. Krompier A, Howard R, Macewen A, Natoli C, Wear JB: Vesicocolonic fistulas in diverticulitis. *J Urol* 1975; 115:664-666.
10. Heiskell CA, Ujiki GT, Beal JM: A study of experimental colovesical fistula. *Am J Surg* 1975; 129:316-318.
11. Shatila AH, Ackerman NB: Diagnosis and management of colovesical fistulas. *Surg Gynecol Obstet* 1976; 143:71-74.
12. Wein AJ, Malloy TR: Simple omental mobilization for genitourinary surgery. *Urology* 1980; 16:515-518.
13. Turner-Warwick R: The use of pedicle grafts in the repair of urinary tract fistulae. *Br J Urol* 1972; 44:644-656.

Stage II benign cystic teratoma with malignant squamous degeneration

VICKI SELTZER, MD, STEVE VOGL, MD

Malignant squamous degeneration of a benign cystic teratoma is a rare occurrence, fewer than 100 cases having been reported in the literature. If the tumor is more advanced than a stage I lesion, survival for longer than one year is not expected, and neither radiation nor chemotherapy alone has been demonstrated to improve the prognosis.[1,2] We present a patient with stage II disease who was treated with a unique regimen of surgery, radiation, and chemotherapy, and who died—free of disease—one and a half years following the diagnosis of a malignancy.

CASE REPORT

During an exploratory laparotomy for a pelvic mass, a 65-year-old woman was found to have a left ovarian tumor (Fig 1) that measured 14 cm. The tumor had metastasized to the vesicouterine septum and the serosal surface of the bladder. A total abdominal hysterectomy, bilateral salpingo-oophorectomy, and omentectomy were done on the basis of a diagnosis of stage II ovarian cancer. Although an aggressive surgical approach was taken, there was a 4-cm plaque of residual tumor left on the serosa of the bladder at the conclusion of surgery. Histologic studies showed squamous cell carcinoma arising from a benign cystic teratoma (Figs 2, 3).

Since traditional therapy for this lesion has not been successful for advanced disease, a new approach was sought. It was decided that since the patient had a squamous cell malignancy and the usual therapy used to treat ovarian cancers has not been shown to be effective, she should be treated with therapy known to be effective for a squamous cell gynecologic malignancy. She received ra-

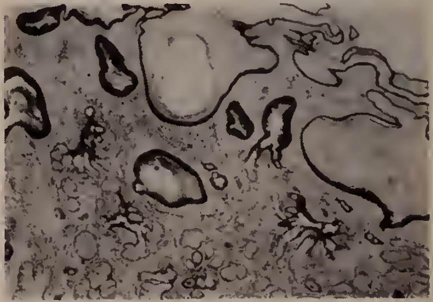


FIGURE 2. An area of the benign cystic teratoma demonstrating skin and skin appendages. (hematoxylin and eosin stain; original magnification X 45)

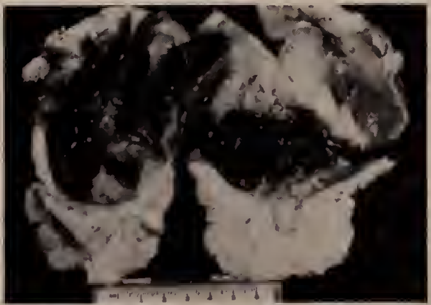


FIGURE 1. Surgical specimen of benign cystic teratoma with malignant degeneration (tumor bisected).

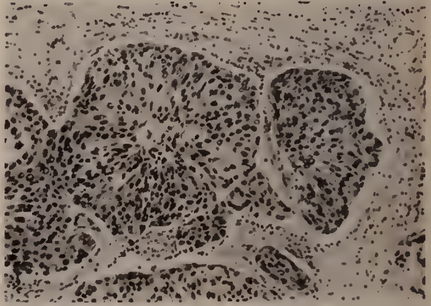


FIGURE 3. Poorly differentiated squamous cell carcinoma arising in the benign cystic teratoma. (hematoxylin and eosin stain; original magnification X 250)

From the Department of Obstetrics and Gynecology, SUNY-Stony Brook (Dr Seltzer), and the Department of Medicine, Division of Medical Oncology, Albert Einstein College of Medicine, Bronx, (Dr Vogl).

Address correspondence to Dr Seltzer, Director, Department of Obstetrics and Gynecology, Queens Hospital Center, 82-68 164th Street, Jamaica, NY 11432.

diation therapy: 5,400 rads to a 15 × 15 cm pelvic field plus a vaginal cesium insertion. She subsequently received methotrexate with leucovorin rescue, bleomycin, and *cis*-platinum.

The patient did well until 17 months after the initial surgery, when she was admitted to the hospital with small and large bowel obstruction which had occurred due to pelvic fibrosis and required laparotomy. She was found to have extensive pelvic adhesions and changes due to radiation, but no evidence of tumor was found. After the laparotomy bilateral bronchopneumonia developed. She suffered an acute myocardial infarction, and expired 18½ months after the tumor had originally been diagnosed.

At autopsy, severe abdominal adhesions were noted, and there was extensive pelvic fibrosis, but no evidence of tumor could be found.

DISCUSSION

Benign cystic teratomas with malignant degeneration are rare lesions. It has been suggested by Gordon et al[3] that those benign cystic teratomas that undergo malignant transformation are more likely to do so in the postmenopausal woman. Although the overall incidence of malignant transformation of a benign cystic teratoma is only approximately 1%, Gordon and colleagues found that of 16 benign cystic teratomas occurring in postmenopausal patients, three had undergone malignant transformation.[3] The malignant component of the teratoma is usually of squamous origin. Unless the tumor is confined to one ovary with no extracapsular spread, survival for more than one to two years is rare. In a review of the literature through 1977, Krumerman and Chung[2] reported only one case of benign cystic teratoma with malignant squamous degeneration in which the tumor had gone beyond the ovaries and the patient survived; she had a verrucous carcinoma, which is known to be more indolent.

The mainstay of treatment of this lesion is surgery. Past reports have concluded that neither postoperative chemotherapy nor radiation therapy will improve the prognosis.[1,2] Since the data available to us at the time of treatment planning indicated that surgery alone would not be curative, and neither radiation nor traditional chemotherapy for ovarian cancer would eradicate this tumor, we chose to treat this patient in a manner that is effective in another pelvic squamous cell malignancy, squamous cell cervical cancer. Due to the aggressive nature of the lesion, we utilized surgery, radiation, and chemotherapy. It was hypothesized that since this malignancy was a squamous cell lesion, its biologic behavior and response to chemotherapy might ultimately be more consistent with that of a squamous cell neoplasm than with that of other germ cell tumors.

In recent years, chemotherapy has been demonstrated to be effective against squamous cell cervical carcinoma.[4] Cavins and Geisler[5] reported a 50% response rate to a moderate dose of methotrexate. The Gynecologic Oncology Group study demonstrated a 50% response rate to *cis*-platinum for patients who had received no prior chemotherapy.[6] Miyamoto et al[7] found a complete remission rate of 80% using bleomycin and mitomycin-C. Vogl and colleagues[8] used bleomycin, methotrexate, and *cis*-platinum therapy to treat patients with advanced and recurrent cervical cancer; they demonstrated a response rate of 89%. Because of these statistics, these drugs are presently used in this institution to treat patients with advanced squamous cell gynecologic malignancies. We have added leucovorin rescue to this regimen in an attempt to diminish the toxicity of the methotrexate.

Because of the virulence of this type of tumor, and because chemotherapy regimens in the past have not been capable of controlling this lesion when it has extended beyond stage I disease, radiation therapy was added to the treatment regimen. There are data that indicate that chemotherapy combined with radiation therapy is effective in eradicating squamous cell gynecologic malignancies.[9]

This approach addresses the fact that perhaps the biologic behavior of these tumors is in keeping with the concept that they are actually squamous neoplasms.

REFERENCES

1. Pantoja E, Rodriguez-Ibanez I, Axtmayer RW, Noy MA, Pelegrina I: Complications of dermoid tumors of the ovary. *Obstet Gynecol* 1975; 45:89-94.
2. Krumerman MS, Chung A: Squamous carcinoma arising in benign cystic teratoma of the ovary. *Cancer* 1977; 39:1237-1242.
3. Gordon A, Rosenshein N, Parmley T, Bhagavan B: Benign cystic teratomas in postmenopausal women. *Am J Obstet Gynecol* 1980; 138:1120-1123.
4. Vogl SE, Seltzer V, Camacho F, Calanog A: Dianhydrogalactitol and cisplatin in combination for advanced cancer of the uterine cervix. *Cancer Treat Rep* 1982; 66:1809-1812.
5. Cavins JA, Geisler HE: Treatment of advanced, unresectable, cervical carcinoma already subjected to complete irradiation therapy. *Gynecol Oncol* 1978; 6:256-260.
6. Thigpen T, Shingleton H, Homesley H, Lagasse L, Blessing J: *Cis*-platinum in treatment of advanced or recurrent squamous cell carcinoma of the cervix. *Cancer* 1981; 48:899-903.
7. Miyamoto T, Takabe Y, Watanabe M, Terasima T: Effectiveness of a sequential combination of bleomycin and mitomycin-C on an advanced cervical cancer. *Cancer* 1978; 41:403-414.
8. Vogl SE, Moukhtar M, Kaplan BH: Chemotherapy for advanced cervical cancer with methotrexate, bleomycin, and *cis*-dichlorodiammineplatinum (II). *Cancer Treat Rep* 1979; 63:1005-1006.
9. Kalra J, Cortes E, Chen S, et al: Effective multimodality treatment for advanced epidermoid carcinoma of the female genital tract. *J Clin Oncol* (in press).

Posterior dislocation of the sternoclavicular joint

MARVIN J. WEINGARTEN, MD, ROBERT TASH, MD, ROBERT M. KLEIN, MD,
RICHARD J. KEARNS, MD

Dislocations of the sternoclavicular joint are uncommon injuries, accounting for 1% of all dislocations in the body.[1] The majority of these are anterior sternoclavicular dislocations; posterior dislocations are rare and may be difficult to diagnose.[2] We report on a patient with a posterior sternoclavicular dislocation and discuss the diagnosis of this injury.

CASE REPORT

A 13-year-old boy sustained blunt trauma to the upper chest in a motor vehicle accident. A chest radiograph taken at a local hospital

---

From the Departments of Radiology, (Drs Weingarten, Tash, and Klein) and Orthopedic Surgery (Dr Kearns), New York Medical College, Westchester County Medical Center, Valhalla, NY.

Address correspondence to Dr Weingarten, Assistant Professor, Department of Radiology, New York Medical College, Westchester County Medical Center, Valhalla, NY 10595.

demonstrated a widened mediastinum. He was transferred to Westchester County Medical Center for further treatment. The patient had no shortness of breath, dysphagia, or other complaints except severe right shoulder pain. On physical examination there was point tenderness of the right sternoclavicular joint with a relative depression of the medial end of the right clavicle compared to the left. He had pain referable to the joint upon movement of the right shoulder, especially flexion and abduction. The neurologic examination was unremarkable. His blood pressure and radial pulse were normal and equal in both arms. Laboratory data were unremarkable.

Radiographs, including anteroposterior and oblique views of the chest, as well as examination of the skull, cervical spine, right shoulder, and clavicle, showed a widened mediastinum but no apparent osseous abnormalities. A computed tomographic (CT) scan of the sternoclavicular joints (Fig 1) showed a retrosternal dislocation of the medial end of the right clavicle, and a mediastinal hematoma displacing the trachea to the left and causing slight tracheal compression. The patient underwent closed reduction under general anesthesia, after which his condition improved. A repeat CT scan the following day (Fig 2) showed a normal right sternoclavicular joint. He was discharged from the hospital with a figure-of-eight clavicular strap in place.

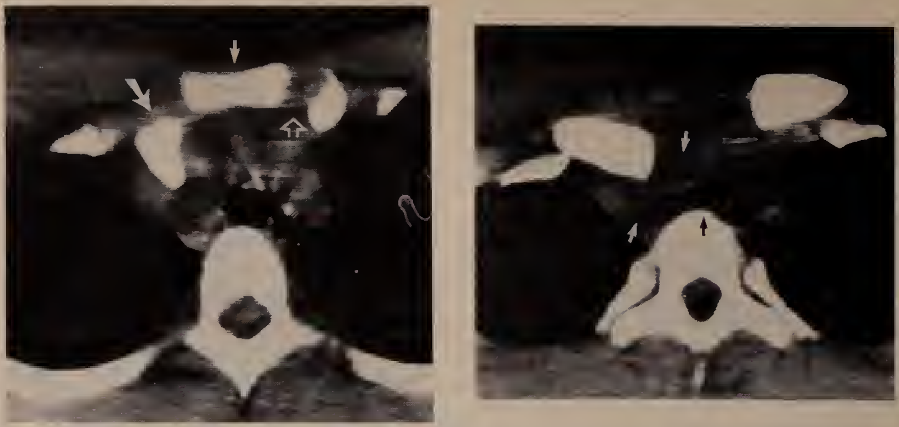


FIGURE 1. Left, CT showing right clavicular head (large arrow) lies posterior to sternum (small arrow). A normal left sternoclavicular joint is shown (open arrow). Right, a mediastinal hematoma (white arrows) compresses and distorts the trachea (black arrow).

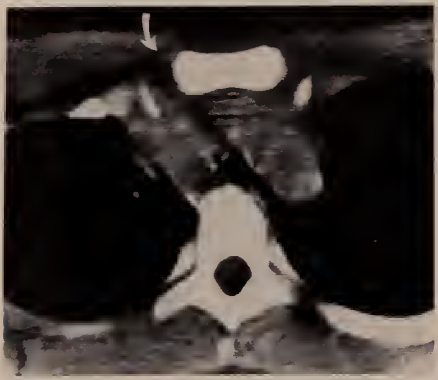


FIGURE 2. Normal right sternoclavicular joint (arrow) after closed reduction.

DISCUSSION

The sternoclavicular joint is a diarthrodial joint formed by the manubrium, the sternal end of the clavicle, and the first costal cartilage.[3] The joint is stabilized by its capsule, the costoclavicular and anterior and posterior sternoclavicular ligaments.[4] The posterior sternoclavicular ligament is thicker than the anterior sternoclavicular ligament, which, in part, explains why anterior sternoclavicular dislocations are more common than posterior dislocations.[2,5] Early diagnosis of a posterior dislocation is important, because closed reduction is seldom successful after 48 hours.[2,6] Also, the close proximity of the trachea, esophagus, and great vessels to the sternoclavicular joint makes posterior dislocation a potentially fatal injury which may cause respiratory or vascular compromise, or both.[6,7]

The diagnosis of posterior sternoclavicular dislocation is often difficult.[2] Frequently, the patient has suffered either a direct blow to the shoulder or a fall on the shoulder. There is usually pain at the joint which increases with any movement. The patient may simultaneously support and guard the injured arm while tilting the head ipsilaterally.[2,4] Early inspection of the chest may reveal loss of the normal contour on the injured side with an apparent depression of the skin over the sternoclavicular joint.[6] However, if a few hours have passed since the injury occurred, swelling of the overlying soft tissues may obscure this finding.[4] Patients may complain of hoarseness, dyspnea, dysphagia, or neurovascular changes in the injured upper extremity such as weakness and numbness.[2,4]

The radiographic diagnosis of this injury is frequently difficult as well. Standard radiographs are often not optimal for evaluation of the sternoclavicular joint because of its oblique orientation and the presence of multiple overlying structures.[8-10] Special views of the joint have been devised but are frequently not informative because they are difficult to obtain and interpret.[2,10,11] Conventional tomography may be helpful[12]; however, it is time consuming, and the images are often blurry. Transaxial computed tomographic scans are ideally suited to evaluation of the sternoclavicular joint.[10,11] They are easy to obtain—with little patient discomfort, are not overly time consuming, and provide excellent anatomic detail. In addition, they provide information regarding the underlying mediastinal structures, which can not be obtained with any other technique. Furthermore, the cumulative radiation dose to the patient is much lower than with the conventional linear tomography.[10]

Early diagnosis of posterior sternoclavicular dislocation is important, but requires a high index of suspicion. The physical examination and clinical findings may be confused by neurovascular changes, and standard radiographs are frequently misleading. Whenever disruption of the sternoclavicular joint is suspected, CT scans of the area should be performed.

REFERENCES

1. Nettles JL, Linscheid RL: Sternoclavicular dislocations. *J Trauma* 1968: 8:158-164.
2. Selesnick FH, Jablon M, Frank C, Post M: Retrosternal dislocation of the clavicle: report of four cases. *J Bone Joint Surg* 1984; 66A:287-291.
3. Gray H: *Gray's Anatomy*, 35th ed. Philadelphia, Saunders, 1973, pp 420-422.
4. Salvatore JE: Sternoclavicular joint dislocation. *Clin Orthop* 1968; 58:51-55.
5. Lee FA, Gwinn JL: Retrosternal dislocation of the clavicle. *Radiology* 1974; 110:631-634.
6. Post M: *The Shoulder: Surgical and Non-Surgical Management*. Philadelphia, Lea and Febiger, 1978, pp 488-500.
7. Elting JJ: Retrosternal dislocation of the clavicle. *Arch Surg* 1972; 104:35-37.
8. Abel MS: Symmetrical anteroposterior projections of the sternoclavicular joints with motion studies. *Radiology* 1979; 132:757-759.
9. Hobbs DW: Sternoclavicular joint: a new axial radiographic view. *Radiology* 1968; 90:801.
10. Destouet JM, Gilula LA, Murphy WA, Sagel SS: Computed tomography of the sternoclavicular joint and sternum. *Radiology* 1981; 138:123-128.
11. Levinsohn EM, Bunnell WP, Yuan HA: Computed tomography in the diagnosis of dislocations of the sternoclavicular joint. *Clin Orthop* 1979; 140:12-16.
12. Morag B, Shahin N: The value of tomography of the sterno-clavicular region. *Clin Radiol* 1975: 26:57-62.

Abdominal distention associated with lactose malabsorption in a premature infant

STUART BEREZIN, MD, ANITA BHOLE, MD, TZEE CHUNG WU, MD, LEONARD J. NEWMAN, MD

Abdominal distention is a common problem in premature infants. Delayed gastric emptying, sepsis, necrotizing enterocolitis, and intestinal obstruction are causes of this problem. We observed a premature infant who developed abdominal distention associated with lactose malabsorption. The problem resolved when the patient was started on a lactose-free formula.

CASE REPORT

A male infant was born to a 19-year-old woman at 29 weeks gestation after spontaneous labor. The birth weight was 1,135 grams. Apgar scores were 3 and 6 at one and five minutes. An initial chest film showed hyaline membrane disease. Mechanical ventilation was required for five days and oxygen therapy for two weeks. Hyperbilirubinemia developed during the first week requiring two days of light therapy.

At four weeks of age the baby developed abdominal distention. The baby was receiving 12 mL of Similac PM 60/40® (Ross) (1 g/kg of lactose) every three hours, plus peripheral hyperalimentation. The baby had been receiving smaller amounts of formula over the previous two weeks. On physical examination, the abdomen was tense, and bowel sounds were present. A stool culture was guaiac negative. A flat plate of the abdomen showed dilated bowel loops. Blood culture, urine culture, spinal fluid culture, and a chest film were unremarkable. Treatment with antibiotics was not begun.

Breath samples for analysis of hydrogen (H_2) concentration were collected before meals and at 30-minute intervals for three hours after feedings (the patient was being fed at three-hour intervals). Breath samples were obtained using an 8 French Argyle® feeding tube. The tube was placed in one nostril to rest in the nasopharynx. Two-mL aliquots of air were drawn into the syringe at the end of each expiration; 50 mL of breath were obtained at each testing. Breath samples were analyzed for hydrogen concentration by use of gas chromatography with a thermistor detector (Carle Instruments, Fullerton, CA). Informed consent was obtained prior to collecting breath samples.

Peak breath H_2 after a feeding of 12 mL of Similac PM 60/40® was 55 ppm. The stool was positive for reducing substance. Pregestimil® (Mead Johnson), a lactose-free formula, was substituted for the previous formula. Abdominal distention subsided within 24 hours. Peak breath H_2 was 4 ppm after a feeding of 12 mL of Pregestimil® (Fig 1). The stool was negative for reducing substance.

Two weeks later, the baby again received Similac PM 60/40®. Abdominal distention recurred after two feedings. Peak breath H_2 after 25 mL of Similac PM 60/40® (1.7 g/kg of lactose) was 86 ppm. The stool was positive for reducing substance. Antibiotic therapy was not given. Pregestimil® was again substituted for Similac PM 60/40®. Abdominal distention subsided within several hours. Peak breath H_2 after 25 mL of Pregestimil® was 5 ppm (Fig 2). The stool was negative for reducing substance.

DISCUSSION

In older children, various symptoms have been associated with lactose malabsorption. Incomplete lactose absorption leads to intraluminal fermentation of lactose by enteric flora, yielding H_2. Increased H_2 production may cause abdominal distention and bloating.[1]

Breath H_2 studies have shown that malabsorption of lactose occurs frequently in premature infants.[2] Previous studies have not described adverse symptoms associated with lactose malabsorption in premature infants. In this report, two episodes of abdominal distention were correlated with elevated breath H_2 levels while the baby was on a lactose containing formula.

It appeared that lactose malabsorption leading to increased H_2 production was the cause of abdominal distention in this patient. We cannot be certain whether the different protein and fat contents of the formulas were also important factors. Further studies will be required to determine if lactose malabsorption is a significant cause of abdominal distention in premature infants.

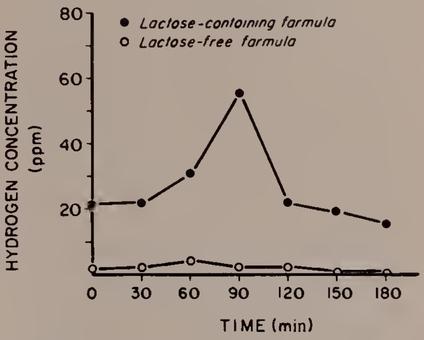


FIGURE 1. Breath hydrogen concentration after ingestion of 12 mL of formula at 4 weeks of age.

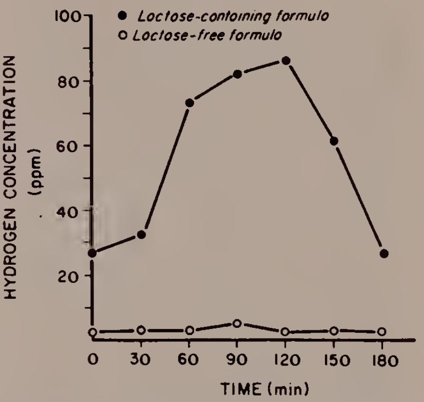


FIGURE 2. Breath hydrogen concentration after ingestion of 25 mL of formula at 6 weeks of age.

REFERENCES

1. Barr RG, Levine MD, Watkins JB: Recurrent abdominal pain of childhood due to lactose intolerance. *N Engl J Med* 1979; 300:1449-1452.

2. MacLean WC, Fink BB: Lactose malabsorption by premature infants: magnitude and clinical significance. *J Pediatr* 1980; 97:383-388.

From the Department of Pediatrics, Section of Gastroenterology and Nutrition, New York Medical College, Valhalla, NY.

Address correspondence to Dr Berezin, Assistant Professor, Department of Pediatrics, New York Medical College, Munger Pavilion, Valhalla, NY 10595.

MERRITT'S TEXTBOOK OF NEUROLOGY

Edited by Lewis P. Rowland, MD. 774 pp. Philadelphia, Lea & Febiger, 1984. $48.50 (hardcover)

Packed within 750 pages of tightly written, well edited text, this volume has regained its place as a choice reference for all neurologic diseases. The preface traces the transition from the masterful single author mode, so successful in the first four editions, to a multiauthored (71 contributors) production. This change has weathered the risk of many separate voices and remains a coherent, succint, and factual text. It is up-to-date—with numerous 1983 references—including sections on acquired immunodeficiency disease and on stroke in the young adult. The Merritt device of providing choice references rather than essays continues, permitting pithy presentations unflawed by excess dialogue. Consistent, too, is a seasoned approach to cause-and-effect, including therapies: the novice is in comfortable hands. Particularly effective is the expansion of the section on pediatric neurology, making the new Merritt an even more comprehensive source.

Credit for this admirable passage without sacrifice of simplicity must go to the editor, a Merritt veteran and now head of the Neurological Institute of New York at the Columbia-Presbyterian Medical Center. The authors include those associated at some time with the Neurological Institute and with Dr Merritt. While spanning subjects from chemistry to virology, a fundamental terse clinical discipline is preeminent. If useful for no other reason, this text should be a first reading for general care physicians as well as for neurologists interested in a sound, quick review and informative literature citations. There is little philosophy or physiology here, and this is not a "how to" for the medical student. This text remains a concrete guide for the informed, graduate clinician who is dealing with a demonstrably ill patient in a referral or hospital setting.

In a welcome move, however, an analysis of symptoms and signs—omitted in previous editions—has been explored, with a new introductory section. It is in this area that future editions may flower. Granted the virtues of brevity, some parts of the baby may have been thrown out with the bath water. It would be wise, for instance, to be more specific about sentinel hemorrhage in aneurysms and to iterate the needs for routine lumbar puncture. There remains a place for a discussion (preferably cryptic) of sensory loss. This section should contain a few words about the effects on extremities in cerebellar disorders, in addition to axial gait, and should emphasize the distinctions between inflow and outflow tremor. Most neurologic diagnoses emerge from a careful history, and the temporal profiles of disease splendidly diagrammed in pediatric differential diagnosis (by Dr. Rapin, p. 450) deserve to be moved to the front of the book with amplification to embrace adult diagnosis. There is no central or organized provision for the overlap between psychiatric and neurologic symptoms, an area that occupies the majority of office practice. The oft-repeated critique of Merritt, that one first had to know what the patient had before you could use the text, is acknowledged by the editor but has only been partially rectified.

A comparison of this edition with its analogue, the text of Adams and Victor, discloses that one complements the other. It would be a wise man who would consult both, but, if a difference be drawn, one (Adams) is more oriented to the foundations of neurology and its perspective and the other (Merritt by Rowland) to its operational practice. The third text in this market, by Gilroy and Meyer, is competent but does not profess the elegance of Adams and Victor and does not enter the arena of first-rate contributions from several dozen experts, offering high value for price.

BENNETT M. DERBY, MD
New York University
School of Medicine
New York City 10016

CLINICAL ANESTHESIA IN NEUROSURGERY

By Elizabeth A. M. Frost, MB, ChB. 480 pp, illustrated. Stoneham, MA, Butterworth Publishers, 1984. $49.95 (hardcover)

The stated purpose of this book is to discuss neurologic disease and to suggest rational approaches to anesthetic care. The book was written for anesthesiologists and neurosurgeons with the intent of fostering mutual understanding. The contributing authors include seven anesthesiologists, eight neurosurgeons, and one lawyer.

This book is divided into five sections: Cerebral Physiology and Evaluation, Neurosurgical and Related Procedures, Central Nervous System Trauma, Postoperative and Intensive Care, and Cerebral Death. The introduction includes a 10-page discussion of historical aspects of neuroanesthesia—which is unique compared to other neuroanesthesia texts. Also unique is the discussion of surgical considerations with respect to pain procedures and pediatric neurosurgery.

A disappointing feature of the book is the absence of illustrations (such as to show anatomy related to percutaneous cervical cordotomy), and the inclusion of illustrations that are of poor quality (shunt placement during carotid endarterectomy, for example) or that are not labeled and therefore not informative as they should be (such as the carotid-cavernous fistula angiogram, the craniosynostosis intraoperative photograph, anatomical drawings of transsphenoidal hypophysectomy, and intraoperative photographs of aneurysm clipping). Some simplistic illustrations are given a whole page, yet contribute little to an understanding of the text. In addition, under an illustration regarding ventricular tachycardia during brain stem manipulation, it is recommended that intravenous propranolol be given, with the caution that this treatment may not permit the anesthesiologist and surgeon to identify subsequent damage to the respiratory center. This treatment is not the recommended first-line approach for ventricular tachycardia and should not be advocated without supporting documentation.

There are many errors of omission in this text: enflurane and increased cerebrospinal fluid (CSF) production; techniques and complications of radiologic embolization procedures; adverse reactions to metrizamide myelography, therapeutic considerations, and treatment of patients to be anesthesized within 48 hours; hazards of shunting during carotid endarterectomy; upward herniation in patients with posterior fossa tumors; and the importance of appropriate opening pressures in CSF shunting.

In some instances, the pathophysiology and supporting documentation are not well developed; for example, the rationale for the use of intravenous and topical lidocaine for anesthesia induction in patients with intracranial hypertension, the controversy regarding epsilon aminocaproic acid therapy in subarachnoid hemorrhage, hepatic toxicity of anticonvulsants, and the description of how myelography is done in patients with acute spinal cord injury and the implications for anesthesia.

Many chapters contain a discussion of surgical techniques and their application and a discussion of anesthetic considerations, presumably written by neurosurgeons, since no anesthesiologist is cited.

Many items are not grouped appropriately, and the reader is not informed that a related topic will be discussed in a later chapter. For example, blood pressure transducer position during surgery in the seated position is mentioned in the section related to intracranial hypertension, but not in the section on cardiovascular complications; mortality in patients with vein of Galen aneurysms is only mentioned in a section on arteriovenous malformations. In one section, nitrous oxide is not recommended during anesthesia and surgery in the seated position. In a later section it is recommended that it be turned off during venous air embolism.

A few syntax and typographical errors occur throughout the book. In addition, the references for chapter 15 appear at the end of the next chapter.

Some statements are blatantly incorrect: "Dexamethasone administration may be palliative in cases of pituitary adenoma but not for adrenal adenomas or carcinoma." The dexamethasone suppression test is what should be discussed here, not therapy. In the discussion of postoperative care after hypophysectomy, the formula for calculating water deficit is incorrect; a similar discussion occurs later in a chapter on "Chronic Supportive Care," where the formula is correct.

In the chapter on cerebral blood flow, there is no mention of normal values for various locations in the brain, nor is there a discussion of the vascular anatomy of the spinal cord. Prophylactic antibiotics are recommended for carotid endarterectomy patients without supporting documentation. No mention is made of recent studies that support pre-treatment with either propranolol or captopril to reduce sodium nitroprusside requirements. A rapid infusion of 100 mL of fluid prior to placing a patient in the sitting position is hardly likely to "acutely increase the intravascular volume." The use of succinylcholine is advocated for patients with intracranial hypertension without a discussion of clinical or experimental studies or the controversy involved with the choice of muscle relaxants. It is recommended that a nasogastric tube be inserted to protect the airway in patients with stage IX and X nerve dysfunction.

In summary, this textbook has the worthy goal of promoting scientific understanding between anesthesiologists and neurosurgeons. Unfortunately, many chapters do not meet this stated purpose since courses of action that are advocated are merely listed, without addressing all aspects of these controversial issues. The novice reader may think that the anesthetic techniques advocated are universally accepted. Since in many instances there is no proof of the benefit versus risk, the reader could accept, evaluate, or reject the techniques described if a rationale for the choices were provided.

M. JANE MATJASKO, MD
University of Maryland Hospital
Baltimore, MD 21201

ALEXANDER FLEMING. THE MAN AND THE MYTH

By Gwyn Macfarlane. 304 pp. Cambridge, Harvard University Press, 1984. $20.00 (hardcover)

This fascinating and well written book combines a biography of Alexander Fleming, the discoverer of penicillin, with a biased and caustic view of his "inaction" during the 12 years that followed that remarkable find. The author, Gwyn Macfarlane, a clinical pathologist, accuses Fleming of not being aware of the importance of his initial observation until Florey, Chain and coworkers at Oxford "rediscovered the wonder drug" and expertly demonstrated its structure and clinical application.

The text is informative, but Macfarlane's prejudice is made obvious by the repeated references to Oxford—where he is a member of the staff—and his constant praise of Florey, at the expense of Fleming.

The reader is provided with a wealth of information, beginning with the serendipitous contamination of a culture plate by the marvelous mold and ending, after a 12-year hiatus, with a demonstration of its clinical efficacy.

Fleming deserved all the credit and worldwide acclaim he received for his initial observation. Florey, Chain, and the Oxford group deserved the praise received from the scientific community for the experiments they performed. I enjoyed the short case histories of the first five patients treated with penicillin and could feel the elation experienced by the investigators when the results became evident.

Undoubtedly, the clinical successes revived the profession's interest, and Fleming rode the crest of the wave created at Oxford. Macfarlane admits that Fleming's charisma helped him gain universal recognition while Florey, as was the custom then, shunned reporters and interviews. All three—Fleming, Florey, and Chain—deserved the admiration and gratitude showered on them. The Nobel prize committee overlooked their relatively minor human failings and gave the award to all three.

Without Fleming's perception, penicillin would not have been available to Florey and Chain. Without the expertise of the Oxford team, the medical profession might not have the present long list of antibiotics to treat and conquer infectious diseases.

Physicians in all fields will enjoy this book, and medical student readers will be introduced to the wonders of basic and clinical research.

JULIUS E. STOLFI, MD
State University of New York
Downstate Medical Center
New York City 11203

ALTERNATIVES IN HEALTH CARE DELIVERY: EMERGING ROLES FOR PHYSICIAN ASSISTANTS

By Reginald D. Carter, PHD, PA, Henry B. Perry, MD, PHD 388 pp. St. Louis, Warren H. Green, Inc. 1984. $42.50 (hardcover)

Any physician or administrator contemplating the employment of a primary care or surgical physician assistant would do well to read this comprehensive book. While there are 50 chapters and 85 contributors, the individual chapters are short and to the point. They are also replete with personal experiences and helpful hints for incorporating a physician assistant into a private practice or a host of specialty alternatives.

Citing a study on the cost effectiveness of physician assistants, one article notes that "75% of adult medical ambulatory visits and 90% of pediatric ambulatory visits can safely be delegated to nonphysician assistants and nurse practitioners." In addition, if those levels of nonphysician care were ever achieved, primary care costs could be reduced by 45%. This brings up the subject of the projected physician surplus (several authors respond to that predicament) and the future role of the physician assistant within the projected surplus.

Part 3 of this book addresses issues such as obtaining hospital privileges for the physician assistant, the legal aspects of hospital privileges, reimbursement for nonphysician providers and sample modifications of hospital bylaws that would allow the physician assistant hospital privileges. Specific articles deal with introducing the primary care or surgical physician assistant into the office setting, with employment guidelines including elements of a contract and employment of physician assistants as house staff. Competency evaluation for physician assistants in nonprimary care specialities is also discussed.

A host of clinical roles for primary care and surgical physician assistants is detailed in separate chapters. Included in the primary care grouping are sections on practice in a rural office, emergency departments, health maintenance organizations, occupational clinics, military units, prisons, and in academic medical centers. Sections of the book also deal with the role of physician assistants in fields such as internal medicine, family practice, and geriatrics. Surgical practice roles can be found in cardiovascular surgery, cardiothoracic surgery, renal transplant teams, burn units, anesthesia-critical care teams, and in orthopedic private offices. Physician assistants can also be junior surgical house officers in a community hospital. Descriptions of these roles are written combining the everyday experiences of physicians and physician assistants actually engaged in that particular type of practice.

In Part 1, the foundations of the physician assistant profession are explored. Articles cite studies of the quality of care delivered by the physician assistant as well as the basic philosophy of the primary care and surgical physician assistant. Training of these professionals is delineated in this section as well as in the appendices, and there is a chapter that deals with certification, registration, and licensure of physician assistants.

Many of the chapters have references for the articles and an index allows for easy location of pertinent material.

This book starts out with the concept that quality care can be delivered by well trained primary care and surgical physician assistants, and the authors convincingly prove their point.

Incidentally, for those who may have been as confused as I was, the editors explain current terminology. Formerly, the title used was physician's assistant, denoting a dependent role. To avoid intimations of dependency, the preferred terminology is physician assistant. While the "'s" is still used for surgical physician's assistant, the preferred term is surgical physician assistant.

ALLEN SPIEGEL, PhD, MPH
SUNY-Downstate Medical Center
New York City 11203

A PRACTICAL HANDBOOK OF JOINT FLUID ANALYSIS

By Robert A. Gatter, MD. 105 pp, illustrated. Philadelphia, Lea & Febiger, 1984. $22.50 (hardcover)

This book is a complete treatise on synovial fluid analysis (synovianalysis). The emphasis of the text is appropriate because the diagnosis of many acute and chronic joint diseases depends on the results of joint fluid analysis. These results are an essential part of evaluating patients with painful swollen joints. The differential diagnosis of joint diseases is critical because early diagnosis and treatment may mean the difference between a complete cure or later secondary degenerative disease with chronic pain and even the need for corrective surgery.

A current authoritative handbook on synovial fluid analysis is timely. The first such publication was by Ropes and Bauer in 1953. Hollander, Jessar, and McCarty updated synovial fluid analysis in the *Bulletin of Rheumatic Diseases* in 1961. An excellent chapter on synovial fluid analysis entitled "Laboratory diagnostic procedures," appeared in *Rheumatic Diseases*, edited by Cohen and published in 1967. Descriptions of the techniques of synovial fluid examination and differential diagnoses are found in all rheumatology textbooks but require library use or the purchase of expensive subspecialty texts.

The author is well qualified to write this book. He is the author of a number of articles dealing with crystal-induced synovitis. With McCarty, he first described hydroxyapatite crystals associated with recurrent synovitis.

While this handbook may be used primarily by rheumatologists and clinical pathologists, orthopedists, general internists, and particularly emergency room physicians should find it useful. It should be included in medical school libraries as a helpful reference for students.

The book contains just over 100 pages and is divided into 12 chapters and three appendices. The chapters describe joint aspiration techniques, gross changes in synovial fluid, white blood cell and differential counting, wet preparation, compensated polarized light and phase microscopy, identification of crystals, tissue fragments, debris and artifacts, staining techniques, and serologic and chemistry studies. One chapter is devoted to x-ray diffraction, transmission microscopy, electron probe analysis, and other sophisticated methods of crystal identification. Chapter 11 is devoted to the differential diagnosis of arthritis and findings of less common forms of this disorder. The last chapter and the appendices are technical and deal with compensated polarized light microscopy, reference laboratories, and the formulations for various chemical stains.

The book is well written, accurate, and quite readable. Each chapter ends with pertinent references. Its highlights are the magnificent color plates (13 pages in all) showing the various crystal types under compensated polarized microscopy. The second chapter, "Joint aspiration: indication and technique," would be better left to other texts. Only knee aspiration is described in this chapter, and this adds little to the book. The author has achieved his stated goals by providing an up-to-date, well illustrated, and comprehensive handbook of synovial fluid analysis.

LEE BARTHOLOMEW, MD
Albany Medical College
Albany, NY 12208

BOOKS RECEIVED

CARDIOLOGY

Bedside Cardiology, Third Edition. By Jules Constant, MD. 458 pp, illustrated. Boston, MA, Little, Brown & Co, 1985. $39.95 (clothcover)

Clinical Echocardiography. Case Studies: Hemodynamic and Pathological Correlations. By Jose Missri, MD. 420 pp, illustrated. New York, NY, Yorke Medical Books, 1984. $45.00 (hardcover)

The Heart: Physiology, Metabolism, Pharmacology, Therapy. By Lionel H. Opie, MD. 392 pp, illustrated. London, Grune & Stratton Ltd. 1984. $11.95 (hardcover)

COMPUTER TECHNOLOGY

Computer Applications in Clinical Practice. An Overview. Edited by Daniel Levinson, MD. 331 pp, illustrated. New York, NY. Macmillan, 1985. $29.95

Computers and Control in Clinical Medicine. Edited by Ewart R. Carson and Derek G. Cramp. 263 pp, illustrated. New York, NY, Plenum Publishing Corp, 1985. $39.50 (hardcover)

DERMATOLOGY

Chronic Ulcers of the Skin. By Bok Y. Lee, MD. 225 pp, illustrated. New York, NY, McGraw-Hill Book Co, 1985. $27.00 (hardcover)

GASTROENTEROLOGY

Inflammatory Bowel Disease. Edited by Tsuneo Shiratori & Hiroshige Nakano. 457 pp, illustrated. New York, NY, Columbia University Press, 1984. $64.50 (hardcover)

INTERNAL MEDICINE

Davidson's Principles & Practice of Medicine. Fourteenth Edition. Edited by John MacLeod. 843 pp, illustrated. New York, NY, Churchill Livingstone Inc, 1984. $28.00 (paperback)

Idiopathic Portal Hypertension. Edited by Kunio Okuda and Masao Omata. 606 pp, illustrated. New York, NY, Columbia University Press, 1984. $69.50 (hardcover)

Textbook of Nuclear Medicine, Volume I: Basic Science. Second Edition. Edited by John Harbert, MD, and Antonio Fernando Goncalves Da Rocha, MD. 526 pp, illustrated. Philadelphia, PA, Lea & Febiger, 1984. $80.00 (hardcover)

Textbook of Nuclear Medicine, Volume II: Clinical Applications. Second Edition. 724 pp, illustrated. Philadelphia, PA, Lea & Febiger, 1984. $95.00 (hardcover)

MISCELLANEOUS

Appleton's Review for National Boards. Part I. By Francis J. Chlapowski, PHD. 422 pp, illustrated. E. Norwalk, CT, Appleton-Century-Crofts, 1985. $29.95 (paperback)

Appleton's Review for National Boards. Part II. By Robin J. O. Catlin, MB. 434 pp, illustrated. E. Norwalk, CT, Appleton-Century-Crofts, 1985. $29.95 (paperback)

The Asthma Handbook: A Complete Guide for Patients and Their Families. By Stuart H. Young, MD, Susan A. Shulman, Martin D. Shulman, PHD. 272 pp, illustrated. New York, NY. Bantam Books, 1985. $3.95 (paperback)

Between Health & Illness: New Notions on Stress and the Nature of Well Being. By Barbara B. Brons, MD. 304 pp. New York, NY. Bantam Books, 1984. $3.95 (paperback)

Dr. Abravanel's Body Type Program for Health, Fitness and Nutrition. By Elliot D. Abravanel, MD, Elizabeth A. King. 320 pp, illustrated. New York, NY, Bantam Books, 1985. $15.95 (hardcover)

Joe Graedon's The New People's Pharmacy. Drug Breakthroughs of the 80's. The All-New Consumer's Guide That Answers Today's Most Pressing Health Questions. By Joe Gradeon, Teresa Graedon. 427 pp, New York, NY. Bantam Books, 1985. $8.95 (paperback)

Personal Pension Plan Strategies For Physicians. By C. Colburn Hardy and Howard J. Wiener, JD, LLM. 132 pp, Oradell, NJ, Medical Economics Books, 1985. $19.95 (hardcover)

The Pill Book of Arthritis. Drugs & Treatment. Edited by Lawrence D. Chilnick. 208 pp, illustrated. New York, NY, Bantam Books, 1985. $3.95 (paperback)

The Pill Book of High Blood Pressure. Drugs & Treatment. Edited by Lawrence D. Chilnick. 208 pp, illustrated. New York, NY, Bantam Books, 1985. $3.95 (paperback)

To Provide Safe Passage: The Humanistic Aspects of Medicine. Edited by David Rabin, MD, Pauline L. Rabin, MD. 258 pp. New York, NY. Philosophical Library, Inc,1985. $19.95 (hardcover)

The Revolutionary 7-Unit Low Fat Diet. By Audrey Eyton, Jean Carper. 256 pp, illustrated. New York, NY, Bantam Books, 1985. $3.95 (paperback)

Stand Tall! Every Woman's Guide to Preventing Osteoporosis. By Morris Notelovitz, MD, Marsha Ware. 208 pp, illustrated. New York, NY, Bantam Books, 1985. $7.95 (paperback)

Tax Strategy For Physicians. Third Edition. By Lawrence Farber. 256 pp. Oradell, NJ, Medical Economics Books, 1985. $25.95 (hardcover)

NEUROLOGY

Practical Management of Stroke. By Graham P. Mulley, MD. 167 pp. Oradell, NJ, Medical Economics Books, 1985. $18.95 (paperback)

OBSTETRICS AND GYNECOLOGY

The Biochemical Effects of Drugs in Pregnancy. Volume I. Drugs Active on the Nervous, Cardiovascular and Haemopoietic Systems. By A. Onnis, P. Grella. 396 pp, illustrated. Somerset, NJ. John Wiley & Sons, Inc, 1984. $88.95 (hardcover)

ONCOLOGY

Infectious Complications of Neoplastic Disease. Controversies in Management. Edited by Arthur E. Brown, MD, Donald Armstrong, MD. 448 pp, illustrated. New York, NY, Yorke Medical Books, 1985. $40.00 (hardcover)

ORTHOPEDIC SURGERY

Arthroscopy of the Knee. A Diagnostic Color Atlas. By David J. Dandy. 159 pp, illustrated. Philadelphia, PA. Lea & Febiger, 1984. $44.00 (hardcover)

PATHOLOGY

Practical Surgical Pathology. By Zeynel A. Karcioglu, MD, Ayten Someren, MD. 1,235 pp, illustrated. New York, NY, Macmillan, 1985. $150.00 (hardcover)

PREVENTIVE MEDICINE

The Value of Preventive Medicine. By Ciba Foundation Symposium 110. 258 pp, illustrated. London, Pitman Books, 1985. $35.00 (hardcover)

PSYCHIATRY

Major Depression. Prevention and Treatment. By Michael R. Lowry, MD. 120 pp. St. Louis, MO. Warren H. Green, Inc, 1984. $15.00 (hardcover)

Modern Synopsis of Comprehensive Textbook of Psychiatry/IV. Fourth Edition. By Harold I. Kaplan, MD, Benjamin J. Sadock, MD. 937 pp, illustrated. Baltimore, MD. Williams & Wilkins, 1985. $38.50 (paperback)

Schizophrenia: Straight Talk for Families and Friends. By Maryellen Walsh. 264 pp. New York, NY, William Morrow & Co, 1985. $15.95 (clothcover)

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Jacob Abramson, MD, Coconut Creek, Florida. Died October 17, 1984; age 79. Dr Abramson was a 1933 alumnus of Wayne State University College of Medicine. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Kings and the Medical Society of the State of New York.

William Dewey Alsever, MD, Syracuse. Died December 28, 1984; age 70. He was a 1939 alumnus of the State University of New York Upstate Medical Center. Dr Alsever was a Diplomate of the American Board of Pediatrics, and a member of the American Academy of Pediatrics, the Onondaga County Medical Society and the Medical Society of the State of New York.

Joseph Albert Barsa, MD, Pearl River. Died January 5, 1985; age 69. Dr Barsa was a 1945 alumnus of Georgetown University School of Medicine. He was a member of the American Psychiatric Association, the Academy of Psychoanalysis, the Medical Society of the County of Rockland and the Medical Society of the State of New York.

Carleton Campbell, MD, Wilton, Connecticut. Died November 30, 1984; age 89. Dr Campbell was a 1925 alumnus of the State University of New York Downstate Medical Center. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Harold S. Davenport, MD, Brooklyn. December 14, 1984; age 56. Dr Davenport was a 1952 alumnus of Meharry Medical College. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Armando De Cardenas, MD, New York. Died September 16, 1984; age 66. Dr De Cardenas was a 1944 graduate of Universidad de La Habana Facultad de Medicina y Farmacia, Havana. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

James A. Doucett, MD, Olean. Died December 20, 1984; age 62. He was a 1946 graduate of Cornell University Medical College. Dr Doucett was a Diplomate of the American Board of Pediatrics, and a member of the American Academy of Pediatrics, the Medical Society of the County of Cattaraugus and the Medical Society of the State of New York.

Frank Michael Ferrante, MD, Bronx. Died November 30, 1984; age 66. Dr Ferrante was a 1943 alumnus of the Medical College of Virginia. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

J. Vincent Flack, MD, Pelham Manor. Died January 17, 1985; age 81. He was a 1931 alumnus of the State University of New York Upstate Medical Center. Dr Flack was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Ophthalmology. He was a member of the American Academy of Ophthalmology and Otolaryngology, the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Abraham W. Freireich, MD, Malverne. Died January 26, 1985; age 79. Dr Freireich was a 1932 graduate of New York University College of Medicine. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the Academy of Medicine, the Nassau County Medical Society and the Medical Society of the State of New York.

Elmer Stewart Gais, MD, New York. Died December 28, 1984; age 84. He was a 1924 graduate of the State University of New York Upstate Medical Center. Dr Gais was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the Clinical Society of the New York Diabetes Association, the Academy of Medicine, the Endocrine Society, the New York County Medical Society and the Medical Society of the State of New York.

Maurice Gershman, MD, Delray Beach, Florida. Died January 5, 1985; age 74. He was a 1935 graduate of the University of Arkansas School of Medicine. Dr Gershman was a Fellow of the American College of Surgeons, a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. His memberships included the Academy of Medicine, the American Academy of Orthopaedic Surgeons, the Pan American Medical Association, the Nassau County Medical Society and the Medical Society of the State of New York.

Max Grainer, MD, New York. Died January 1, 1985; age 89. He was a 1922 graduate of the University of Toronto Faculty of Medicine, Toronto. Dr Grainer was a member of the New York County Medical Society and the Medical Society of the State of New York.

Joseph Haas, MD, New York. January 9, 1985; age 95. He was a 1917 graduate of Fordham University School of Medicine. Dr Haas was a Qualified Fellow of the International College of

Surgeons, and a member of the Academy of Medicine, the American Urological Association, the American Public Health Association, the New York County Medical Society and the Medical Society of the State of New York.

Mary Brickett Holt, MD, Bay Shore. Died January 4, 1985; age 81. Dr Holt was a 1931 alumna of Cornell University Medical College. She was a member of the American Psychiatric Association, the Suffolk County Medical Society and the Medical Society of the State of New York.

Kenneth Jones, MD, Geneva. Died December 13, 1984; age 61. He was a 1951 alumnus of the University of London Faculty of Medicine, London. Dr Jones was a member of the New York State Society of Anesthesiologists, the Academy of Medicine, the American Society of Anesthesiologists, Inc, the Ontario County Medical Society and the Medical Society of the State of New York.

Walter J. Karwowski, MD, New York Mills. Died January 17, 1985; age 73. Dr Karwowski was a 1936 alumnus of the State University of New York Upstate Medical Center. He was a Fellow of the American Academy of Family Physicians, and a member of the Academy of Medicine, the American Public Health Association, the Medical Society of the County of Oneida and the Medical Society of the State of New York.

Leon Katowicz, MD, New York. Died January 18, 1985; age 71. Dr Katowicz was a 1939 graduate of Uniwersytet Warszawski Akademie Medyczna, Warsawa. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Orthopedic Surgery. He was a member of the Academy of Medicine, the American Academy of Orthopaedic Surgeons, the New York County Medical Society and the Medical Society of the State of New York.

Thomas Levin, MD, Sarasota, Florida. Died December 26, 1984; age 84. Dr Levin was a 1922 alumnus of McGill University Faculty of Medicine, Montreal. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Phillip C.L.Y. Liverman, MD, Glens Falls. Died July 9, 1984; age 42. Dr Liverman was a 1973 graduate of the University of Virginia School of Medicine. He was a Diplomate of the American Board of Internal Medicine, and a member of the American Society of Nephrology, the Medical Society of the County of Warren and the Medical Society of the State of New York.

Sherman William McIlmoyl, MD, Daytona Beach, Florida. Died December 14, 1984; age 80. He was a 1923 graduate of Queens University Faculty of Medicine, Kingston. Dr McIlmoyl was a Fellow of the American College of Surgeons and a Qualified Fellow of the International College of Surgeons. He was a member of the Medical Society of the County of Rensselaer and the Medical Society of the State of New York.

Harry John Mellen, MD, Albany. Died December 21, 1984; age 71. Dr Mellen was a 1938 alumnus of the University of Pennsylvania School of Medicine. He was a Diplomate of the American Board of Obstetrics and Gynecology, and a member of the Medical Society of the County of Albany and the Medical Society of the State of New York.

Robert Stephen Millen, MD, Glen Head. Died January 7,1985; age 78. He was a 1932 graduate of the University of Virginia School of Medicine. Dr Millen was a Fellow of the American College of Obstetricians and Gynecologists and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the Academy of Medicine, the New York Obstetrical Society, the American Fertility Society, the Pan American Medical Association, the Nassau County Medical Society and the Medical Society of the State of New York.

Isidor Palais, MD, Bronx. Died January 3, 1985; age 91. He was a 1920 graduate of the State University of New York Downstate Medical Center. Dr Palais was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Urology. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Libby Pulsifer, MD, Rochester. Died December 15, 1984; age 85. Dr Pulsifer was a 1926 graduate of Rush Medical College. She was a Fellow of the American College of Gastroenterology, and a member of the Academy of Medicine, the New York State Society of Internal Medicine, the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Abner R. Robbins, MD, Tamarac, Florida. Died December 30, 1984; age 80. Dr Robbins was a 1929 graduate of the University of Pennsylvania School of Medicine. He was a Fellow of the American College of Cardiology, and a member of the New York Cardiological Society, the American Geriatrics Society, the Nassau County Medical Society and the Medical Society of the State of New York.

Julius Rudnick, MD, North Woodmere. Died December 28, 1984; age 79. He was a 1930 graduate of the St. Louis University School of Medicine. Dr Rudnick was a Fellow of the American Psychiatric Association and a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Samuel Edward Sinberg, MD, Miami, Florida. Died February 2, 1984; age 88. Dr Sinberg was a 1925 alumnus of Columbia University College of Physicians and Surgeons. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Arthur Robert Sohval, MD, New York. Died January 14, 1985; age 81. He was a 1929 graduate of Columbia University College of Physicians and Surgeons. Dr Sohval was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the Academy of Medicine, the American Fertility Society, the Endocrine Society, the New York County Medical Society and the Medical Society of the State of New York.

David Soloway, MD, Walpole, Massachusetts. Died October 12, 1984; age 78. Dr Soloway was a 1934 alumnus of the University of Western Ontario Faculty of Medicine, London. He was a member of the Academy of Medicine, the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089–1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), an abbreviated title of no greater than 40 spaces for use as a running foot, and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Systême Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear on the text. When there are six or fewer authors, all should be listed; where there are seven or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

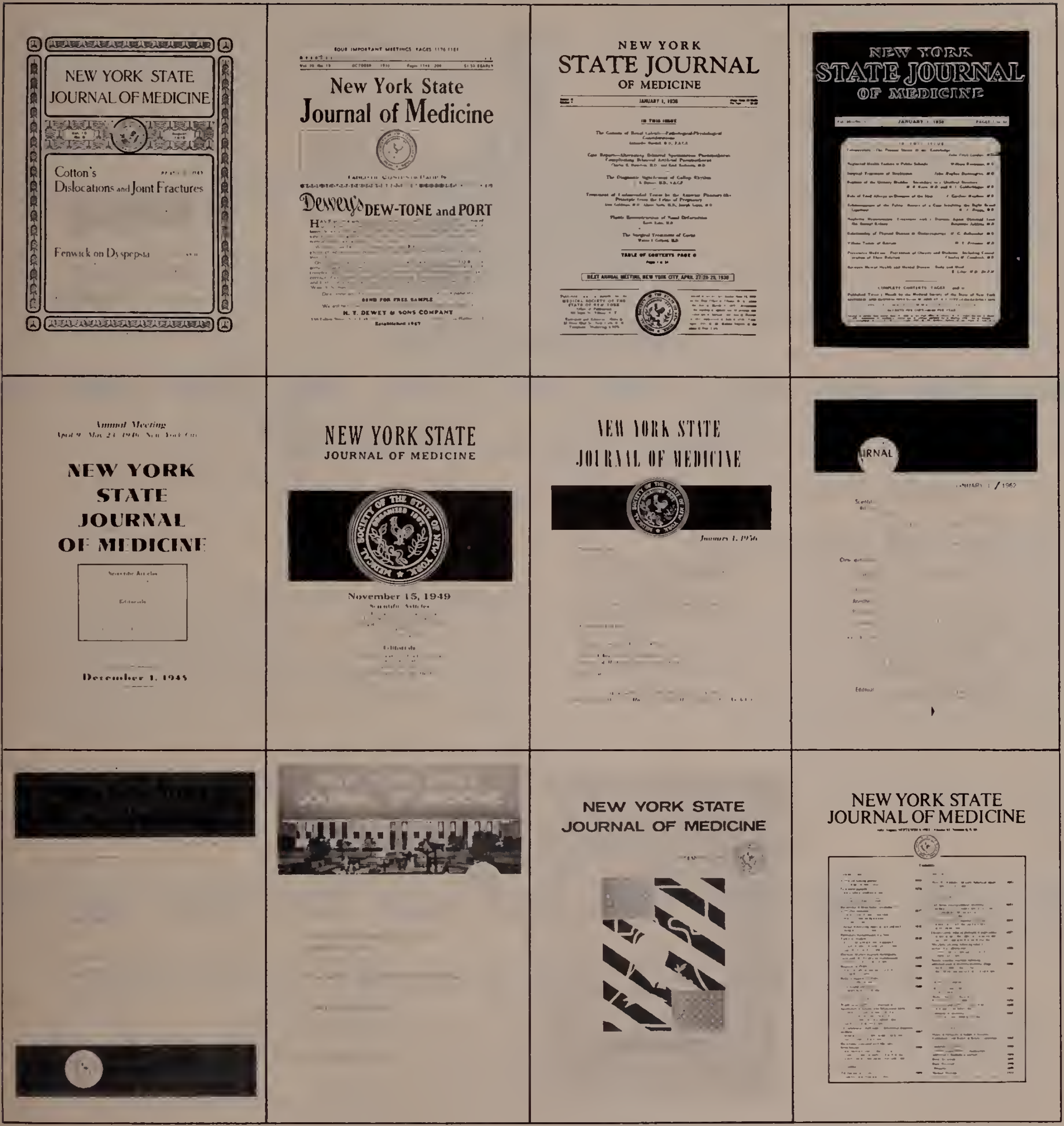
1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. McGuigan JE. Peptic ulcer. In: Petersdorf RG, Adams RD, Braunwald E, Isselbacher KJ, Martin JB, Wilson JD, eds. *Harrison's Principles of Internal Medicine.* New York, McGraw-Hill, 1983, pp 1697–1712.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Alan Blum, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

Cover to cover . . . . always good reading.



NEW YORK STATE JOURNAL OF MEDICINE

Cotton's Dislocations and Joint Fractures

Fenwick on Dyspepsia

New York State Journal of Medicine

Dewey's DEW-TONE and PORT

NEW YORK STATE JOURNAL OF MEDICINE

NEW YORK STATE JOURNAL OF MEDICINE

NEW YORK STATE JOURNAL OF MEDICINE

December 1, 1945

NEW YORK STATE JOURNAL OF MEDICINE

November 15, 1949

NEW YORK STATE JOURNAL OF MEDICINE

January 1, 1956

NEW YORK STATE JOURNAL OF MEDICINE

NEW YORK STATE JOURNAL OF MEDICINE

In 1985, the *New York State Journal of Medicine* promises to provide timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news. The journal will make a welcome gift to a colleague or student in the United States or abroad.

A free copy of the December 1983 issue, a special issue on smoking, will be included with each subscription while the supply lasts.

SUBSCRIPTION ORDER FORM Clip and mail

Circulation Department
New York State Journal of Medicine
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042

Name______________________________

Address______________________________

____

	1 Year	2 Years	3 Years
U.S. & Canada	☐ $25	☐ $45	☐ $60
Foreign	☐ $30	☐ $55	☐ $75

Payment required with order.

Classified Advertising

New York State Journal of Medicine

GENERAL INFORMATION

All classified advertisements are payable in advance, except for government agencies and advertising agencies; a 15% commission to recognized advertising agencies.

Frequency: Monthly
Mailing date: 15th of the month
Closing date: 30 days before mailing date

Circulation: 27,000 (mainly physicians & medical libraries)

DISPLAY ADVERTISING

	1 time	*6 time*	*12 time*	*Width*		*Depth*
1/4-page	$450	$400	$350	3-1/2″	×	4-7/8″
1/6-page	210	190	170	2-1/8″	×	4-7/8″
1/8-page	190	170	150	3-1/2″	×	2-3/8″
1/16-page	95	85	75	3-1/2″	×	1-1/8″
1/18-page	70	60	50	2-1/8″	×	1-1/2″

PARAGRAPH ADVERTISING

The first several words are set in bold face caps.

Rates: $25.00 per insertion for 50 words or less; additional words are 30 cents each. On request, Dept. numbers will be assigned by the Journal for replies to be forwarded to the advertiser. Dept. numbers are 50 cents per insertion. There is no frequency discount for paragraph ads.

Counting Words: Two initials, each abbreviation, figures consisting of 5 numerals or less, are counted as separate words. For replies, your name and address, or telephone number should appear at the end of the ad and be counted in the total number of words.

For further information, please call the Advertising Department at 516-488-6100.

CLASSIFIED ADVERTISING ORDER FORM

New York State Journal of Medicine
Attn: Classified Advertising
420 Lakeville Rd., P.O. Box 5404
Lake Success, N.Y. 11042

Date ____________________

Name ____

Address ____

City/State/Zip ____

Payment enclosed: $____________

Number of Insertions: ____________

Assign Dept. Number: ____________

Please make check payable to New York State Journal of Medicine.

Health Care Expo '85 is the most comprehensive educational program and exhibition of its kind ever undertaken. For one week this summer, participants will gather from around the world to engage in accredited programs, to attend open multidisciplinary seminars and symposia, and to visit acres of the most exciting and interesting health care exhibits in the world today.

An unprecedented opportunity to enrich your practice

More than three hundred one-hour courses providing Category I CME credits in thirty-four subject areas will be offered during the week. Designed by the Continuing Education Division of Georgetown University School of Medicine, the program features a distinguished, multidisciplinary faculty from throughout the country who will discuss state-of-the-art advances in a wide range of specialty and family practice areas. Each day's programming provides registrants, their families, or guests, with a rich choice of topics that can be selected according to their personal and professional interests. Courses and exhibits may be arranged on a daily and/or weekly basis to maximize time utilization and provide either an intensely focused learning experience or a broad overview of current developments. Call or write now for full details.

HealthCareExpo'85

Please send me a Registration Packet, including information on travel and lodging arrangements.

Name

Street Address

City State ZIP Country

(Area Code)
Telephone Number

MAIL TO: Health Care Expo '85, 404 Park Avenue South, N.Y., N.Y. 10016

or **CALL TOLL-FREE FOR IMMEDIATE RESPONSE:** (800) 221-3987
within New York State (212) 532-9400

MD-01R1

Join the Best and the Brightest at the Health Capital of the World

WASHINGTON, D.C. CONVENTION CENTER—AUGUST 18-24, 1985

- Accredited courses for physicians, nurses and allied health care professionals
- Informative industry health care exhibits for the whole family
- Displays by leading federal agencies and voluntary associations (including NIH, ACS, AHA)
- Health lectures for the lay public
- Presentations by government experts
- Enjoy the relaxing and stimulating vacation surroundings of Washington, D.C.

CME Courses

Aerospace Medicine/Allergy/Arthritis/Cardiology/Computers in Medicine/Cancer: (Colon, Breast, Gyn, Melanoma, Lung, Prevention)/Diabetes/Ethics in Medicine/Gastroenterology/Infectious Diseases/Lasers/Neurology/Obstetrics/Pain/Pre & Post Operative Care/Sexually Transmitted Disease/Sports Medicine/Trauma/Travel Medicine/Urology/Vascular Surgery

Distinguished Board of Advisors

Rufus R. Hessberg, MD, Aerospace Medical Ass'n./Sanford C. Milwit, American Cancer Society/John G. Leonardy, MD, American College of Allergists/Robert S. Bolan, PhD, American Diabetes Ass'n./Irving S. Wright, MD, American Federation for Aging Research/Linda Hiddemen Barondess, American Geriatrics Society/Dudley H. Hafner, American Heart Ass'n./Fred L. Allman, Jr., MD, American Orthopedic Society for Sports Medicine/Dallas F. Whaley, CAE/American Rheumatism Ass'n./Arthur Ulene, MD, Chairman, Lifetime; Commentator, NBC Today Show/ Matthew F. McNulty, Jr., ScD, The Medical Ctr., Georgetown University/George E. Schreiner, MD, Dir., Nephrology Div., Georgetown Univ. Medi. Ctr./R. Adams Cowley, MD, Maryland Inst. EMS/Howard Ennes, Natl. Ctr. for Health Education/Edward H. Van Ness, Natl. Health Council, Inc./Mary Larkin, RN, Natl. Intravenous Therapy Ass'n., Inc./C. Joseph Stetler, Pharmaceutical Manufacturers Ass'n./Gerald P. Murphy, MD, Roswell Park Memorial Institute/Howard A. Rusk, Jr., President, World Rehabilitation Fund, Inc./Hon. Paul Rogers, National Council on Patient Information and Education/Franklin Shaffer, EdD, Natl. League for Nursing/Mark R. Knowles, Natl. Pharmaceutical Council, Inc./John L. Quigley, N.E. Hospital Assembly, Inc./John A. Ruvane, Pharmaceutical Advertising Council, Inc.

Need A Temporary Physician?
During: vacations, CME's, illness, recruiting.
Want Free Time While You Practice Medicine?
Join CompHealth's Locum Tenens Physician Group.
For further information, call 412-741-3310.

CompHealth A Physician Group
WILSON ROSS, Regional Administrator
114 Centennial Ave., Sewickley, PA 15143

NATIONAL EMERGENCY SERVICES, INC. (N.E.S.)

Directorships, full- and part-time emergency department opportunities are available throughout New York State and the country for primary care physicians with ER experience. Enjoy a variety of practice settings, competitive remuneration, paid malpractice insurance, and flexible scheduling. For information and application materials contact the recruitment department at (516) 365-4010, (Outside N.Y. State 1-800-645-4848) or send C.V. to N.E.S., One Hollow Lane, Suite 304, Lake Success, N.Y. 11042.

Registrations must be received by June 10, 1985 for:

MIAMI-NICE POSTGRADUATE COURSE: ADVANCES IN INTERNAL MEDICINE

NICE, FRANCE June 23-28, 1985

Sponsored by: University of Miami School of Medicine and Universite de Nice, Faculte de Medicine

Course Directors: Jacques J. Bourgoignie, M.D.
Laurence B. Gardner, M.D. Rene Masseyeff, M.D.

Lectures and workshops presented by faculty from the University of Miami School of Medicine and the Universite de Nice, Faculte de Medicine will concentrate on the underlying pathogenesis and mechanism of disease for specific internal medical disorders, indications and contraindications for treatment and specific complications which may result from therapy. Practical patient problems and management will be focused upon in workshop sessions.

Course Schedule: (Monday through Friday) Didactic presentations from 8:00 am to 1:00 pm. Workshop sessions from 6:00 to 8:00 pm.

Course Hours: 30 AMA Category 1 hours (6 hours per day)

Course Fee: $525 (US Dollars)

For information contact: Division of CME, University of Miami School of Medicine, P.O. Box 016960 (D23-3), Miami, FL 33101. Telephone: (305) 547-6716.

CHIEF EXECUTIVE OFFICER

The Medical Society of the State of New York invites applications for the position of Executive Vice-President. The EVP is the Chief Executive Officer and directs the activities of the 27,000 member Society, located at Lake Success, N.Y.

Non-Physicians may apply. Salary and benefits negotiable. All replies will be considered in confidence.

Send Curriculum Vitae to:
Chairman, EVP Search Committee

Medical Society of the
State of New York
420 Lakeville Road
Lake Success, NY 11042

Index to Advertisers

Administrative Health Management ... 24A
Ayerst Laboratories ... 2A,3A,4A,28A,29A,30A
Blue Cross/Blue Shield of Greater NY ... 19A
Classified Advertising ... 5A,18A,20A,26A
CyCare ... 27A
Health Expo '85 ... 17A
Eli Lilly and Co. ... 2nd Cover
Marion Laboratories ... 9A,10A
Medical Quadrangle ... 22A
Roche Laboratories ... 6A,7A,8A, 3rd Cover, 4th Cover
Stuart Pharmaceuticals ... 25A,26A
30 Central Park South ... 21A
Upjohn Company ... 23A

From Blue Cross and Blue Shield of Greater N.Y.

The last claim form!

Introducing

Amicus 1™

MEDICAL OFFICE MANAGEMENT SYSTEM

A computer system that does something you could never do before

For over 50 years, Blue Cross and Blue Shield of Greater New York has been the most consistently innovative force in helping physicians and patients meet the financial demands of progressive medical care. Now, with advances in the technology of computer networking, we can help you do something never before possible. Amicus 1 lets you transmit claims directly by phone to our computer for faster, more efficient processing of our medical and Medicare B* claims.

Direct communication provides better service

Just as direct communication between people is preferred for accuracy, computer-to-computer communication reduces the potential for errors and delays. When you purchase Amicus 1, you gain significant benefits. Faster collections and fewer errors. Greater productivity. And significant relief of cash flow pain.

Handles most of your accounting and financial management functions

Of course, Amicus 1 does more than just send claims to our computer. It assists you in all your billing and accounts receivable needs. It provides up to the minute financial reports on your practice. And more. All with just a few short entries.

A complete system featuring the IBM® Personal Computer XT

We've developed a ready-to-use system featuring an IBM Personal Computer XT. In addition, Amicus 1 includes a high speed printer; a tape backup storage unit; and a fast modem for telephone data transmission and access to medical and financial services. The Amicus 1 system includes installation with a three-day training program for your office staff. It also includes an on-site service contract plus a special telephone response line available during business hours.

Fill out your last claim form.

Just complete and mail the coupon below. Or, call us at **212 490-4180** to learn how Amicus 1 can help reduce your paperwork, increase your productivity and improve your cash flow.

Tell me how Amicus 1 can work for my practice.

☐ Yes, I'd like to see a personal demonstration.

☐ Please send me more information. NYSJM

1. NAME

2. SPECIALTY

3. ADDRESS *(Street, city, state, ZIP code)*

4. TELEPHONE ( ) 5. OFFICE MANAGER

Send completed form to: **Medical Business Services**
P.O. Box 1707 Grand Central Station
New York, N.Y. 10163

Medical Business Services
Div. of Blue Cross® Blue Shield® of Greater New York

---

*Applies to the 16 counties where Blue Cross and Blue Shield of Greater N.Y. is the Medicare carrier
IBM® is a registered trademark of International Business Machines Corporation
Amicus 1™ is a trademark of Blue Cross and Blue Shield of Greater New York

Don't let your lungs go to pot.

Smoking marijuana is a lot more dangerous than you think And a lot less cool.

AMERICAN LUNG ASSOCIATION
The Christmas Seal People®

Space contributed by the publisher as a public service

A defense against cancer can be cooked up in your kitchen.

There is evidence that diet and cancer are related. Follow these modifications in your daily diet to reduce chances of getting cancer:

1. Eat more high-fiber foods such as fruits and vegetables and whole-grain cereals.

2. Include dark green and deep yellow fruits and vegetables rich in vitamins A and C.

3. Include cabbage, broccoli, brussels sprouts, kohlrabi and cauliflower.

4. Be moderate in consumption of salt-cured, smoked, and nitrite-cured foods.

5. Cut down on total fat intake from animal sources and fats and oils.

6. Avoid obesity.

7. Be moderate in consumption of alcoholic beverages.

No one faces cancer alone.
AMERICAN CANCER SOCIETY

REAL ESTATE FOR SALE OR RENT

GASTROINTESTINAL ENDOSCOPY SUITES. Rent by procedure or hour. Fully equipped with latest Olympus equipment including video. Full-time gastrointestinal assistants. "Park Avenue Brownstone" location. Endo-Center, Inc. (212) 751-0365.

WANTED: MEDICAL OFFICE BUILDINGS. As principals we are seeking "existing buildings"—fully tennanted by physicians, dentists and health care services. Contact: Paul Gellert, Gelco Associates, 155 West 68th St., N.Y., N.Y. 10023; (212) 724-7900.

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

OZONE PARK, QUEENS: Lower level of busy Medical Building, stable community, for any medical related activity. 3,400 square feet available. Will subdivide to suit and consider any arrangements. Very favorable terms, great opportunity. Call (718) 848-6790 days or (718) 835-5259 nights and weekends. Ample finders fee available.

BAY SHORE, leasing new luxurious medical building. Located near Southside Hospital. Minimum suite 740 sq. ft. T.V. Monitored hallway. Large parking area, handicapped designed. Individual gas heat, a/c-electric. (516) 665-4064, 10 A.M. to 5 P.M., Monday to Friday.

COUNTRY ESTATE: for gracious living or weekend retreat. Catskills. 100 miles from N.Y.C. 9 room ranch, large living room with fireplace, dining room, 4 bed rooms, 3 baths, superb kitchen with Jenair stove and microwave, family room, large storage cellar, separate 4 room guest house. 4 car garage, large barn, trout pond, bass pond, skiing, snowmobiling, deer, wild turkey, magnificent view. $239,500 (201) 391-4801 or (914) 351-2346.

PROFESSIONAL OFFICE FOR RENT, 5 large rooms, Hillside-150th Street. Partial paneling, carpeting, one block hospital, subway (E&F lines). Any specialty. Telephone (718) 297-5050.

FOREST HILLS, Cord Meyer area, all new 7-room medical suite, two baths, available immediately, near all transportation and parking. Call owner (516) 482-1782.

OFFICE SPACE AVAILABLE, Central Bergen County, NJ. Share expenses. Call Dr. Rhine (201) 836-7722.

GREENWICH VILLAGE—Near St. Vincent's Hospital. Plot suitable for building construction to accommodate medical offices. Rental $2,000 per month. Rockville Mgt. Corp., (212) 675-9249.

REAL ESTATE FOR SALE OR RENT—CONT'D

FIRST OFFERING: CENTRAL PARK WEST MEDICAL—86W. & C.P.W. Prime location, subway & buses at door. 100–12,000 sq. ft. for rent, time sharing, built to suit, doorman, concierge. MUSEUM WEST MEDICAL—79W. & Columbus. 1 suite remaining. LANDMARK MEDICAL—Carroll Gardens, Court St., join major radiology group. 2 suites built to suit available. FLUSHING MEDICAL ARTS CENTER—across from Flush. Hospital, suite available. Call Professional Enterprises award winning developers of Prof. Space (718) 229-8440.

AMITYVILLE-SOUTH. 1100 square feet office space for rent in established modern professional building. Centrally located at Nassau-Suffolk border, near major parkways, close to Brunswick Hospital. Ample private parking, private front and rear entrance, street level, accessible to handicapped. Available June 1st. Call Dr. Walters (516) 264-2610.

MEDICAL OFFICE BUILDING, Poughkeepsie, New York. Accessible all area hospitals. Attractive 2 levels with individual reception areas, administrative offices, examining rooms, private offices. Excellent condition, ample parking, $310,000.00. Contact Jeffery A. Croshier, Salesperson, Canterbury Agency, Inc., (914) 297-6650.

SPACE FOR RENT in new Medical Building. Orangeburg, Rockland County. (914) 359-1877.

GARDEN CITY, LUXURIOUS EXPANDED RANCH with living room, fire place, den, eat-in-kitchen, 5 bedrooms, 3 baths, central air conditioning. With five room spacious medical suite attached. Almost an acre of land, ample parking, prestigious area. Owner will take back mortgage at 2 percent below market rate. Asking $595,000.00. June Michel Real Estate, 1205 Franklin Ave., Garden City, NY 11530 (516) 248-9503.

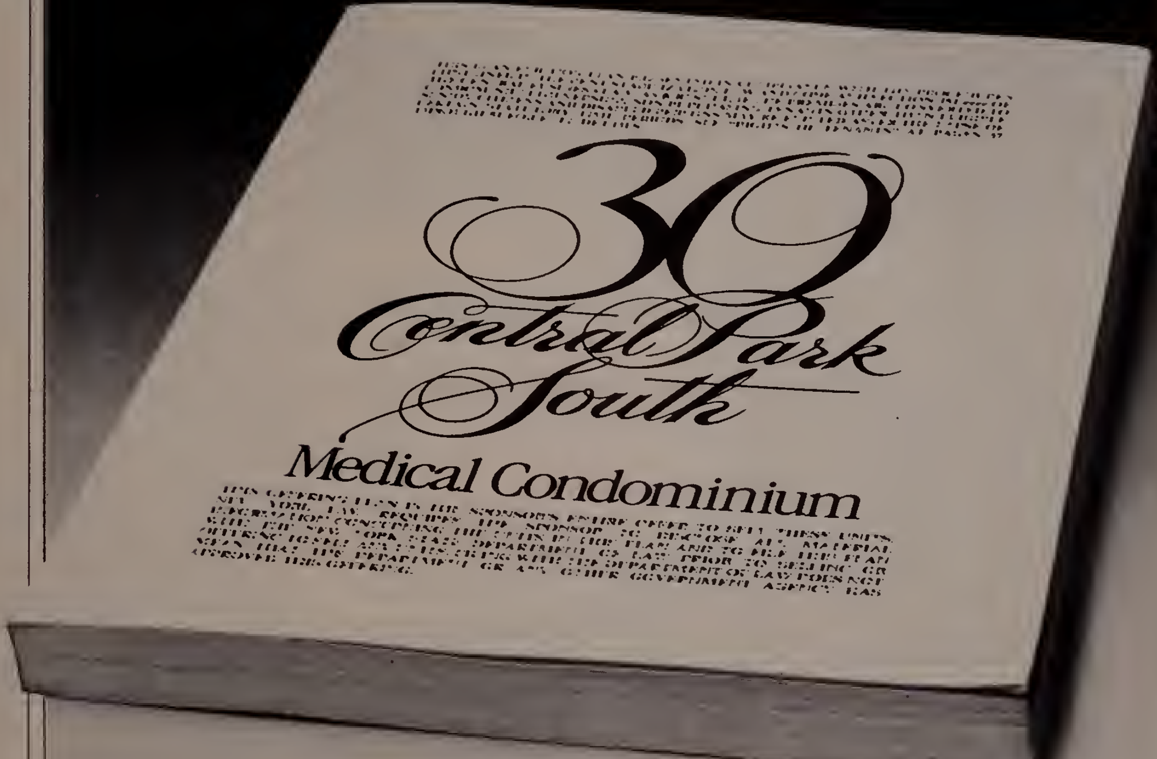
MISCELLANEOUS

LOOKING FOR ADDRESS OR PHONE NUMBER of Mrs. (Max) Henrietta Greenberg, mother-in-law of New York State Medical Doctor. Please call Eugene Sole, M.D. (718) 436-3726.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate, with no points, fees or charges of any kind. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

SEMINARS—HILTON HEAD ISLAND—Weekly Business Management Seminars. Accredited, live presentations. Key note lecture by Eliot Janeway. Spring, Summer and Fall Sessions at Sea Pines Plantation. 1-800-542-5428.

Medical breakthrough on Central Park South!



When the medical condominium offering plan was declared effective at 30 Central Park South, over 50% of the physicians and dentists who were "insiders" wisely decided to move forward and buy the offices they once only leased.

They must have known they couldn't find a better location or a better building for their midtown offices. But their real breakthrough came in discovering all the advantages of *owning* professional space.

If you would like to share in this amazing discovery, we're ready to show you how our current prices can work to your advantage as well. For a private appointment, please call 212-319-0951.

Sales and Marketing Agent: Gilbert · Charles · Beylen, Inc.
Sponsor: 30 Central Park South Realty Company
This advertisement is not an offering which can be made only by formal prospectus.

PHILIPS INTERNATIONAL

FOR PHYSICIANS AND SURGEONS WITH A PART-TIME EXPOSURE ONLY

Now offering Medical Malpractice Insurance on an ***occurrence basis*** at drastically reduced rates thru Frontier Insurance Co., Monticello, NY.

With the tail on claims made policies, you might lose your shirt.

Call or write for further information and application.

MEDICAL MQ QUADRANGLE

Medical Quadrangle, Inc.
P. O. Box 456
318 Bedford Road
Bedford Hills, New York 10507
914 666-0170
212 829-3654
"Specializing in Medical Liability Insurance"

continued from p 8A

Park Memorial Institute, 666 Elm St, Buffalo, NY 14263. Tel: (716) 845-2339.

NIAGARA

June 19–22. **Common Problems and Difficult Decisions in Hand Surgery.** Niagara Falls. Info: American Society for Surgery of the Hand, 3025 South Parker Rd, Ste 65, Aurora, CO 80014.

ONONDAGA

June 6–7. **34th Annual Otolaryngology Meeting.** Info: Program Coordinator CME, SUNY Upstate Medical Center, 750 East Adams St, Syracuse, NY 13210. Tel: (315) 473-4606.

ROCKLAND

June 4. **Footcare in the Diabetic.** *June 11.* **Pharmacological Intervention in Pain.** *June 25.* **Hypertension—Management Modalities.** Nyack. Info: Myrna Hahn, Nyack Hospital, N. Midland Ave, Nyack, NY 10960. Tel: (914) 358-6200, ext 2509.

ST. LAWRENCE

June 13. **Beta-Blockers in Hypertension.** Canton-Potsdam Hospital. Info: Sebastian A. Mazzotta, MD, Medical Society of the County of St. Lawrence, 53 South Main St, Norwood, NY 13668. Tel: (315) 353-2332.

AROUND THE NATION

CALIFORNIA

June 4–8. **49th Annual Scientific Meeting of the American Rheumatism Association (ARA) and the 20th Annual Scientific Meeting of the Arthritis Health Professions Association (AHPA).** Disneyland Hotel. Info: Cindy L. Thompson, 1314 Spring St, NW, Atlanta, GA 30309. Tel: (404) 872-7100.

FLORIDA

June 6–9. **Neonatal & Pediatric Respiratory Disease Conference.** Clearwater. 16 Cat 1 credits. Info: Myrtle E. Larson, RN, Gulf Coast Lung Association, 6160 Central Ave, St Petersburg, FL 33707. Tel: (813) 347-6133.

MARYLAND

June 3–7. **Mechanisms of DNA Damage & Repair.** Gaithersburg. Info: Ms. Kathy C. Stang, A353 Physics Building, National Bureau of Standards, Gaithersburg, MD 20899. Tel: (301) 921-2255.

MASSACHUSETTS

June 17–21. **Medical Technology Assessment for Health Professionals.** Cambridge. Info: Director of the Summer Session, Room E19-356, Massachusetts Institute of Technology, Cambridge, MA 02139. Tel: (617) 253-2101.

OHIO

June 7–8. **Critical Hemodynamic Diag-**

continued on p 24A

Motrin®

ibuprofen, Upjohn

600 mg Tablets

More convenient for your patients.

Upjohn

© 1984 The Upjohn Company

The Upjohn Company • Kalamazoo, Michigan 49001 USA

J-4044 January 1984

Increase your level of reimbursement
with AHM's vital seminar:

Insurance Coding:
Procedural CPT-85 and Diagnostic ICD-9-CM"

A one-day professional workshop designed to increase the proficiency of your staff in collecting what's due you. The latest revisions on specific California regulations.

Here are just a few of the techniques your staff will learn:
1. Why correct coding increases your level of reimbursement — now and in the future. 2. How the insurance carriers use coding. 3. How to recognize and code each procedural component. 4. Understanding relationships of diagnoses and procedures. 5. Step-by-step through the ICD-9-CM — where to begin. 6. How to avoid the "no-pay" and "desperation" codes.

• How coding will input into fee profiles of the future. • Who codes the procedures and diagnoses that you don't! • How the insurance carriers use codes to lower your reimbursement. • Recognition of the diagnosis from the hospital chart or office record. • Use of simplified terminology to relate procedures as they appear "on your records" to the CPT-85. • Defining levels of service for higher reimbursement. • Your patient's chart — how to organize it for ease of coding and office efficiency. • Keys to unlock the complexity of the ICD-9-CM.

Any or all of the following staff members will benefit from our seminar: Insurance Secretary, Receptionist, Patient Interviewer, Credit & Collections Counselor, Bookkeeper, Assistants, New Staff Members, Office Manager, Supervisors, Coordinators, and Doctors.

Eastern United States Seminar Schedule

Somerset, NJ	Apr. 29, 1985	Marriott Hotel • 110 Davidson Ave.
Saddle Brook, NJ	Apr. 30, 1985	Treadway Inn at Saddle Brook • 50 Kenney Pl.
White Plains, NY	May 1, 1985	White Plains Hotel • S. Broadway & Lyon Pl.
Long Island, NY	May 2, 1985	Long Island Marriott • 101 James Doolittle Blvd.
New York, NY	May 3, 1985	Grand Hyatt • Park Ave. at Grand Central
Boston, MA	May 7, 1985	Radisson Ferncroft • Rt. 1, Ferncroft Village
Providence, RI	May 8, 1985	Marriott Inn • 115 Charles & Orms Sts.
Springfield, MA	May 9, 1985	Sheraton Inn • 1080 Riverdale St. at I-91
Hartford, CT	May 10, 1985	Sheraton-Hartford • Trumbull St. & Civic Center Plaza
Philadelphia, PA	May 21, 1985	Sheraton-Valley Forge Hotel • First Ave. & Hwy. 363
Baltimore, MD	May 22, 1985	Cross Keys Inn • 5100 Falls Road
Washington, D.C.	May 24, 1985	The Washington Hilton • 1919 Connecticut Ave. NW
Rochester, NY	June 10, 1985	Marriott Inn-Thruway • 5257 W. Henrietta Rd.
New Haven, CT	June 12, 1985	Park Plaza & Convention Ctr. • 155 Temple St.
Long Island/ Smithtown, NY	June 13, 1985	Sheraton Smithtown • 110 Vanderbilt Motor Parkway
New York, NY	June 14, 1985	Grand Hyatt New York • Park Ave. at Grand Central

SPECIAL SEMINAR: AVOID MALPRACTICE SUITS!
Medical/Legal Guidelines for Today's Medical Office Staff
Protect yourself from legal liabilities. This seminar addresses everyday issues that affect your practice.

Washington, D.C.	June 24, 1985	Crystal City Marriott • 1999 Jefferson Davis Highway
Baltimore, MD	June 25, 1985	Cross Keys • 5100 Falls Road
Philadelphia, PA	June 26, 1985	Sheraton-Valley Forge • Route 363
Saddlebrook, NJ	July 29, 1985	Treadway Inn • 50 Kenney Place
Long Island, NY	July 30, 1985	Sheraton Smithtown • 110 Vanderbilt Motor Parkway
New York, NY	July 31, 1985	Plaza Hotel • Fifth Avenue at 59th St.

All seminars are taught by our highly qualified AHM staff members, backed by our company's 22 years experience of counseling in almost all facets of the health care industry. Tuition includes a comprehensive work-book/reference manual, sample forms and resource material plus refreshments. Seminar is tax deductible and is fully guaranteed or your tuition will be refunded if you are not completely satisfied. Over 38,700 physicians and their support staff have attended AHM seminars nationwide.

Fee: $135.00 each for one attendee from your practice.
$115.00 each for two or more attendees from your practice.

Attendance is limited...please register early!
Register: Call Toll Free 800-543-4332

ADMINISTRATIVE HEALTH MANAGEMENT GROUP, INC.
2600 Far Hills Avenue • Dayton, Ohio 45419

continued from p 22A

nosis, Monitoring & Management. Akron. Info: Registrar, American College of Physicians, 4200 Pine St, Philadelphia, PA 19104. Tel: (215) 243-1200.

PENNSYLVANIA

June 5–7. **Current Copncepts in Oncology and Hematologic Malignancies.** Philadelphia. Info: Registrar, American College of Physicians, 4200 Pine St, Philadelphia, PA 19104. Tel: (215) 243-1200.

VIRGINIA

June 19–23. **1985 Annual Pennsylvania Allergy Conference.** Williamsburg. Info: Mr. Walter F. Fullam, 500 University Dr, Hershey, PA 17033. Tel: (717) 534-6489.

AROUND THE WORLD

CANADA

June 10–14. **Advances in Internal Medicine, Selected Topics.** Banff, Alberta. Info: Registrar, American College of Physicians, 4200 Pine St, Philadelphia, PA 19104. Tel: (215) 243-1200.

DENMARK

June 19–22. **Satellite Symposia on Genetic Toxicology of the Diet.** Copenhagen. Info: John H. Weisburger, PHD. Naylor Dana Institute for Disease Prevention, Dana Rd, Valhalla, NY 10595. Tel: (914) 592-2600.

FINLAND

June 30–July 2. **Monitoring of Occupational Exposure to Genotoxicants.** Helsinki. Info: John H. Weisburger, PHD, Naylor Dana Institute for Disease Prevention, Dana Rd, Valhalla, NY 10595. Tel: (914) 592-2600

HAWAII

June 17–21. **Symposium on the Temporomandibular Joint.** Maui. Info: Dept of Radiology, University of California, San Francisco, CA 94143.

Angina comes in many forms...

So does SORBITRATE®

(ISOSORBIDE DINITRATE)

Unsurpassed flexibility in nitrate therapy.

© 1985 ICI AMERICAS INC.

See following page for brief summary of prescribing information.

SORBITRATE™
(ISOSORBIDE DINITRATE)

Please consult full prescribing information before use. A summary follows:

INDICATIONS AND USAGE: SORBITRATE (isosorbide dinitrate) is indicated for the treatment and prevention of angina pectoris. All dosage forms of isosorbide dinitrate may be used prophylactically to decrease frequency and severity of anginal attacks and can be expected to decrease the need for sublingual nitroglycerin.

The sublingual and chewable forms of the drug are indicated for acute prophylaxis of angina pectoris when taken a few minutes before situations likely to provoke anginal attacks. Because of a slower onset of effect, the oral forms of isosorbide dinitrate are not indicated for acute prophylaxis.

CONTRAINDICATIONS: SORBITRATE is contraindicated in patients who have shown purported hypersensitivity or idiosyncrasy to it or other nitrates or nitrites. Epinephrine and related compounds are ineffective in reversing the severe hypotensive events associated with overdose and are contraindicated in this situation.

WARNINGS: The benefits of SORBITRATE during the early days of an acute myocardial infarction have not been established. If one elects to use organic nitrates in early infarction, hemodynamic monitoring and frequent clinical assessment should be used because of the potential deleterious effects of hypotension.

PRECAUTIONS: General: Severe hypotensive response, particularly with upright posture, may occur with even small doses of SORBITRATE. The drug should therefore be used with caution in subjects who may have blood volume depletion from diuretic therapy or in subjects who have low systolic blood pressure (eg, below 90 mmHg). Paradoxical bradycardia and increased angina pectoris may accompany nitrate-induced hypotension. Nitrate therapy may aggravate the angina caused by hypertrophic cardiomyopathy.

Marked symptomatic, orthostatic hypotension has been reported when calcium channel blockers and organic nitrates were used in combination. Dose adjustment of either class of agents may be necessary.

Tolerance to this drug and cross-tolerance to other nitrates and nitrites may occur. Tolerance to the vascular and antianginal effects of isosorbide dinitrate or nitroglycerin has been demonstrated in clinical trials, experience through occupational exposure, and in isolated tissue experiments in the laboratory. The importance of tolerance to the appropriate use of isosorbide dinitrate in the management of patients with angina pectoris has not been determined. However, one clinical trial using treadmill exercise tolerance (as an end point) found an 8-hour duration of action of oral isosorbide dinitrate following the first dose (after a 2-week placebo washout) and only a 2-hour duration of effect of the same dose after 1 week of repetitive dosing at conventional dosing intervals. On the other hand, several trials have been able to differentiate isosorbide dinitrate from placebo after 4 weeks of therapy and, in open trials, an effect seems detectable for as long as several months.

Tolerance clearly occurs in industrial workers continuously exposed to nitroglycerin. Moreover, physical dependence also occurs since chest pain, acute myocardial infarction, and even sudden death have occurred during temporary withdrawal of nitroglycerin from the workers. In clinical trials in angina patients, there are reports of anginal attacks being more easily provoked and of rebound in the hemodynamic effects soon after nitrate withdrawal. The relative importance of these observations to the routine, clinical use of isosorbide dinitrate is not known. However, it seems prudent to gradually withdraw patients from isosorbide dinitrate when the therapy is being terminated, rather than stopping the drug abruptly.

Information for Patients: Headache may occur during initial therapy with SORBITRATE. Headache is usually relieved by the use of standard headache remedies or by lowering the dose and tends to disappear after the first week or two of use.

Drug Interactions: Alcohol may enhance any marked sensitivity to the hypotensive effect of nitrates.

Isosorbide dinitrate acts directly on vascular smooth muscle; therefore, any other agent that depends on vascular smooth muscle as the final common path can be expected to have decreased or increased effect depending on the agent.

Carcinogenesis, Mutagenesis, Impairment of Fertility: No long-term studies in animals have been performed to evaluate the carcinogenic potential of this drug. A modified two-litter reproduction study in rats fed isosorbide dinitrate at 25 or 100 mg/kg/day did not reveal any effects on fertility or gestation or any remarkable gross pathology in any parent or offspring fed isosorbide dinitrate as compared with rats fed a basal-controlled diet.

Pregnancy Category C: Isosorbide dinitrate has been shown to cause a dose-related increase in embryotoxicity (increase in mummified pups) in rabbits at oral doses 35 and 150 times the maximum recommended human daily dose. There are no adequate and well-controlled studies in pregnant women. SORBITRATE should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when SORBITRATE is administered to a nursing woman.

Pediatric Use: The safety and effectiveness of SORBITRATE in children has not been established.

ADVERSE REACTIONS: Adverse reactions, particularly headache and hypotension, are dose-related. In clinical trials at various doses, the following have been observed.

Headache is the most common (reported incidence varies widely, apparently being dose-related, with an average occurrence of about 25%) adverse reaction and may be severe and persistent. Cutaneous vasodilation with flushing may occur. Transient episodes of dizziness and weakness, as well as other signs of cerebral ischemia associated with postural hypotension, may occasionally develop (the incidence of reported symptomatic hypotension ranges from 2% to 36%). An occasional individual will exhibit marked sensitivity to the hypotensive effects of nitrates and severe responses (nausea, vomiting, weakness, restlessness, pallor, perspiration, and collapse) may occur even with the usual therapeutic dose. Drug rash and/or exfoliative dermatitis may occasionally occur. Nausea and vomiting appear to be uncommon. Case reports of clinically significant methemoglobinemia are rare at conventional doses of organic nitrates. The formation of methemoglobin is dose-related and, in the case of genetic abnormalities of hemoglobin that favor methemoglobin formation, even conventional doses of organic nitrate could produce harmful concentrations of methemoglobin.

DOSAGE AND ADMINISTRATION: For the treatment of angina pectoris, the usual starting dose for sublingual SORBITRATE is 2.5 to 5 mg, for chewable tablets, 5 mg; for oral (swallowed) tablets, 5 to 20 mg, and for controlled-release forms, 40 mg.

SORBITRATE should be titrated upward until angina is relieved or side effects limit the dose. In ambulatory patients, the magnitude of the incremental dose increase should be guided by measurements of standing blood pressure.

The initial dosage of sublingual or chewable SORBITRATE for prophylactic therapy in angina pectoris patients is generally 5 or 10 mg every 2 to 3 hours. Adequate controlled clinical studies demonstrating the effectiveness of chronic maintenance therapy with these dosage forms have not been reported.

SORBITRATE in oral doses of 10 to 40 mg given every 6 hours or in oral controlled-release doses of 40 to 80 mg given every 8 to 12 hours is generally recommended. The extent to which development of tolerance should modify the dosage program has not been defined. *The oral controlled-release forms of isosorbide dinitrate should not be chewed.*

DOSAGE FORMS AVAILABLE: Sublingual Tablets (2.5, 5, 10 mg); Chewable Tablets (5, 10 mg). Oral Tablets (5, 10, 20, 30, 40 mg), Sustained Action Tablets (40 mg)

STUART PHARMACEUTICALS
Division of ICI Americas Inc.
Wilmington, DE 19897

STR-2282

MISCELLANEOUS—CONT'D

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuariat certification and employee statements of participation—partnership agreements and professional incorporation—No insurance required—references upon request—Jack Wachstock, PC, 3000 Marcus Ave., Lake Success, N.Y. 11530 (516) 437-4848.

DOCTOR, YOU CAN'T BEAT the quality or the price! Holter Monitor Scanning Service. Physician owned, trained and supervised. No contracts to sign. We can arrange for lease/purchase of Holter equipment. New dual channel recorders with test cables $1,550. Now using UP Service for faster turn around time. DCG Interpretation (313) 879-8860.

ARCHITECTURE & PLANNING, Comprehensive services from analysis of alternative office locations through design and construction. Experienced in both new construction and renovation. Lewis & Gould (212) 807-6588.

1985 CME CRUISE/CONFERENCES on Selected Medical Topics—Caribbean, Mexican, Hawaiian, Alaskan, Mediterranean. 7-14 days year-round. Approved for 20-24 CME Cat. 1 credits (AMA/PRA) & AAFP prescribed credit. Distinguished professors. Fly Roundtrip Free on Caribbean, Mexican & Alaskan Cruises. Excellent group fares on finest ships. Registration Limited. Pre-scheduled in compliance with present IRS requiremnets. Information: International Conferences, 189 Lodge Ave., Huntington Station, NY 11746. (516) 549-0869.

AVOID MALPRACTICE SUITS—Free 30 Day Trial "Guidelines For Medical and Surgical Emergencies". Write professional type orders for laboratory and treatment. Masson Publishing Company—U.S.A., 133 East 58th Street, New York, NY 10022.

ARCHITECTURAL DESIGN AND CONSTRUCTION: Designers, builders of custom professional offices. Consulting services available. Whether you're opening a new office, relocating, remodeling or expanding an existing office in New York City area and adjacent New Jersey counties. Donald Young, Inc. Tel: (718) 948-5191.

BORROW $50,000 (or less) on your signature alone. Doctor? Lawyer? Executive earning $40,000? You could qualify for Profesco Professional Funding. No pre-payment penalty; Competitive rates; Financing arranged in 72 hours. For details; application: M. Fisher, Account Executive, John Hancock-Profesco, Suite 505, 3601 Hempstead Turnpike, Levittown, NY 11756, (516) 579-4455 or reply Dept. 381 c/o NYSJM.

We've been studying medicine almost as long as you have.

Over 17 years and untold manhours. That's what CyCare has invested in the study of health care. We've long since earned our diploma. While many aspiring competitors failed to make the grade.

We chose one specialty. The delivery of health care is a specialized business. Your data processing company should understand it thoroughly. That's tough to do if they're also marketing to banks, factories and the like.

From the beginning, CyCare decided to commit only to the medical industry. Our staff of over 600 has been living and breathing it ever since.

Experience only CyCare can claim. CyCare has studied with thousands of physicians, administrators, and nurses. We've worked with nearly 850 clients of all specialties and size. In 17 years, we've treated data processing challenges of every kind.

You know experience is the best teacher. So choose a company that's been around long enough to learn.

Our knowledge benefits you. Our experience taught us that each client is different. We designed systems that easily accomodate any type of case. We learned you didn't want useless "bells and whistles", so we developed practical software that enhances the delivery of patient care. We discovered the fear of system obsolescence. So we created modular systems that can be expanded at any time.

Learning never stops. Like you, we never stop learning. We invest more in research each year than most of our competitors gross in sales. We listen to your ideas, look for new ways to improve your practice, and stay abreast of industry needs. It's the only way to take the lead.

Compare our credentials. Examine CyCare thoroughly. Demand as much from us as you demand from yourself. Look at our experience, our financial stability. Examine our products and talk to our clients. Find out why CyCare is the leading supplier to medical group practices, HMOs and ambulatory care facilities nationwide.

Put us to the test. We're prepared.

Ask about CyCare's C100 APPOINTMENT SCHEDULING. The physician time management system for small and medium size practices.

☐ Rush free details to me about CyCare.

☐ Have a representative contact me.

My business card/letterhead is attached.

No. of Phys.____

Mail to: CyCare, 520 Dubuque Building Dubuque, Iowa 52001
319/556-3131
NYSJM 5/85

Sales and Service Offices:
Atlanta, GA, Cherry Hill, NJ. Chicago, IL, Dallas, TX; Denver, CO; Miami, FL Minneapolis, MN: New York, NY, Portland, OR: San Diego CA; Spokane, WA: Canada: Toronto, Ont.

Authorized WANG National ISO

cycare
a step ahead...

Angina Protection with Benefits for a Lifetime

ONCE-DAILY CONTROL WITH HEART-SAVING BENEFITS

By reducing heart rate and cardiac contractility, INDERAL LA helps protect the heart from the potentially serious and debilitating consequences of ischemia. A highly effective antianginal agent for around-the-clock control of symptoms, INDERAL LA also provides cardiovascular protection for a sense of security in the years ahead.

PROTECTION AND EXPERIENCE NO CALCIUM BLOCKER CAN MATCH

Unlike calcium blockers, INDERAL LA—either alone or with a nitrate—is recommended for early treatment of angina in the majority of patients. Equally important, INDERAL LA delivers the proven performance and safety profile of INDERAL tablets—confirmed by millions of patients during 16 years of clinical use.

START WITH 80 MG ONCE DAILY

Dosage may be increased to 160 mg once daily, as needed, to achieve optimal control. INDERAL LA should not be used in congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma. Please see next page for further details and brief summary of prescribing information.

ONCE-DAILY **INDERAL LA**

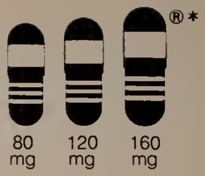
(PROPRANOLOL HCl) LONG ACTING CAPSULES

Ayerst

The appearance of INDERAL LA capsules is a registered trademark of Ayerst Laboratories

JUST ONCE EACH DAY
FOR SIMPLIFIED CORE
THERAPY IN ANGINA

ONCE-DAILY
INDERAL® LA
(PROPRANOLOL HCl) LONG ACTING CAPSULES



Just once each day...

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*
INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**
DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules.
CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain. Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use. Effects on plasma volume appear to be minor and somewhat variable INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction. Propranolol may increase oxygen requirements by increasing left ventricular fiber length end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential. The significance of the membrane action in the treatment of arrhythmias is uncertain.

The mechanism of the antimigraine effect of propranolol has not been established. Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction. Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

Propranolol is not significantly dialyzable.
INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary
CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock; 2) sinus bradycardia and greater than first degree block; 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL
WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g., dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes In these patients, it may be more difficult to adjust the dosage of insulin

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case this resulted after an initial dose of 5 mg propranolol
PRECAUTIONS. *General:* Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL is not indicated for the treatment of hypertensive emergencies

Beta adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure

Clinical Laboratory Tests Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose There are no adequate and well-controlled studies in pregnant women INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman.

Pediatric Use Safety and effectiveness in children have not been established
ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular bradycardia, congestive heart failure; intensification of AV block, hypotension; paresthesia of hands; thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type

Central Nervous System lightheadedness, mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances; hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics.

Gastrointestinal nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic: pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory bronchospasm.

Hematologic agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous: alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol
DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION—*Dosage must be individualized* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained. Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily
PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

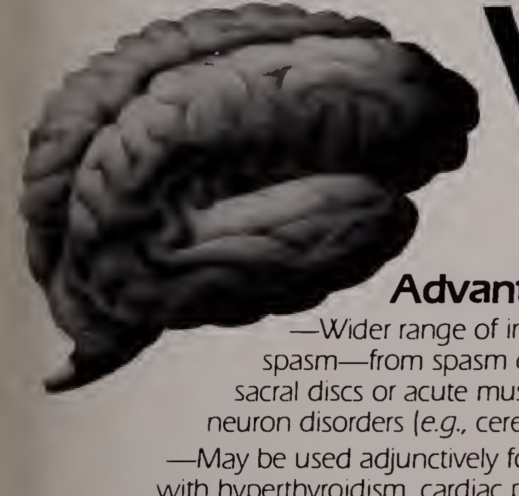
*The appearance of INDERAL LA capsules is a registered trademark of Ayerst Laboratories

8833/384

Ayerst.
AYERST LABORATORIES
New York, N.Y. 10017

THE SPASM/PAIN/SPASM CYCLE

In skeletal muscle spasm due to local pathology, responsive to the distinct actions of



Adjunctive
VALIUM® Ⓒ
diazepam/Roche
2-mg, 5-mg, 10-mg scored tablets

Advantages cyclobenzaprine cannot claim

—Wider range of indications as adjunctive therapy for skeletal muscle spasm—from spasm due to local pathology (*e.g.*, herniated lumbosacral discs or acute muscle strain) to spasm associated with upper motor neuron disorders (*e.g.*, cerebral palsy and paraplegia).

—May be used adjunctively for relieving skeletal muscle spasm in patients with hyperthyroidism, cardiac patients and patients being treated with anticholinergics or guanethidine-type antihypertensives.

—Can be administered to patients less than 15 years and more than 6 months of age.

—Scored tablets in three strengths provide greater dosage flexibility.

Since drowsiness, fatigue and ataxia sometimes occur, patients should be cautioned against driving or operating hazardous machinery and should also be advised against simultaneous ingestion of alcohol.

References: 1. Rankin EA: *Contin Educ 3*(1):46-50, Jan 1975. **2.** When muscle spasm hobbles your patient. *Patient Care 8*(11):20-37, Jun 1, 1974.

Valium® (diazepam/Roche) Ⓒ
Before prescribing, please consult complete product information, a summary of which follows:
Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety Anxiety or tension associated with the stress of everyday life usually does not require treatment with an anxiolytic Symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal, adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not as sole therapy)
The effectiveness of Valium in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies The physician should periodically reassess the usefulness of the drug for the individual patient
Contraindicated: Known hypersensitivity to the drug Children under 6 months of age Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy
Warnings: Not of value in psychotic patients Caution against hazardous occupations requiring complete mental alertness When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication, abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures Advise against simultaneous ingestion of alcohol and other CNS depressants Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months After extended therapy, gradually taper dosage Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence

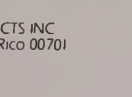
***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed, drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action Usual precautions indicated in patients severely depressed or with latent depression, or with suicidal tendencies Observe usual precautions in impaired renal or hepatic function Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation
The clearance of Valium and certain other benzodiazepines can be delayed in association with Tagamet (cimetidine) administration The clinical significance of this is unclear
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported, should these occur, discontinue drug Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy
Dosage: Individualize for maximum beneficial effect *Adults* Anxiety disorders, symptoms of anxiety, 2 to 10 mg b.i.d to q.i.d., alcoholism, 10 mg t.i.d or q.i.d in first 24 hours, then 5 mg t.i.d or q.i.d. as needed, adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d or q.i.d., adjunctively in convulsive disorders, 2 to 10 mg b.i.d to q.i.d *Geriatric or debilitated patients.* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated (See Precautions.) *Children.* 1 to 2½ mg t.i.d or q.i.d initially, increasing as needed and tolerated (not for use under 6 months).
How Supplied: For oral administration, round, scored tablets with a cut out "V" design—2 mg, white, 5 mg, yellow, 10 mg, blue— bottles of 100 and 500; Prescription Paks of 50, available in trays of 10. Tel-E-Dose® packages of 100, available in boxes of 4 reverse-numbered cards of 25, and in boxes containing 10 strips of 10

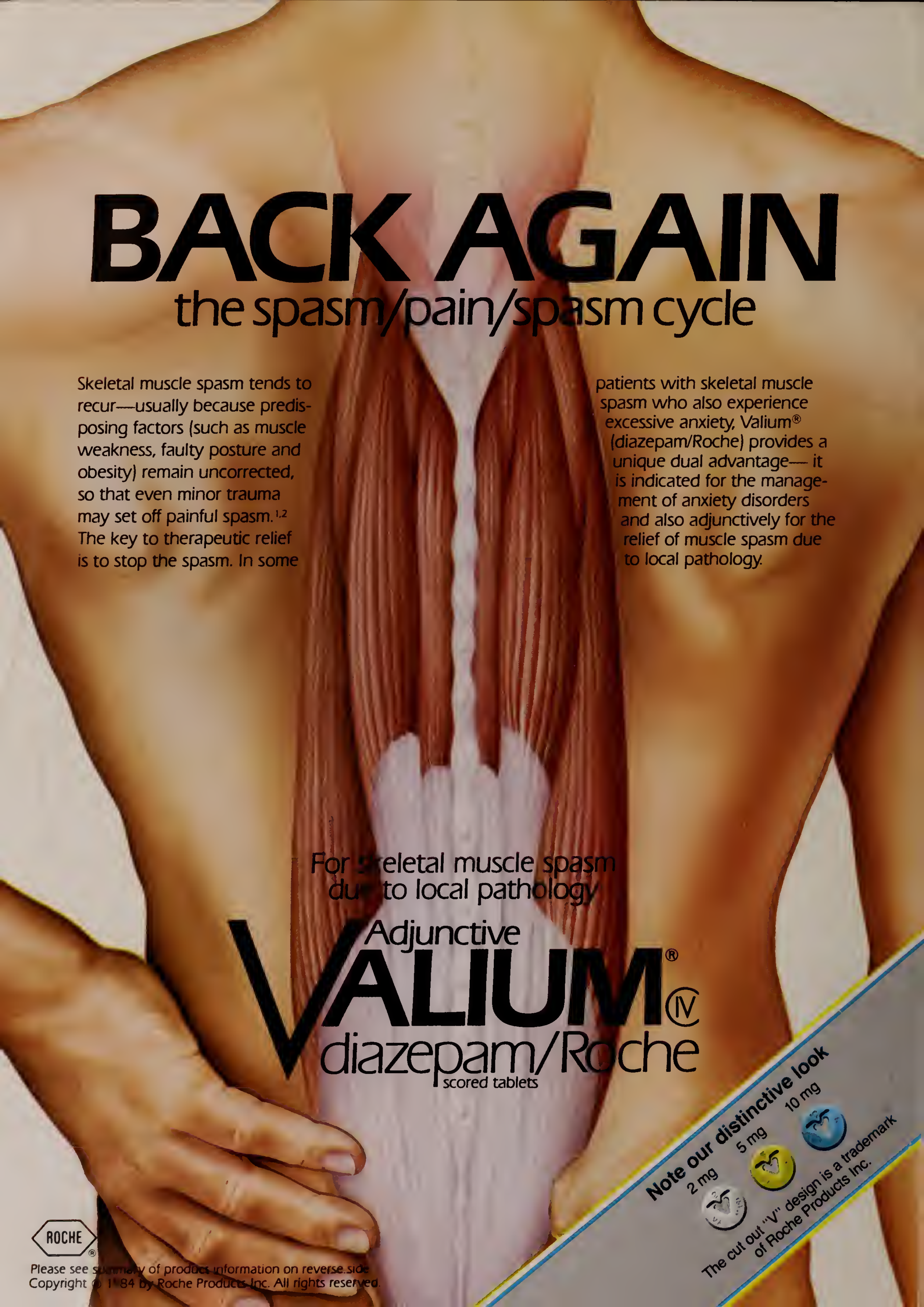
Imprint on tablets
2 mg—2 VALIUM® (front)
ROCHE (scored side)

5 mg—5 VALIUM® (front)
ROCHE (scored side)

10 mg—10 VALIUM® (front)
ROCHE (scored side)



ROCHE®
ROCHE PRODUCTS INC
Manati, Puerto Rico 00701



BACK AGAIN

the spasm/pain/spasm cycle

Skeletal muscle spasm tends to recur—usually because predisposing factors (such as muscle weakness, faulty posture and obesity) remain uncorrected, so that even minor trauma may set off painful spasm.[1,2] The key to therapeutic relief is to stop the spasm. In some patients with skeletal muscle spasm who also experience excessive anxiety, Valium® (diazepam/Roche) provides a unique dual advantage— it is indicated for the management of anxiety disorders and also adjunctively for the relief of muscle spasm due to local pathology.

For skeletal muscle spasm due to local pathology

Adjunctive

VALIUM® Ⓘⓥ

diazepam/Roche

scored tablets

Note our distinctive look
2 mg
5 mg
10 mg
The cut out "V" design is a trademark of Roche Products Inc.

ROCHE®

Please see summary of product information on reverse side

Copyright © 1984 by Roche Products Inc. All rights reserved.

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

JUNE 1985 • Volume 85, Number 6

Contents

COMMENTARIES

Humoral hypercalcemia of malignancy: The role of parathyroid hormone 235
CHARLES F. SHARP, JR, MD

Parathyroid adenoma in children 237
HENRY MANNIX, JR, MD

Clinical and pathologic features of endolymphatic stromal myosis 237
J. R. NEWLAND, MD

Hemodynamic monitoring of the patient in shock 238
RICHARD A. STEIN, MD, PAUL HANSARD, MD

Sarcoidosis revisited 240
G. MICHAEL DUFFELL, MD

RESEARCH PAPERS

The relationship of thrombocytopenia to the onset of persistent pulmonary hypertension of the newborn in the meconium aspiration syndrome 245
MICHAEL J. HORGAN, MD, NOEL J. M. CARRASCO, MD, HERMAN RISEMBERG, MD

Humoral abnormalities in three patients with idiopathic chronic polyneuropathy 248
P. K. COYLE, MD, STEVEN E. SCHUTZER, MD, A. B. STERMAN, MD, NANCY PERESS, MD, FREDERICK MILLER, MD

ESSAYS IN PEDIATRICS

Management of diabetes mellitus in children 251
IRENE N. SILLS, MD

HISTORY

Medical cataphoresis: Electrical experimentation and 19th century therapeutics 257
JOHN S. HALLER, JR

CASE REPORTS

Sarcoidosis of the breast 262
MARY ELLEN REITZ, MD, IRVING SEIDMAN, MD, DANIEL F. ROSES, MD

Intrathymic parathyroid adenoma in a child 263
RICHARD H. K. WU, MD, RAKESH SHREEDHAR, MD, NARAYAN PAI, MD, JULIO LEVY, MD, PAUL H. GERST, MD

Amniotic fluid embolism 265
MARGARET E. BATES, MD, UMA L. VERMA, MD, NERGESH A. TEJANI, MD, DANIEL J. DITROIA, MD

Amniotic fluid embolism 267
PERVEZ AHMED, MD, CHARLES TRAUBE, MD, OYA FRESKO, MD

Spontaneous remission in acute nonlymphocytic leukemia 269
AZRA RAZA, MD, LIVELEEN M. GILL, MD, IRENE RAKOWSKI, HARVEY D. PREISLER, MD

LETTERS TO THE EDITOR

Humoral hypercalcemia of malignancy (pseudohyperparathyroidism) 242
SEBASTIAN A. PETER, MD

Endolymphatic stromal myosis—a misconception 242
JOHN H. MALFETANO, MD, NICOLAS K. SALIBA, MD

The impact of third-generation cephalosporins on clinical outcome of gram-negative meningitis in a municipal hospital 243
CHARLES E. CHERUBIN, MD, SHLOMO MAAYAN, MD, ROBERT H. K. ENG, MD, SHARON M. SMITH, PHD

Ethical double standard 244
STANLEY L. LEE, MD

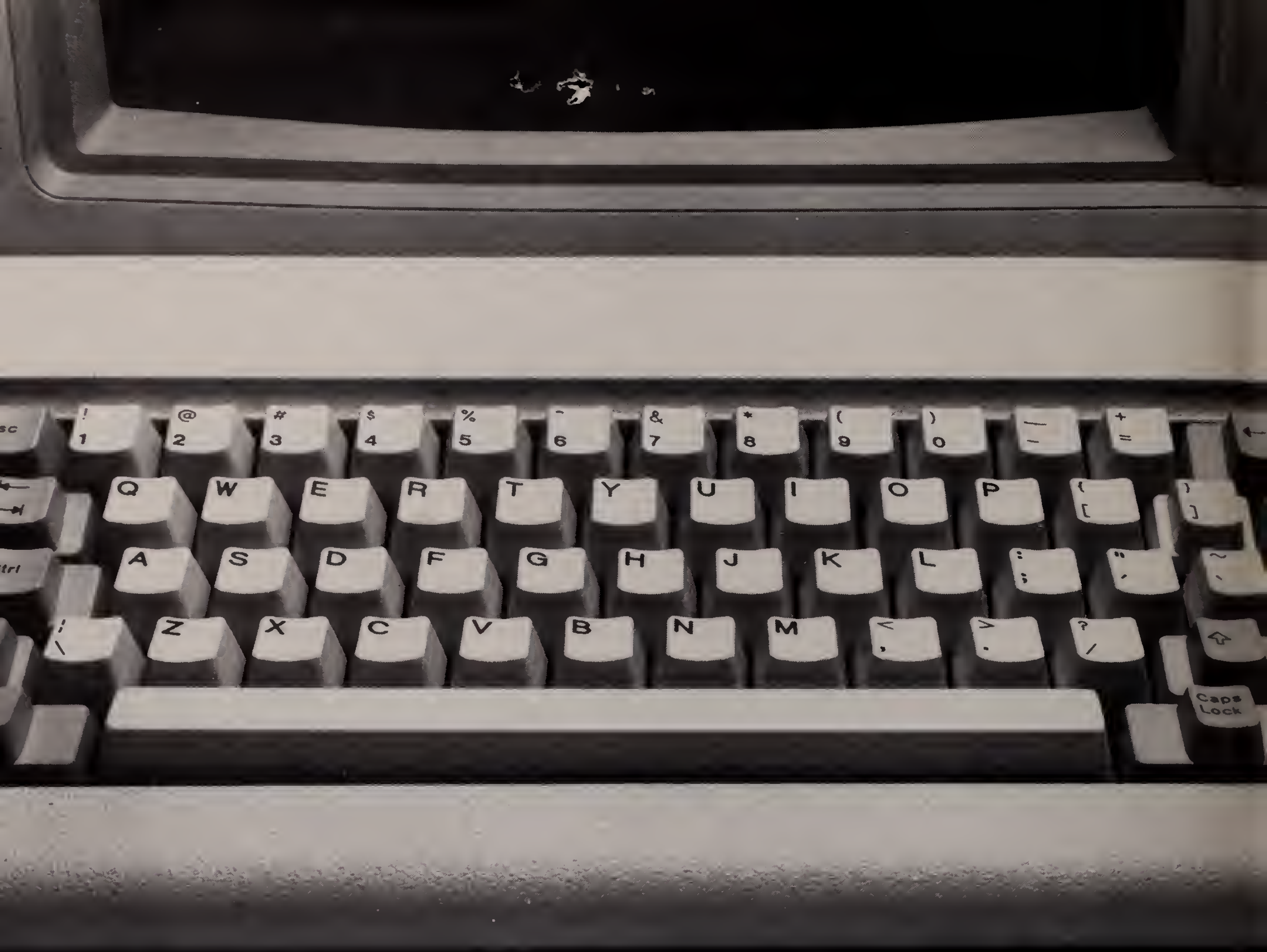
BOOK REVIEWS

OBITUARIES 274
GUIDELINES TO AUTHORS 277
MEDICAL MEETINGS AND LECTURES 4A

LIBRARY OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA
JUN 24 1985

Library of the Colleg
of Philadelphia De
19 S. 22nd St.
Phila, PA 19103

M. D. S.



CHEMICAL NOW GIVES SMALL BUSINESSES THE KEYS TO THE BANK.

Chemical Bank introduces a way to get into the bank without leaving your office.

It's our PRONTO Business Banker℠ for small businesses. And when you hook our software up to your personal computer, you'll find you're making an important business connection. In fact, it'll be like getting the keys to the bank. So you can enter anytime to do your business banking.

For instance, during the day you may be too busy with clients to get to the bank. It's times like these when you'll find the PRONTO Business Banker most convenient. You'll be able to transfer funds and check your balances right at your desk. Or if you're running a little short of cash when paying your bills, you can draw from your business' revolving credit line, just by using a few keys.

All your transactions will be updated every morning. So you'll know your financial situation at the start of every workday. And you can get a computer printout just by using a few more keys.

What's more, over the years Chemical Bank has specialized in helping small businesses grow. And to make sure you keep growing, we've put that same expertise into our PRONTO Business Banker.

For a personal demonstration or more information on Chemical Bank's PRONTO Business Banker, call us toll-free at 1-800-782-1100. Or visit one of our 260 branches.

The PRONTO Business Banker for small businesses. Now Chemical gives you more than just great service. We give you the keys to the bank.

SPECIAL BUSINESS OFFER

If you act now, you'll get the option of purchasing a 1200-baud General DataComm modem at the low price of $295. This top quality modem regularly retails for $600. Just ask us for details.

CHEMICALBANK

©1985 Chemical Bank

Member FDIC

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
ELIZA H. CALDWELL, MD
URSULA MATULONIS (Medical Student)
GERALD P. MURPHY, MD
RICHARD B. NOLAN, MD
CARL E. POCHEDLY, MD
JOHN T. PRIOR, MD

Editor	ALAN BLUM, MD
Deputy Editor and Book Review Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Managing Editor	VICKI GLASER
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Administrative Assistant	IMELDA MORRISSEY
Editorial Assistant	JEANNE CONNERY
Librarian	ELLA ABNEY
Library Assistant	ELEANOR BURNS
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1985

RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MARK C. HOMONOFF, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. Synergistic Media Sales, 50 East 42nd St., Suite 504, New York, N.Y. 10017. Telephone: (212) 682-2200.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

ALLISON B. LANDOLT, MD, Westchester	*President*
BERNARD J. PISANI, MD, New York	*Past-President*
DANIEL F. O'KEEFFE, MD, Warren	*President-Elect*
JOHN A. FINKBEINER, MD, New York	*Vice-President*
JOHN H. CARTER, MD, Albany	*Secretary*
GEORGE LIM, MD, Oneida	*Assistant Secretary*
CHARLES D. SHERMAN, JR, MD, Monroe	*Treasurer*
MORTON KURTZ, MD, Queens	*Assistant Treasurer*
JOHN L. CLOWE, MD, Schenectady	*Speaker*
CHARLES N. ASWAD, MD, Broome	*Vice-Speaker*

Councilors

Term Expires 1985	JAMES H. COSGRIFF, JR, MD, Erie SAMUEL M. GELFAND, MD, Nassau RICHARD A. HUGHES, MD, Warren ANTONIO F. LaSORTE, MD, Broome
Term Expires 1986	ROBERT A. MAYERS, MD, Westchester THOMAS D. PEMRICK, MD, Rensselaer RALPH E. SCHLOSSMAN, MD, Queens STANLEY A. STECKLER, MD, Suffolk
Term Expires 1987	DAVID M. BENFORD, MD, New York DUANE M. CADY, MD, Onondaga JAMES M. FLANAGAN, MD, Wayne M. THEODORE TANENHAUS, MD, Brooklyn

Trustees

CARL GOLDMARK, JR, MD, New York, *Chairman*
G. REHMI DENTON, MD, Albany
RALPH M. SCHWARTZ, MD, New York
EDGAR P. BERRY, MD, New York
RICHARD D. EBERLE, MD, Onondaga
JOSEPH F. SHANAPHY, MD, Richmond
VICTOR J. TOFANY, MD, Monroe

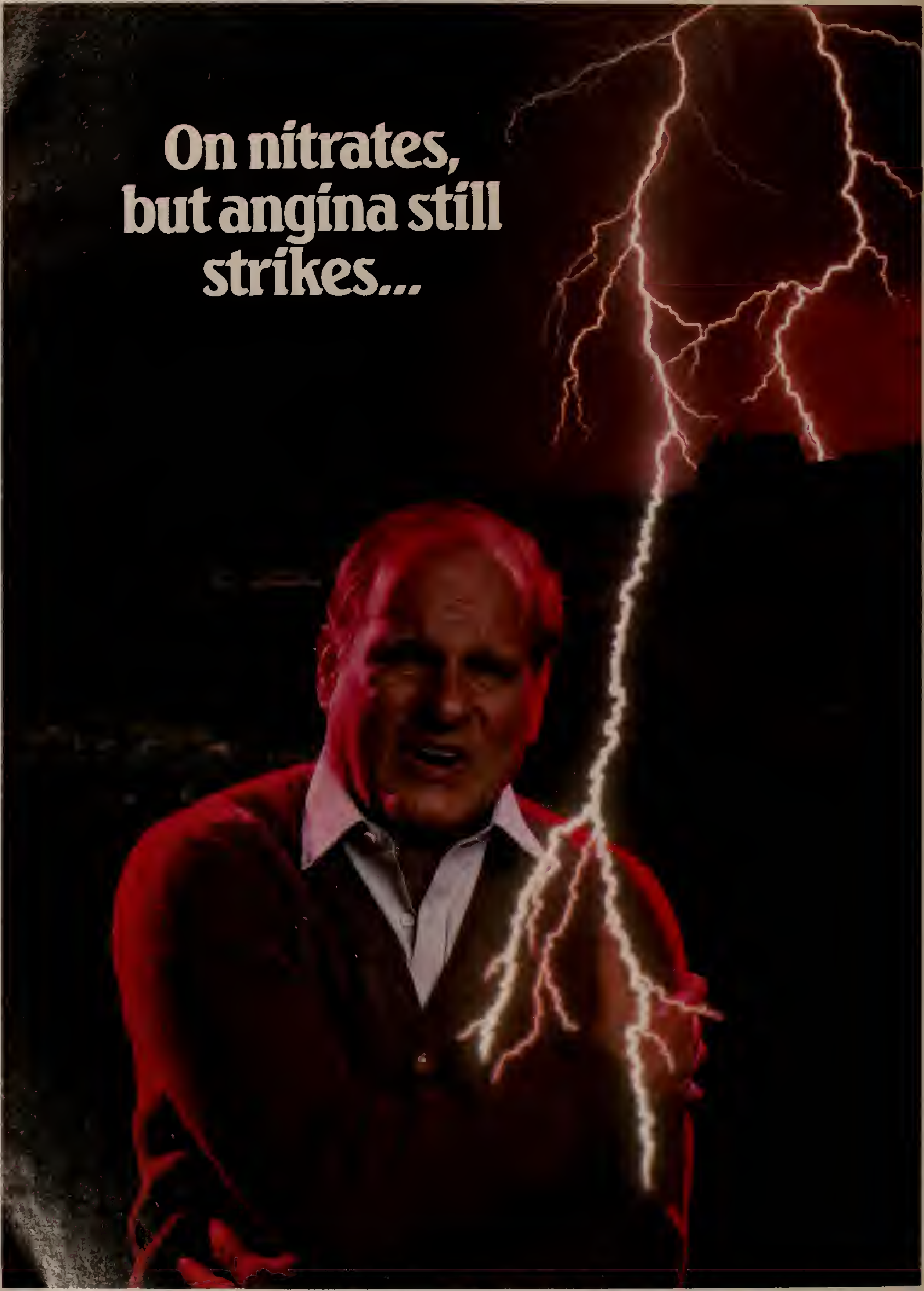
Executive Vice-President	GEORGE J. LAWRENCE, JR, MD
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Associate	IRMA A. ERICKSON
Director, Division of Scientific Publications	ALAN BLUM, MD
Director, Division of Management Services	GEORGE FORREST, JR
Assistant Director, Division of Governmental Affairs	JERRY S. HOFFMAN, ESQ
Director, Division of Communications	EDWARD HYNES
Director, Division of Insurance and Membership Benefits	BERNARD M. JACKSON
Director, Division of Membership	EUNICE SKELLY
Director, Division of Finance	PHILIP G. KEATING
Asst. Director, Division of Scientific Activities	ELIZABETH DAEPP
Director, Division of Advertising-Sales and Meeting Services	ROBERT MILLER
General Counsel	DONALD R. MOY, JD
Director, Division of Computer Services	IVAN H. NAJMAN
Acting Director, Division of Medical Services	MARIO MENGHINI
Director, Division of Long-Range Planning	DEBORAH R. SIEGEL
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. Editorial material published in the *Journal* is covered by copyright. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.


On nitrates,
but angina still
strikes...

After a nitrate, add ISOPTIN®

(verapamil HCl/Knoll)

To protect your patients, as well as their quality of life, add Isoptin instead of a beta blocker.

First, Isoptin not only reduces myocardial oxygen demand by reducing peripheral resistance, but also increases coronary perfusion by preventing coronary vasospasm and dilating coronary arteries — both normal and stenotic. These are antianginal actions that no beta blocker can provide.

Second, Isoptin spares patients the beta-blocker side effects that may compromise the quality of life.

With Isoptin, fatigue, bradycardia and mental depression are rare. Unlike beta blockers, Isoptin can safely be given to patients with asthma, COPD, diabetes or peripheral vascular disease. Serious adverse reactions with Isoptin are rare at recommended doses; the single most common side effect is constipation (6.3%).

Cardiovascular contraindications to the use of Isoptin are similar to those of beta blockers: severe left ventricular dysfunction, hypotension (systolic pressure <90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no artificial pacemaker is present) and second- or third-degree AV block.

So, the next time a nitrate is not enough, add Isoptin... for more comprehensive antianginal protection without side effects which may cramp an active life style.

ISOPTIN. Added antianginal protection without beta-blocker side effects.

Please see brief summary on following page.

ISOPTIN®
(verapamil HCl/Knoll)
80 mg and 120 mg scored, film-coated tablets

Contraindications: Severe left ventricular dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (except in patients with a functioning artificial ventricular pacemaker), 2nd- or 3rd-degree AV block. **Warnings:** ISOPTIN should be avoided in patients with severe left ventricular dysfunction (e.g., ejection fraction < 30% or moderate to severe symptoms of cardiac failure) and in patients with any degree of ventricular dysfunction if they are receiving a beta blocker. (See *Precautions.*) Patients with milder ventricular dysfunction should, if possible, be controlled with optimum doses of digitalis and/or diuretics before ISOPTIN is used. (Note interactions with digoxin under *Precautions.*) ISOPTIN may occasionally produce hypotension (usually asymptomatic, orthostatic, mild and controlled by decrease in ISOPTIN dose). Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported. Such elevations may disappear even with continued treatment; however, four cases of hepatocellular injury by verapamil have been proven by rechallenge. Periodic monitoring of liver function is prudent during verapamil therapy. Patients with atrial flutter or fibrillation and an accessory AV pathway (e.g. W-P-W or L-G-L syndromes) may develop increased antegrade conduction across the aberrant pathway bypassing the AV node, producing a very rapid ventricular response after receiving ISOPTIN (or digitalis). Treatment is usually D.C.-cardioversion, which has been used safely and effectively after ISOPTIN. Because of verapamil's effect on AV conduction and the SA node, 1° AV block and transient bradycardia may occur. High grade block, however, has been infrequently observed. Marked 1° or progressive 2° or 3° AV block requires a dosage reduction or, rarely, discontinuation and institution of appropriate therapy depending upon the clinical situation. Patients with hypertrophic cardiomyopathy (IHSS) received verapamil in doses up to 720 mg/day. It must be appreciated that this group of patients had a serious disease with a high mortality rate and that most were refractory or intolerant to propranolol. A variety of serious adverse effects were seen in this group of patients including sinus bradycardia, 2° AV block, sinus arrest, pulmonary edema and/or severe hypotension. Most adverse effects responded well to dose reduction and only rarely was verapamil discontinued. **Precautions:** ISOPTIN should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacologic effects. Studies in a small number of patients suggest that concomitant use of ISOPTIN and beta blockers may be beneficial in patients with chronic stable angina. Combined therapy can also have adverse effects on cardiac function. Therefore, until further studies are completed, ISOPTIN should be used alone, if possible. If combined therapy is used, close surveillance of vital signs and clinical status should be carried out. Combined therapy with ISOPTIN and propranolol should usually be avoided in patients with AV conduction abnormalities and/or depressed left ventricular function. Chronic ISOPTIN treatment increases serum digoxin levels by 50% to 70% during the first week of therapy, which can result in digitalis toxicity. The digoxin dose should be reduced when ISOPTIN is given, and the patients should be carefully monitored to avoid over- or under-digitalization. ISOPTIN may have an additive effect on lowering blood pressure in patients receiving oral antihypertensive agents. Disopyramide should not be given within 48 hours before or 24 hours after ISOPTIN administration. Until further data are obtained, combined ISOPTIN and quinidine therapy in patients with hypertrophic cardiomyopathy should probably be avoided, since significant hypotension may result. Clinical experience with the concomitant use of ISOPTIN and short- and long-acting nitrates suggest beneficial interaction without undesirable drug interactions. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. *Pregnancy Category C:* There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor and delivery only if clearly needed. It is not known whether verapamil is excreted in breast milk; therefore, nursing should be discontinued during ISOPTIN use. **Adverse Reactions:** Hypotension (2.9%), peripheral edema (1.7%), AV block: 3rd degree (0.8%), bradycardia: HR < 50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%), elevations of liver enzymes have been reported. (See *Warnings.*) The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain: ecchymosis, bruising, gynecomastia, psychotic symptoms, confusion, paresthesia, insomnia, somnolence, equilibrium disorder, blurred vision, syncope, muscle cramp, shakiness, claudication, hair loss, macules, spotty menstruation. **How Supplied:** ISOPTIN (verapamil HCl) is supplied in round, scored, film-coated tablets containing either 80 mg or 120 mg of verapamil hydrochloride and embossed with "ISOPTIN 80" or "ISOPTIN 120" on one side and with "KNOLL" on the reverse side. Revised August, 1984. 2385

KNOLL PHARMACEUTICAL COMPANY
30 NORTH JEFFERSON ROAD, WHIPPANY, NEW JERSEY 07981

2406

Medical Society of the State of New York
ANNUAL CME ASSEMBLY
MARCH 7-9, 1986
The New York Hilton, New York City
Combined With The Annual Meeting of
The House of Delegates
MARCH 6-9

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meetings and lectures unless the information is submitted at least two months prior to the event.*

JULY 1985

AROUND THE STATE

ORANGE

July 8–12. **2nd International Conference on Physical Activity, Aging & Sports.** West Point. Info: The Center for the Study of Aging, 706 Madison Ave, Albany, NY 12208.

TOMPKINS

July 22–26. **Update Your Medicine—Ithaca 1985.** Info: Cornell University Programs in Professional Education, Division of Summer Session, Extramural Study & Related Programs, B12 Ives Hall, Ithaca, NY 14853. Tel: (607) 256-7259.

AROUND THE NATION

CALIFORNIA

July 8–11. **Family Practice Board Review Course 1985.** San Francisco. Info: CME, University of California, School of Medicine, Rm. 569-U, San Francisco, CA 94143. Tel: (415) 666-4251.

AROUND THE WORLD

CANADA

July 11–13. **National Conference on Pediatric/Adult Allergy & Clinical Im-**

continued on p 8A

Classified Advertising

PHYSICIANS WANTED

MEDICAL DIRECTOR — EMERGENCY DEPARTMENT: Small community hospital located approximately 60 miles south of Rochester has an immediate position available for an emergency department Director. Responsibilities combine clinical and administrative functions. Post graduate training in a primary care area and emergency department experience required. Administrative experience desirable but not necessary. This career position offers an annual competitive income, director's stipend, paid professional liability insurance, paid health and life insurance benefits, plus more. For details contact Jean McGinty, Spectrum Emergency Care, Inc., P.O. Box 27352, St. Louis, MO 63141; 1-800-325-3982; 314-878-2280.

ANESTHESIOLOGIST: Upstate New York, Acute Medical/Surgical Hospital with active medical staff looking for Board Eligible or Certified Anesthesiologist. Excellent quality of life and setting. Interested candidates should send resume and economic expectations to Dept. 387 c/o NYSJM.

STAFF POSITIONS for Primary Care Physicians available at HMO Clinic Settings in Miami-Ft. Lauderdale Area. Attractive Salary, benefits and settings. Call or write Health-Med Systems, Inc., 19337 N.W. 2nd Ave., Miami, Fla. 33169. Phone (305) 651-8830.

PSYCHIATRISTS: Staff psychiatrists welcome. Excellent opportunity for building a practice. Call Nassau Hospital, Mineola, Long Island, New York, Department of Psychiatry at (516) 663-2691. Please ask for Nancy Yngstrom.

BOARD CERTIFIED INTERNIST: Preventive & Occupational Health—a leader in field offers varied opportunities (full-time, half-time or per diem) at our clinics and corporate medical units. Send C.V. to Executive Health Examiner, 777 Third Ave., N.Y., N.Y. 10017, Attn: Personnel Director N. [EOE M/F].

PHYSICIAN — EMERGENCY DEPARTMENT: Modern, well-equipped, mid-sized community hospital located one hour south of Buffalo has position available for an emergency department staff physician. Position requires some emergency department experience and two years post graduate training in a primary care specialty. This is a career opportunity for a physician dedicated to a career in emergency medicine. Competitive guaranteed income, paid professional liability insurance and other incentives provided. For details contact Jean McGinty, Spectrum Emergency Care, Inc., P.O. Box 27352, St. Louis, MO 63141; 1-800-325-3982; 314-878-2280.

PHYSICIAN WANTED: N.J. licensed medical doctor to supervise nutritional facility. Salary negotiable, plus paid malpractice insurance. Hours flexible. No knowledge of nutrition required! For interview call (201) 659-9519. Ask for Terry.

PHYSICIANS WANTED—CONT'D

ASSISTANT DIRECTOR, Department of Emergency Medicine in 950 bed voluntary hospital with 25,000 ER visits per year. Major affiliate of SUNY/Buffalo. BC/BE in IM, FM, EM or Surgery required. Teaching and research interests encouraged. Emergency service experience strongly preferred. Excellent opportunity in reorganized setting with developing trauma program. Position available July 1, 1985. Address inquiries to: Michael F. Noe, M.D., Vice President for Professional Affairs, Buffalo General Hospital, 100 High Street, Buffalo, N.Y. 14203 (716-845-2872). An Equal Opportunity Employer.

SOUTHERN CALIFORNIA: Prestigious HMO is seeking experienced specialists and general practitioners for our facilities in Los Angeles and Orange Counties. Located in close proximity to major teaching centers, we offer the opportunity for continued professional development and rewarding clinical practice. Excellent compensation and benefits including paid malpractice, life, disability, medical and dental coverage, paid vacations, sick leave, educational leave and retirement plan. Please send C.V. to: Director/Physician Recruitment, CIGNA Healthplans of California, 700 N. Brand Blvd., Suite 500-38, Glendale, CA 91203.

NEW YORK, NEW PALTZ—Seeking full-time experience or residency trained physicians; ACLS certified; for state of the art FEC in beautifull Mid-Hudson Valley, 80 miles from NYC. Start date is July 15, 1985. Package includes Health, Life, Disability and Malpractice Insurance. Excellent salary, attractive scheduling and potential profit sharing. Send CV/Inquiries to Gary W. Greer, M.D., RD 3 Box 44A, Cogan Station, PA., 17728: phone 1-717-494-0420.

ALL MEDICAL AND SURGICAL SPECIALTIES sought by quality oriented, nationwide, referral service for medical-legal consultation. Highly rewarding opportunity for qualified experts to provide objective reviews. Specific terms strictly between participating expert and requesting party. Send inquiries to ENET, 2020 W. State Street, Suite 164, Milwaukee, Wisconsin 53223.

FEMALE OB/GYN B.E./B.C. to join another female obstetrician in central New York State. Position available due to enormous growth of practice. Majority of population childbearing age. Guaranteed income. Ancillary help, office space contiguous to 150-bed hospital. Family oriented lifestyle. Outstanding schools and recreational area. Reply Dept. 389 c/o NYSJM.

CARDIOLOGIST—BC/BE—to organize and direct a model rural heart station-222 bed hospital. 10 internists on staff. Guaranteed income for first year. Contact Sister Mary Pierre, Administrator, Mercy Hospital of Watertown, Watertown, New York 13601. Reply with resume.

POSITIONS WANTED

INTERNIST WITH GERIATRIC BACKGROUND seeks association in private practice, preferably Queens or Nassau, but will consider other areas in or around New York City. Call (718) 658-8252 after 6 P.M. weekdays or weekends.

PRACTICES AVAILABLE

MID HUDSON VALLEY AREA: Internist after 35 years of practice wishes to retire. Office and 3 bedroom home recently aluminum sided with two car garage on corner lot close to St. Francis Hospital and Vassar Brothers Hospital. Completely equipped air conditioned office attached to house with separate entrance. Office easily adaptable to a two man practice. Reply Dept. 391 c/o NYSJM.

SELLING PRACTICES IS OUR ONLY BUSINESS—Want to maximize the return on your investment? Before you buy or sell, call for a consultation to assure the best financial and transfer terms. We guide you through the entire sales process from initial meeting to closing. Serving 100 mile radius of New York City. Countrywide Business Brokerage, Inc., 319 East 24th Street, Suite 23-G, New York, NY 10010 (212) 686-7902.

FAMILY PRACTICE AVAILABLE WITH OFFICE BUILDING: Fully equipped. Located Ballston Spa, N.Y. near Albany, N.Y. Doctor retiring after 35 years. Information from Norman Fox, 404 Broadway, Saratoga Springs, N.Y. 12866. (518) 587-7778.

FOR SALE: PRACTICE/HOME/OFFICE. 32 years busy FP/IM. Fully equipped x-ray, lab, EKG, darkroom, Pt. Equipt. 7 room office, 1½ acres, 8 room house, inground pool. Prime Bergen City location, available immediately. Call evenings (201) 825-0535.

PRACTICES WANTED

YOUNG INTERNIST, wishes to purchase Nassau County office based or nursing home based private practice. Available immediately. Strictly confidential. Reply Dept. 365, c/o NYSJM.

OPHTHALMOLOGIST, BOARD CERTIFIED desires to purchase practice in New York City or suburban area. Confidence maintained. Please contact Dept. 168, c/o NYSJM.

EQUIPMENT

WANTED: a good fluoroscopic screen in place of my old one. Prof-Xray screen needed 12 in. x 12 in., Direction 11824A, Code 10-3050. Urgent! Call (607) 756-9941, collect.

FOR PHYSICIANS AND SURGEONS WITH A PART-TIME EXPOSURE ONLY

Now offering Medical Malpractice Insurance on an ***occurrence basis*** at drastically reduced rates thru Frontier Insurance Co., Monticello, NY.

With the tail on claims made policies, you might lose your shirt.

Call or write for further information and application.

MEDICAL
MQ
QUADRANGLE

Medical Quadrangle, Inc.
P. O. Box 456
318 Bedford Road
Bedford Hills, New York 10507
914 666-0170
212 829-3654
"Specializing in Medical Liability Insurance"

MISCELLANEOUS

PROFESSIONAL MISCONDUCT ATTORNEYS. WILLIAM L. WOOD, JR., formerly Executive Director of the New York State Office of Professional Discipline and ANTHONY Z. SCHER, formerly Director of Prosecutions, announce the opening of their law office, WOOD & SCHER. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct proceedings, malpractice, licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. WOOD & SCHER, Attorneys at Law, One Chase Road, Scarsdale, New York 10583, Telephone 914-723-3500.

DOCTOR, YOU CAN'T BEAT the quality or the price! Holter Monitor Scanning Service. Physician owned, trained and supervised. No contracts to sign. We can arrange for lease/purchase of Holter equipment. New dual channel recorders with test cables $1,550. Now using UP Service for faster turn around time. DCG Interpretation (313) 879-8860.

ARCHITECTURE & PLANNING, Comprehensive services from analysis of alternative office locations through design and construction. Experienced in both new construction and renovation. Lewis & Gould (212) 807-6588.

MISCELLANEOUS—CONT'D

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professional incorporation—No insurance required—references upon request—Jack Wachstock, PC, 3000 Marcus Ave., Lake Success, N.Y. 11530 (516) 437-4848.

WHERE'S YOUR STAFF? WITH US! Office Managers, Medical Secretaries/Receptionists/RN's, LPN's, and more . . . Eden Health Services, 280 Madison Avenue, New York, New York, 10016, (212) 685-8600.

MISCELLANEOUS—CONT'D

ACCOUNTING AND TAX SERVICES—Professional accounting services including preparation of individual income tax and business tax returns, consultation on all tax and accounting matters. Call anytime. Marvin J. Siegel, CPA, (718) 275-0858. If no answer leave message. References available upon request.

AVOID MALPRACTICE SUITS—Free 30 Day Trial "Guidelines For Medical and Surgical Emergencies". Write professional type orders for laboratory and treatment. Masson Publishing Company—U.S.A., 133 East 58th Street, New York, NY 10022.

ARCHITECTURAL DESIGN AND CONSTRUCTION: Designers, builders of custom professional offices. Consulting services available. Whether you're opening a new office, relocating, remodeling or expanding an existing office in New York City area and adjacent New Jersey counties. Donald Young, Inc. Tel: (718) 948-5191.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate, with no points, fees or charges of any kind. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

EXCLUSIVE

NEWS FOR NEW YORK

TALWIN® Nx

Ⓒ IV Each tablet contains pentazocine HCl, USP, equivalent to 50 mg base and naloxone HCl, USP, equivalent to 0.5 mg base.

NOW APPROVED FOR MEDICAID

T-540 NDC 0024-1951-04
100 tablets
Ⓒ IV
Talwin® Nx
Each tablet contains pentazocine hydrochloride, USP, equivalent to 50 mg base and naloxone hydrochloride, USP, 0.5 mg.
Caution: Federal law prohibits dispensing without prescription.
Winthrop

Talwin® Nx

Ⓒ IV Each tablet contains pentazocine HCl, USP, equivalent to 50 mg base and naloxone HCl, USP, equivalent to 0.5 mg base.

Major Analgesic Gains Reimbursement Status

Albany—TALWIN Nx has recently been included on the New York State Medicaid list of reimbursable drugs.

Physicians can now make available to their Medicaid patients the many advantages of TALWIN Nx for moderate to severe pain conditions such as fractures, acute lumbosacral pain, severe sprains, joint pain, and acute trauma.

The newly listed TALWIN Nx is characterized by dependable efficacy and a proven record of safety.[1] One tablet provides pain relief equivalent to one grain of codeine, yet allows patients to function.*

TALWIN Nx offers a 15 to 30 minute onset of action, and is logical therapy for persistent pain because tolerance buildup is rare. The product is especially appropriate for many elderly patients since it has a low risk of constipation.†

*Since sedation, dizziness, and occasional euphoria have been noted, ambulatory patients should be warned not to operate machinery, drive cars, or unnecessarily expose themselves to hazards.

†See important product information concerning contraindications, warnings, adverse reactions, patient selection, and prescribing and precautionary recommendations on following page.

1. Data from the Boston Collaborative Drug Surveillance Program, July 1984.

TALWIN Nx now approved for the Medicaid Drug List by New York State.

Winthrop-Breon

© 1985 Winthrop-Breon Laboratories

Talwin® Nx Ⓒ IV

Each tablet contains pentazocine HCl, USP, equivalent to 50 mg base and naloxone HCl, USP, equivalent to 0.5 mg base.

Analgesic for Oral Use Only

Contraindications: Hypersensitivity to either pentazocine or naloxone.

TALWIN® Nx is intended for oral use only. Severe, potentially lethal, reactions may result from misuse of TALWIN® Nx by injection either alone or in combination with other substances. (See **Drug Abuse and Dependence** section.)

Warnings: *Drug Dependence*. Can cause physical and psychological dependence. (See **Drug Abuse and Dependence**.) *Head Injury and Increased Intracranial Pressure*. As with other potent analgesics, respiratory depressant effects of the drug may elevate cerebrospinal fluid pressure due to CO_2 retention; these effects may be markedly exaggerated in the presence of head injury, other intracranial lesions, or a preexisting increase in intracranial pressure. Can obscure the clinical course of patients with head injuries; in such patients, use with extreme caution and only if deemed essential. *Usage with Alcohol*. Due to potential for increased CNS depressant effects, alcohol should be used with caution. *Patients Receiving Narcotics*. Pentazocine is a mild narcotic antagonist. Withdrawal symptoms have occurred in patients previously given narcotics, including methadone. *Certain Respiratory Conditions*. Should be administered with caution in respiratory depression from any cause, severely limited respiratory reserve, severe bronchial asthma and other obstructive respiratory conditions, or cyanosis. *Acute CNS Manifestations*. Therapeutic doses have resulted in hallucinations (usually visual), disorientation, and confusion which have cleared spontaneously within hours. Such patients should be closely observed and vital signs checked. Reinstitution of the drug should be done cautiously since acute CNS manifestations may recur.

Precautions: *CNS Effect*. Use cautiously in patients prone to seizures; seizures have occurred though no cause and effect relationship has been established. *Impaired Renal or Hepatic Function*. Decreased metabolism of pentazocine in extensive liver disease may predispose to accentuation of side effects; it should be administered with caution in renal or hepatic impairment. In long-term use, precautions should be taken to avoid increases in dose by the patient. *Biliary Surgery*. Some evidence suggests that unlike other narcotics pentazocine causes little or no elevation in biliary tract pressures; the clinical significance of these findings is not yet known. *Information for Patients*. Since sedation, dizziness, and occasional euphoria have been noted, ambulatory patients should be warned not to operate machinery, drive cars, or unnecessarily expose themselves to hazards. May cause physical and psychological dependence taken alone and may have additive CNS depressant properties in combination with alcohol or other CNS depressants. *Myocardial Infarction*. Use with caution in patients with myocardial infarction who have nausea or vomiting. *Drug Interactions*. *Usage with Alcohol*: See **Warnings.** *Carcinogenesis, Mutagenesis, Impairment of Fertility*. No long-term studies in animals to test for carcinogenesis have been performed. *Pregnancy Category C*. Should be given to pregnant women only if clearly needed. *Labor and Delivery*. Use with caution in women delivering premature infants. Effect on mother and fetus, duration of labor or delivery, need for forceps delivery or other intervention or resuscitation of newborn, or later growth, development, and functional maturation of the child is unknown. *Nursing Mothers*. Caution should be exercised when administered to a nursing woman. *Pediatric Use*. Safety and effectiveness in children below the age of 12 years have not been established.

Adverse Reactions: *Cardiovascular:* Hypotension, tachycardia, syncope. *Respiratory:* Rarely, respiratory depression. *Acute CNS Manifestations:* In rare instances, hallucinations (usually visual), disorientation, and confusion which have cleared spontaneously within a period of hours. Such patients should be closely observed and vital signs checked. Acute CNS manifestations may recur if the drug is reinstituted. *Other CNS Effects:* Dizziness, lightheadedness, hallucinations, sedation, euphoria, headache, confusion, disorientation; infrequently weakness, disturbed dreams, insomnia, syncope, visual blurring and focusing difficulty, depression; rarely tremor, irritability, excitement, tinnitus. *Autonomic:* Sweating; infrequently flushing; and rarely chills. *Gastrointestinal:* Nausea, vomiting, constipation, diarrhea, anorexia, rarely abdominal distress. *Allergic:* Edema of the face; dermatitis, including pruritus; flushed skin, including plethora; infrequently rash, rarely urticaria. *Ophthalmic:* Visual blurring and focusing difficulty. *Hematologic:* Depression of white blood cells (especially granulocytes), which is usually reversible, moderate transient eosinophilia. *Other:* Headache, chills, insomnia, weakness, urinary retention, paresthesia.

Drug Abuse and Dependence: *Controlled Substance*. TALWIN Nx is a Schedule IV controlled substance.

Dependence and withdrawal symptoms have been reported with orally administered pentazocine. Patients with a history of drug dependence should be under close supervision. Possible abstinence syndromes in newborns after prolonged use of pentazocine during pregnancy have been reported. In prescribing for chronic use, the physician should take precautions to avoid increases in dose by the patient. Tolerance to the analgesic effect is rarely reported; there is no long-term experience with oral use of TALWIN Nx.

The amount of naloxone present (0.5 mg per tablet) has no action when taken orally and will not interfere with the pharmacologic action of pentazocine; however, this amount of naloxone given by injection has profound antagonistic action to narcotic analgesics. TALWIN Nx has a lower potential for parenteral misuse than the previous oral pentazocine formulation, but is still subject to patient misuse and abuse by the oral route.

Severe, even lethal, consequences may result from misuse of tablets by injection either alone or in combination with other substances, such as pulmonary emboli, vascular occlusion, ulceration and abscesses, and withdrawal symptoms in narcotic dependent individuals.

Overdosage: *Treatment:* Oxygen, intravenous fluids, vasopressors, and other supportive measures should be employed as indicated. Assisted or controlled ventilation should also be considered. For respiratory depression, parenteral naloxone is a specific and effective antagonist.

Please consult full product information before prescribing.

WIN 5-51296

Winthrop-Breon

Winthrop-Breon Laboratories
Division of Sterling Drug Inc.
New York, NY 10016

continued from p 4A

munology. Toronto. 20 Cat 1 credits. Info: Rayna Saville, Pediatric CME, SUNY/Buffalo Children's Hospital, 219 Bryant St, Buffalo, NY 14222. Tel: (716) 878-7630.

AUGUST 1985

AROUND THE STATE

MANHATTAN

Aug 26–30. **Radiation Physics & Biology.** 35 Cat 1 credits. Info: NYU Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Aug 30–Sept 1. **Diagnosis of Diseases of the Chest.** 22 Cat 1 credits. Grand Hyatt. Info: Dawne Ryals, PO Box 610203, DFW Airport, TX 75261-0203. Tel: (214) 659-9590.

NASSAU

Aug. 6. **Symposium on Coronary Artery Disease and Cardiac Rehabilitation.** 7 Cat 1 credits. Garden City Hotel. Info: M. Joseph Anto, MD, Division of Cardiology, Nassau County Medical Center, East Meadow, NY 11554. Tel: (516) 542-3581.

SARATOGA

Aug 6–10. **The Saratoga Conference on Internal Medicine.** 16 Cat 1 credits. Saratoga Springs. Info: Maureen A. Darragh, MSW, Office of CME, Albany Medical College, 75 New Scotland Ave, Albany, NY 12208. Tel: (518) 447-9611, ext 6823.

WARREN

Aug. 15–16. **Current Advances in Pediatrics.** Lake Luzerne. Info: St. Mary's Hospital, 1300 Massachusetts Ave, Troy, NY 12180. Tel: (518) 272-5000, ext 400.

continued on p 16A

BALANCED CALCIUM CHANNEL BLOCKADE!

Low incidence of side effects
CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

Reduces angina attack frequency*
42% to 46% decrease reported in multicenter study.[1]

Increases exercise tolerance*
In Bruce exercise test,[2] control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes ($P<.005$).

CARDIZEM®
(diltiazem HCl)

THE BALANCED CALCIUM CHANNEL BLOCKER

*Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

References:

1. Strauss WE, McIntyre KM, Parisi AF, et al: Safety and efficacy of diltiazem hydrochloride for the treatment of stable angina pectoris: Report of a cooperative clinical trial. Am J Cardiol 49:560-566, 1982.
2. Pool PE, Seagren SC, Bonanno JA, et al: The treatment of exercise-inducible chronic stable angina with diltiazem: Effect on treadmill exercise. Chest 78 (July suppl):234-238, 1980.

Please see full prescribing information on following page.

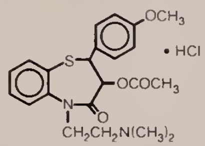
2/84

PROFESSIONAL USE INFORMATION

cardizem®
(diltiazem HCl)
30 mg and 60 mg tablets

DESCRIPTION

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one,3-(acetyloxy)-5-[2-(dimethylamino)ethyl]-2,3-dihydro-2-(4-methoxyphenyl)-, monohydrochloride,(+)-cis-. The chemical structure is:



Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform. It has a molecular weight of 450.98. Each tablet of CARDIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration.

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth muscle.

Mechanisms of Action. Although precise mechanisms of its antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways:

1. Angina Due to Coronary Artery Spasm: CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial and subendocardial. Spontaneous and ergonovine-induced coronary artery spasm are inhibited by CARDIZEM.
2. Exertional Angina: CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal and maximal exercise work loads.

In animal models, diltiazem interferes with the slow inward (depolarizing) current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Diltiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance.

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricular conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load. Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect; cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of diltiazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by diltiazem.

Intravenous diltiazem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Diltiazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, diltiazem significantly prolongs sinus cycle length (up to 50% in some cases).

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Diltiazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40%. CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 60 mg are given; a 120-mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem.

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm.** CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina).** CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS).

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia.
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing.
Cardiac Failure	Administer inotropic agents (isoproterenol, dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

The oral/LD_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort acute anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy**—CARDIZEM may be safely coadministered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

Cardizem 30-mg tablets are supplied in bottles of 100 (NDC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1771-49). Each green tablet is engraved with MARION on one side and 1771 engraved on the other. CARDIZEM 60-mg scored tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1772-49). Each yellow tablet is engraved with MARION on one side and 1772 on the other.

Issued 4/1/84

Another patient benefit product from

PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MISSOURI 64137

NEW YORK STATE
JOURNAL OF MEDICINE

June 1985 • Volume 85, Number 6

COMMENTARIES

Humoral hypercalcemia of malignancy

The role of parathyroid hormone

Hypercalcemia is a common complication in patients with cancer and is usually an ominous sign. The incidence of hypercalcemia is approximately 15% among all patients with cancer, but is greater among patients with carcinomas of the breast and lung.[1] Hypercalcemia is an important problem because the nausea, vomiting, confusion, and weakness that may result can lead to delay in instituting diagnostic or therapeutic regimens, or may be confused with the catabolic features of the cancer itself.

Seventy-five percent to 85% of patients with hypercalcemia associated with malignancy have skeletal metastases which can usually be demonstrated by roentgenograms, bone scans, or pathologic evaluation of the skeleton. A smaller percentage of patients may have hypercalcemia with no demonstrable skeletal lesions. Some of these patients have concomitant primary hyperparathyroidism, which is more frequent in patients who also have cancer.[1] In up to 20% of hypercalcemic patients with cancer, however, the tumor is producing and elaborating a humoral substance which is capable of stimulating osteoclastic resorption of bone and of causing hypercalcemia. This syndrome is known as humoral hypercalcemia of malignancy, or pseudohyperparathyroidism.[1] It occurs most frequently in association with epidermoid bronchogenic carcinoma, squamous cell carcinoma of the esophagus, carcinoma of the head and neck, hypernephroma, and ovarian cancer. Other solid tumors, including hepatocellular carcinoma, have been implicated.

Patients with humoral hypercalcemia of malignancy (HHM) are as vexing to the clinician as they are fascinating to the scientist. The report by Peter[2] in this issue of the *Journal* illustrates the difficulty of localizing the source of the hypercalcemic factor and raises the question whether tumors secrete parathyroid hormone (PTH). In fact, most investigators no longer believe native PTH to be the cause of hypercalcemia in HHM.[3] Nonetheless, the possibility that some form of PTH may play a role still remains. It has been suggested that a parathyroid hormone-like substance may be an important cause of this syndrome.[4]

The historical background is illuminating. In 1941, Fuller Albright[5] first proposed that ectopic production of PTH by non-parathyroid tumors might cause hypercalcemia. Subsequently, Skrabaneck et al[6] reported tumor extracts containing immunoreactive PTH. Some investigators[6,7] demonstrated that serum immunoreactive parathyroid hormone (SIPTH) was measurable or elevated in patients with cancer and hypercalcemia, rather than being suppressed, as would be expected in non-parathyroid hypercalcemia. Metabolic alkalosis was found to be a consistent feature, in contrast to the hyperchloremic metabolic acidosis that usually accompanies severe primary hyperparathyroidism.[8] More recently, findings of reduced plasma 1,25-dihydroxy-vitamin D concentrations and marked hypercalciuria have suggested suppressed SIPTH secretion in most patients with humoral hypercalcemia of malignancy.[9]

Cyclic AMP levels are a sensitive reflection of the effect of circulating active PTH and have been used by some investigators to define the syndrome of HHM.[9] Controversy remains over this theory, because elevated levels of cyclic AMP can also be found in normocalcemic patients with cancer.[10]

Histologic analysis of bone from patients with HHM reveals subtle but important differences from the findings in patients with severe primary hyperparathyroidism. Whereas in the former the bone shows osteoclastic resorption with small amounts of fibrous tissue formation,[11,12] osteoblastic activity, which is normally stimulated in primary hyperparathyroidism, seems to be absent in biopsies from patients with HHM.[12]

The most direct evidence of parathyroid hormone activity should be the level of parathyroid hormone in the circulation. It would be helpful if PTH assays discriminated hypercalcemia associated with cancer from primary hyperparathyroidism in all cases. In reality, there is often considerable overlap of SIPTH values, which can lead to confusion. This has been one impetus for the development of the other diagnostic tests cited above. Using a bovine aminoterminal anti-PTH antisera, Rude et al[10] obtained excellent discrimination between hyperparathyroidism and cancer: none of the patients with HHM had elevated PTH levels. However, antisera vary in specificity and sensitivity; moreover, assay technique and expression of data may influence results.[7] The most meaningful result is a level that is unequivocally elevated or suppressed.

Recent studies[13] have demonstrated stimulation of renal plasma membrane adenylate cyclase activity by tumor extracts which could be competitively inhibited by the bovine PTH fragment analogue (norleucine^{8,18}) tyrosine^{34}-bovine PTH (3–34) amide. Antisera against PTH failed to show PTH immunoreactivity, and adenylate cyclase activity could not be blocked by addition of antisera. The bioactivity was mainly present in fractions of molecular size much larger than either PTH or pro-PTH.

Another recent study[14] of tumor extracts showed PTH bioactivity in a renal glucose-6-phosphate-dehydrogenase system, also in a large molecular weight fraction, and also independent of the presence of PTH blocking antibodies.

Is ectopic PTH production an important cause of HHM? If it is, one first has to explain why PTH is undetectable or at low levels in most studies. One must then account for the hypochloremic alkalosis, relative hypercalciuria, suppressed levels of 1,25-dihydroxy-vitamin D, and the absence of osteoblastic activity on bone analysis, in contrast to the findings in typical primary hyperparathyroidism.

The identity of the putative humoral factor (or factors) responsible for producing HHM remains elusive. Mundy and others[3] have suggested that a transforming growth factor may be responsible. No doubt, future studies will provide the answers and will offer new insights into the mechanism of bone turnover.

Meanwhile, the clinician is confronted with an acutely ill patient with hypercalcemia of unknown cause. Frequently, the presence of a malignancy is obvious; occasionally it is not, as in the case reported by Peter.[2]

The first step must be to control the hypercalcemia by aggressive therapy. This facilitates patient compliance with subsequent diagnostic studies and generally makes the patient more comfortable and alert. Technetium diphosphonate bone scanning is the most sensitive test to exclude metastatic disease, and false negatives are rare when solid tumors are present. (Multiple myeloma can result in a negative scan.)

Some of the diagnostic criteria proposed by Lafferty[8] remain useful, but Boyd and Ladenson[15] have reported that serum calcium and albumin concentrations have the greatest discriminatory value in distinguishing hypercalcemia of malignancy from primary hyperparathyroidism.

Despite the problems described earlier, radioimmunoassay for PTH remains the single most important diagnostic test in a patient with hypercalcemia of unknown cause. If in a reliable clinical assay the level of SIPTH is elevated, then primary hyperparathyroidism must be suspected. It is helpful to keep in mind that primary hyperparathyroidism may coexist with a non-parathyroid malignancy; but if a reasonable search fails to uncover evidence of tumor, then surgical exploration of the neck may be indicated. In the case report presented by Peter[2] the serum calcium level did not respond to sub-total parathyroidectomy, and it was only later, at laparatomy, that a diagnosis of malignancy was established. Selective venous catheterization failed to demonstrate a mediastinal source of PTH—indirect evidence that the source of PTH was a tumor. Had an SIPTH level been obtained from the hepatic vein and contrasted with a peripheral level, there would have been irrefutable evidence for PTH secretion by the tumor. As autopsy was refused, the true diagnosis is not known.

The controversy regarding the role of PTH in HHM is far from over. There is good evidence to suggest that a parathyroid hormone-like peptide or even PTH itself can be secreted by a variety of tumors. The argument over the exact nature of the peptide and the frequency of occurrence continues.

CHARLES F. SHARP, Jr, MD
Center For Metabolic Bone Disease and Endocrinology
Los Angeles, CA 90007

1. Singer FR, Sharp CF Jr, Rude RK: Pathogenesis of hypercalcemia in malignancy. *Miner Electrolyte Metab* 1979; 2:161–178.
2. Peter S: Humoral hypercalcemia of malignancy (pseudohyperparathyroidism). *NY State J Med* 1985; 85:242.
3. Mundy GR, Ibbutson KJ, D'Souza SM, Simpson EL, Jacobs JW, Martin TJ: The hypercalcemia of cancer: clinical implications and pathogenetic mechanisms: *N Engl J Med* 1984; 310:1718–1727.
4. Arnaud CD, in Wyngaarden JB, Smith LH (eds): *Cecil Textbook of Medicine*. Philadelphia, WB Saunders Co, 1985, pp 1431–1442.
5. Case Records of the Massachusetts General Hospital. *N Engl J Med* 1931; 225:789–791.
6. Skrabaneck P, McPartlin J, Powell D: Tumor hypercalcemia and "ectopic hyperparathyroidism." *Medicine* 1980; 59:262–282.
7. Habener JF, Segre GV: Parathyroid hormone radioimmunoassay. *Ann Intern Med* 1979; 91:782–785.
8. Lafferty FW: Pseudohyperparathyroidism. *Medicine* 1966; 45:247–260.
9. Stewart AF, Horst R, Deftos LJ, Cadman EC, Lang R, Broadus AE: Biochemical evaluation of patients with cancer-associated hypercalcemia: evidence for humoral and nonhumoral groups. *N Engl J Med* 1980; 303:1377–1383.
10. Rude RK, Sharp CF Jr, Fredericks RS, et al: Urinary and nephrogenous adenosine 3′5′-monophosphate in the hypercalcemia of malignancy. *J Clin Endocrinol Metab* 1981; 52:765–771.
11. Sharp CF Jr, Rude RK, Terry R, Singer FR: Abnormal bone and parathyroid histology in carcinoma patients with pseudohyperparathyroidism. *Cancer* 1982; 49:1449–1455.
12. Stewart AF, Vignery A, Silverglate A, et al: Quantitative bone histomorphometry in humoral hypercalcemia of malignancy: uncoupling of bone cell activity. *J Clin Endocrinol Metab* 1982; 55:219–227.
13. Strewler GJ, Williams RD, Nissenson RA: Human renal carcinoma cells produce hypercalcemia in the nude mouse and a novel protein recognized by parathyroid hormone receptors. *J Clin Invest* 1983; 71:769–774.
14. Stewart AF, Insogna KL, Gottzman D, Broadus AE: Identification of adenylate cyclase-stimulating activity and cytochemical glucose-6-phosphate dehydrogenase-stimulating activity in extracts of tumors from patients with humoral hypercalcemia of malignancy. *Proc Natl Acad Sci USA* 1983; 80:1454–1458.
15. Boyd JC, Ladenson JH: Value of laboratory tests in the differential diagnosis of hypercalcemia. *Am J Med* 1984; 77:863–872.

Parathyroid adenoma in children

As pointed out by Wu et al,[1] in a case report in this issue of the *Journal* a variety of syndromes are associated with hypercalcemia in children and infants. The common finding of multiple hyperplasia in such cases presents a surgical challenge of considerable magnitude. As reported in 1975, the overall ratio of adenoma to hyperplasia in the literature was 5 to 1.[2]

A report by Girard et al[3] in 1982 reviewed the world literature to that time (86 cases) and added a case of a 15-year-old boy who had symptoms of anemia and growth retardation. Studies revealed primary hyperparathyroidism. An adenoma was removed, with relief of symptoms. The authors divided the patients into those with familial disease and those with no known genetic disorders. They emphasized that neonatal hyperparathyroidism was a more severe disease than that seen in older children. In patients with familial disease, the vast majority had parathyroid hyperplasia, while adenoma was a usual finding in the other group. Girard's review emphasized that aggressive surgery with subtotal parathyroidectomy was the essential treatment of neonatal hyperparathyroidism.

Allo et al[4] reviewed 53 patients under the age of 30 who were treated for primary hyperparathyroidism between 1971 and 1980. The patients had renal colic, renal calculi, malaise, and hypertension; symptoms related to bone disease were uncommon. In 26 patients symptoms developed before the age of 18, and 18 patients underwent surgery before they reached 18 years of age. In the older group, 27 patients had adenomas and one had hyperplasia, while in the children under 18 there were eight patients with adenomas and nine with hyperplasia. There was one child with neonatal parathyroid hyperplasia. The results of treatment were excellent, although one child had persistent elevated serum calcium levels and one child had a persistent decrease in serum calcium.

The symptoms of hyperparathyroidism in children are not always easy to interpret. Girard pointed out that during the neonatal period lethargy, poor eating habits, hypertension, dehydration, constipation or diarrhea, and respiratory problems were seen and could be present as early as one day after birth. This diagnosis was often not considered, especially if there was no family history of this disorder. Serum calcium levels in these infants are usually quite high. In older children, some of the above symptoms are more prominent; signs of conditions such as kidney disorders, bone disease, and hypertension are also more commonly seen.

In my own series of three patients, one had renal calculi, the second had failure to thrive and a bone cyst, and the third was initially treated as suffering from anorexia nervosa. Calcium studies were positive in all three patients, and surgery was successful in all of them.

Localization of parathyroid disease before surgery is being studied. In addition to older tests such as barium swallow, newer techniques such as ultrasound and computerized axial tomography are being advocated. The last two techniques are still underdeveloped and accuracy seems to vary with the level of experience of the radiologist. Venous sampling is not indicated in children unless several operations are required. Thallium subtraction scans combined with ultrasound techniques may become the standard test for hyperparathyroidism in the future.

HENRY MANNIX, Jr, MD
140 Woodland St
Hartford, CT 06105

1. Wu R, Shreedhar R, Pai BN, Levy J, Gerst PH: Intrathymic parathyroid adenoma in a child. *NY State J Med* 1985; 85:263–265.
2. Mannix H: Primary hyperparathyroidism in children. *Am J Surg* 1975 129:528–531.
3. Girard RM, Belanger A, Hazel B: Primary hyperparathyroidism in children. *Can J Surg* 1982; 25:11–13, 32.
4. Allo M, Thompson NW, Harness JK, Nishiyama RH: Primary hyperparathyroidism in children, adolescents, and young adults. *World J Surg* 1982; 6:771–776.

Clinical and pathologic features of endolymphatic stromal myosis

Endolymphatic stromal myosis (ESM) is classified under the general category of uterine sarcomas, which comprise about 2% of all neoplasms of the uterus.[1] Kempson[2] has suggested that there are five groups in this classification: pure sarcoma, mixed sarcoma, malignant mixed müllerian tumors, unclassified sarcoma, and malignant lymphoma. ESM falls into the pure sarcoma group, which is further subdivided into the pure homologous and pure heterologous types (ESM is in the homologous category). The origin of ESM is, as the name suggests, most likely from the endometrial stromal cell. Other histogeneses that have been suggested include vascular origins or from adenomyosis.[3] Accordingly, ESM could result directly from the basal endometrium, by development from undifferentiated tissue, or by vascular spread.[4]

The median age of presentation in patients with ESM is in the middle 40s, with an age range from eight to 61 years.[2,5,6] The most frequent sign is abnormal vaginal bleeding followed by abdominal or pelvic pain.

On gross examination, the uterus is enlarged, usually in a symmetrical fashion.[3] The tumor may protrude above the surface of the uncut specimen with a nodular appearance, and it will often bulge above a cut surface and have a white or yellow to orange color.[4,6] There is a worm-like or cord-like appearance in the tumor, which is a growth pattern associated with ESM. The tumor may extend beyond the enlarged uterus.

On microscopic examination the tumor consists of cells that resemble the endometrial stromal cell. The cells are arranged in groups of various shapes: some are cord-like with rounded margins. A characteristic feature in the appearance of these groups of cells is their apparent projection into spaces that resemble lymphatic channels ("endolymphatic"). The nuclei of the cells are round to oval in shape with scant cytoplasm. These presumed endometrial stromal cells are similar to those seen in a mid to late proliferative phase endometrium.[4]

In this issue of the *Journal,* Malfetano and Saliba[7] emphasize the malignant nature of endolymphatic stromal myosis and suggest a more descriptive name—low-grade endometrial stromal sarcoma. This feature of ESM has been realized for many years.[4] Norris and Taylor[6] examined a series of endometrial stromal tumors, and they found that stromal nodules with pushing margins acted in a benign fashion and did not extend beyond the uterus. Those nodules with infiltrating margins could be divided into two groups on the basis of mitotic activity: if there were less than 10 mitoses per 10 high power field, the tumors acted in a benign fashion; if there were more than 10 mitoses per 10 high power field, the five-year survival for patients was 55%. The former group of tumors was designated endolymphatic stromal myosis, and the latter stromal sarcoma. Kempson and Bari,[2] as well as Fekete and Vellios,[3] in their reviews of uterine sarcomas, used Norris and Taylor's criteria[6] and essentially agreed with their conclusions.

In rare instances, stromal sarcoma has arisen from endolymphatic stromal myosis. Yoonessi and Hart[8] reported such a case. Smith and colleagues[9] reported on a patient who originally had endolymphatic stromal myosis with extensive pelvic involvement which dedifferentiated into a stromal sarcoma. In all sections of the tumor that were examined, mitotic activity was low and the picture was that of endolymphatic stromal myosis. It was necessary to leave some of the tumor in the patient. Seven years later, this patient again sought treatment for a large mass in the pelvis, which on biopsy was found to be a uterine stromal sarcoma.

Endolymphatic stromal myosis is best considered a low-grade sarcoma and is thus classified as a uterine sarcoma. The cell of origin is most likely the endometrial stromal cell. Abnormal vaginal bleeding, abdominal or pelvic pain, and uterine enlargement are common clinical signs. It is important for the pathologist to distinguish between ESM and stromal sarcoma because of the much poorer prognosis of patients with the latter. This is done by counting mitoses. Finally, ESM may rarely transform into a stromal sarcoma.

J. R. NEWLAND, MD
Director of Laboratories
Department of Pathology
and Laboratory Medicine
University of Nebraska Medical Center
Omaha, NE 68105

1. Boutselis JG, Ullery JC: Sarcoma of the uterus. *Obstet Gynec* 1962; 20:23-35.
2. Kempson RL, Bari W: Uterine sarcomas. *Hum Pathol* 1970; 1:331-349.
3. Feteke PS, Vellios F: The clinical and histologic spectrum of endometrial stromal lesions: A report of 41 cases. *Int J Gynecol Pathol* 1984; 3:198-212.
4. Hertig AT, Gore H: Tumors of the female sex organs, part 2 with supplement. *Atlas of Tumor Pathology,* Section IX—fascicle 33. Armed Forces Institute of Pathology, 1960.
5. Baggish MS, Woodruff JD: Uterine stromatosis. Clinicopathologic features and hormone dependency. *Obstet Gynecol* 1972; 40:487-498.
6. Norris HJ, Taylor HB: Mesenchymal tumors of the uterus. I. A clinical and pathological study of 53 endometrial stromal tumors. *Cancer* 1966; 19:755-766.
7. Malfetano JH, Saliba NK: Endolymphatic stromal myosis—a misconception. *NY State J Med* 1985; 85:242-243.
8. Yoonessi M, Hart WR: Endometrial stromal sarcomas. *Cancer* 1977; 40:898-906.
9. Smith ML, Faaborg LL, Newland JR: Dedifferentiation of endolymphatic stromal myosis to poorly differentiated uterine stromal sarcoma. *Gynecol Oncol* 1980; 9:108-113.

Hemodynamic monitoring of the patient in shock

When amniotic fluid is introduced into the mother's circulation, the resulting syndrome is associated with high mortality. The clinical presentation includes cardiovascular decompensation with hypotension, tachycardia, peripheral signs of a shock state, respiratory distress and hypoxia, and coagulopathy—most often disseminated intravascular coagulation.

Two case studies of survivors of amniotic fluid emboli are presented in this issue of the *Journal.*[1,2] The two cases differ in clinical presentation and, in part, in therapy. Corticosteroids were used in one instance and heparin in the other. The Swan-Ganz catheter was integral to the successful management of both cases. It enabled periodic measurement of intracardiac pressures and the consequent propitious administration of inotropic drugs, intravenous fluids, and blood. This technique and subsequent treatment appear to be largely responsible for the survival of both patients during the critical 48 hours following initial presentation.

When viewed together, these reports support the early use of right heart hemodynamic monitoring in a patient with respiratory and cardiovascular decompensation. As with any invasive technique, however, the decision to institute right heart hemodynamic monitoring should be based on the following: the availability of personnel trained in catheter insertion and maintenance, as well as data analysis; an understanding of the relative importance of the information in the therapeutic decision-making process; the inability to obtain the pertinent information by less invasive means; and a thorough knowledge of the risks and complications of the procedure and the clinical states that increase the risk of cardiac catheterization beyond acceptable levels.

The multi-lumen polyvinyl balloon-tipped flow-directed catheter was first described in 1970 by Swan et al.[3] In most hospitals, trained staff and the requisite pressure and cardiac output equipment are available in several locations. Upon being introduced by cut down or percutaneously into a peripheral vein (antecubital, subclavian, internal jugular, or femoral), the catheter is advanced into the right atrium, right ventricle, pulmonary artery, and the pulmonary "capillary" wedge (PCW) position. In this position forward flow and pulmonary artery pressure are blocked from the lumen of the catheter, and the pressure recorded is that of the pulmonary venous system, which, in most settings, is nearly equal to the left atrial pressure and the filling pressure of the left ventricle during diastole. Catheter advance and repetitive "wedging" are facilitated by the inflatable balloon at the catheter tip. The location of the catheter tip is discerned from the pressure wave forms seen on the monitor, thus making the use of fluoroscopic equipment optional. A thermistor near the tip of the catheter allows repeated cardiac output measurements (thermodilution technique).

There are many indications for the placement of a Swan-Ganz catheter, including hypotension (for example, shock following a myocardial infarction, with sepsis, or of unknown cause), severe acute refractory congestive heart failure, respiratory distress syndrome, and suspected cardiac tamponade; the catheter is also used to monitor blood volume output in the high risk patient. In a patient in shock—such as can result from amniotic fluid emboli—left ventricular function can be assessed from thermodilution cardiac output measurements, and measurement of the PCW pressure will help define the basis of reduced output. Reduced output may be related to decreased venous return to the heart (a hypovolemic state or peripheral circulatory collapse), in which case a low PCW pressure is usually noted, or to left ventricular failure, in which case the PCW pressure will be high. In addition to defining the physiologic basis of hypotension or reduced cardiac output, periodic measurements via the Swan-Ganz catheter will allow the careful administration of fluids to expand intravascular volume, or the replacement of blood without the risk of pulmonary edema which can result from excessive expansion. Intravenous drugs that have inotropic effects or that reduce the after-load and pre-load on the heart can also be monitored by this technique. The two patients with amniotic fluid emboli described by Ahmed et al[1] and Bates et al[2] demonstrate different cardiovascular findings and differing Swan-Ganz measurements. In one case[1] there was a marked reduction in venous return to the heart with low right heart and PCW pressures. The patient responded to volume expansion. In the other case,[2] right heart and PCW pressures were above normal, and the use of dopamine proved successful. In addition, periodic measurement of the PCW and pulmonary artery pressures allowed rapid replacement of blood loss without volume overload and pulmonary edema.

The risks of placing and maintaining a Swan-Ganz catheter have been better defined in the past several years as experience has accumulated. Significant, and in some cases fatal, complications include arrhythmias and vascular, intracardiac, or pulmonary lesions. Premature ventricular contractions are common, occurring in greater than 15% of procedures,[4] with rare instances of ventricular tachycardia and fibrillation.[5] Because of the proximity of the right bundle of His to the path of the catheter, right bundle branch block occasionally occurs.[6,7] Only rarely, in the presence of pre-existing left bundle branch block—with a possible resultant complete heart block—is this a life threatening complication.

Vascular complications include deep vein thrombosis,[8,9] which has been reported as an asymptomatic finding in two thirds of cases with indwelling Swan-Ganz catheters, inadvertent perforation of an adjacent artery, and perforation of the pulmonary artery.[10] Right heart lesions are commonly found in autopsy studies of patients who had Swan-Ganz catheters in place for greater than 48 hours[11,12]; the lesions include subendocardial and valvular hemorrhage, nonbacterial thrombotic endocarditis, and infective endocarditis. The actual incidence of important intracardiac lesions is unknown, but the frequency of valvular hemorrhage may be as high as 31%.[11] Pulmonary complications include infarction from embolic thrombi, prolonged placement of the catheter in the wedge position, or endothelial trauma from the catheter.[12] Rarely, septic pneumonia associated with bacterial endocarditis is noted.[5] Technical complications such as knotting of the catheter in the heart, or the systemic or pulmonary vascular system have been reported. Manipulation with guide wires is usually successful in such cases.[13,14]

Swan-Ganz catheters have significantly enhanced the ability to diagnose and treat the patient in shock by permitting the repetitive recording of pressures in the right heart, pulmonary artery, and pulmonary capillary wedge position, as well as measurement of cardiac output. The value of such data, however, is only as great as the physician's knowledge of the physiology of the cardiovascular and pulmonary systems. Furthermore, an optimal safety level during the procedure requires a skilled and experienced team that is attentive to limiting the duration of the indwelling catheter.

RICHARD A. STEIN, MD
PAUL HANSARD, MD
Department of Medicine
SUNY-Downstate Medical Center
450 Clarkson Ave
Brooklyn, NY 11203

1. Ahmed P, Traube C, Fresko O: Amniotic fluid embolism. *NY State J Med* 1985; 85:267-268.
2. Bates ME, Verma, UL, Tejani NA, DiTroia DJ: Amniotic fluid embolism. *NY State J Med* 1985; 85:265-267.
3. Swan HJ, Ganz W, Forrester J, Marcus H, Diamond G, Chonette D: Catheterization of the heart in man with the use of a flow-directed balloon tipped catheter. *N Engl J Med* 1970; 283:477-451.
4. Forrester JS, Diamond GA, Swan HJC: Bedside diagnosis of latent cardiac complications in acutely ill patients. *JAMA* 1972; 222:59-63.
5. Moser KM, Spragg RG: Use of the balloon-tipped pulmonary artery catheter in pulmonary disease. *Ann Intern Med* 1983; 98:53-58.
6. Thomson IR, Dalton BC, Lappas DG, Lowenstein E: Right bundle-branch block and complete heart block caused by Swan-Ganz catheter. *Anesthesiology.* 1979; 51:359-362.
7. Luck JC, Engel TR: Transient right bundle branch block with "Swan-Ganz" catheterization. *Am Heart J* 1976; 92:263-264.

8. Choastre J, Cornud F, Bouchama A, Viau F, Benaacerraf R, Gilbert C: Thrombosis as a complication of pulmonary-artery catheterization via the internal jugular vein: prospective evaluation by phlebography. *N Engl J Med* 1982; 306:278–280.

9. Deepak V, Obeid FN, Horst M: Deep-vein thrombosis mimicking acute appendicitis. A complication of Swan-Ganz catheterization. *JAMA* 1982; 248:3010–3011.

10. Deren MM, Barash PG, Hamond GL, Saieh T: Perforation of the pulmonary artery requiring pneumonectomy after the use of a flow-directed (Swan-Ganz) catheter. *Thorax* 1979; 34:550–553.

11. Ford SE, Manley PN: Indwelling cardiac catheters. An autopsy study of associated endocardial lesions. *Arch Pathol Lab Med* 1982; 106:314–317.

12. Lange HW, Galliani CA, Edwards JE: Local complications associated with indwelling Swan-Ganz catheters: autopsy study of 36 cases. *Am J Cardiol* 1983; 52:1108–1111.

13. Mond HG, Clark DW, Nesbitt SJ, Schlant RC: A technique for unknotting an intracardiac flow-directed balloon catheter. *Chest* 1975; 67:731–733.

14. Brandstetter RD, Alarakhia N, Coli L, Gitler B: Distal kinking of a pulmonary artery catheter as a cause of fatal hemoptysis. *NY State J Med* 1984; 84:521–522.

Sarcoidosis revisited

Sarcoidosis, a granulomatous disease that can affect many organ systems, was first written about in 1877 by Jonathan Hutcheson.[1] He described a 60-year-old woman who developed large violaceous patches on her face and arms; Hutcheson coined the term "Mortimer's malady" in honor of the patient, Mrs Mortimer. Two years later Ernest Besnier,[2] a French dermatologist, described a patient with a purple swelling of the nose associated with ulceration of the nasal mucosa, as well as swelling of the earlobes and fingers; he named the disease lupus pernio. Later that same year another dermatologist, Cesar Boeck,[3] described the microscopic features of the disease, which he called multiple benign sarcoid of the skin. By the early 1900s, a number of additional cases had been reported, and it became apparent that sarcoidosis was a disease that affected many organ systems, with extensive tissue involvement.

In this issue of the *Journal*, Reitz, Seidman, and Roses[4] describe a case of sarcoidosis with breast involvement. This well studied case vividly points out the need for physician awareness that sarcoidosis can involve virtually any tissue of the body and can often mimic other diseases. Although the patient's condition had been diagnosed earlier, the presence of a discrete breast mass with regional lymphadenopathy suggested carcinoma of the breast, as pointed out by the authors.

Sarcoidosis is now recognized as a relatively common ailment, with an uneven worldwide distribution. In the United States it is more common in the Southeast and is seen more frequently in blacks. It occurs most often in the third and fourth decades of life, although cases have been found in children as well as in the elderly.

Despite the fact that sarcoidosis can affect many organ systems, it has a predilection for the lymphatic system causing lymphadenopathy, particularly in the thorax. The lungs appear to be the major target organ for the disease, suggesting that the unknown stimulus that initiates the disease process first gains entry to the body by way of the respiratory system. Despite extensive research during the past hundred years, the etiology of sarcoidosis remains unknown. However, the clinical aspects of the disease have been well described, and in recent years, much of the pathophysiology has been clarified through the work of Crystal[5] and his group at the National Institutes of Health. Clinical and pathologic studies previously had suggested that sarcoidosis had an early inflammatory and granulomatous phase followed by a more chronic, fibrotic phase. The characterization of chest films suggests that sarcoidosis can be divided into three stages: stage I showing bilateral hilar and mediastinal adenopathy with clear lung fields; stage II showing hilar and mediastinal adenopathy associated with diffuse interstitial pulmonary infiltrates; and stage III showing no adenopathy but extensive diffuse interstitial infiltrates that clinically and on biopsy represent diffuse interstitial fibrosis. Clinical studies have shown that the majority of patients with stage I disease experience spontaneous resolution and that only a few progress to stages II and III. A number of clinical studies have also suggested that patients with active sarcoidosis have an altered immune response.

Crystal and colleagues[6,7] have suggested that the pathogenesis of interstitial lung disorders in general, and sarcoidosis in particular, can be divided into four phases. The first phase represents the initial injury to the pulmonary alveolar epithelial cells, resulting in acute alveolitis. The second phase represents a more chronic alveolitis with varying degrees of alveolar injury. In phase three there is a derangement of collagen with the formation of varying degrees of interstitial fibrosis. Phase four represents advanced lung damage with interstitial fibrosis and honeycombing changes. The authors suggest that the transition from one phase to the next and the degree of resulting damage depends on several factors, including the chronicity of exposure to the unknown agent that sets the process in motion, the effectiveness of the lung defense mechanisms in protecting against the initial damage, the extent of the injury itself, the integrity of the alveolar epithelial basement membrane, and unknown individual susceptibility factors. They have found that the early alveolitis of phase one can be characterized by bronchoalveolar lavage using a fiberoptic bronchoscope and can be quantitated using radioactive gallium lung scanning. With this technique Crystal and colleagues have shown that the early alveolitis of sarcoidosis is an immunologically mediated lymphocytic inflammatory response, and that the progression from early alveolitis to granuloma formation is mediated through the release of chemotactic substances from activated lymphocytes and monocytes. The mechanisms that determine whether the initial alveolitis and granuloma formation progress to fibrosis have not yet been clarified. A number of groups in this country and abroad are evaluting the use of dimercaprol and gallium scanning to further clarify the pathogenesis of the disease and to establish the usefulness of these tests in guiding therapy.

The drug of choice in the treatment of sarcoidosis is prednisone, given in sufficient dosages and for a sufficient period of time to suppress the alveolitis and to block the progression from granuloma formation to fibrosis. It is not clear, however, which patients are the best candidates for early therapy. Clear cut indications for therapy include hypercalcemia and hypercalciuria, granulomatous uveitis, and significant involvement of vital organs such as the heart and kidney. Indications for therapy in patients with predominantly pulmonary involvement are less clear. Few physicians would treat patients with stage I sarcoidosis, that is those patients with bilateral hilar and paratracheal adenopathy and clear lung fields on radiographic examination. Therapy is indicated in patients with stage II sarcoidosis who have either significant and progressive symptoms or evidence of progressive disease such as worsening pulmonary function. Few physicians would consider therapy in patients with stage III sarcoidosis unless there is evidence of significant diffuse inflammation and active granuloma formation. The current guidelines regarding therapy will quite likely change as a better understanding of the pathogenesis of the disease evolves.

G. MICHAEL DUFFELL, MD
Director
Division of Pulmonary Diseases
Emory University School of Medicine
Atlanta, GA 30322

1. Hutchinson J: *Illustrations of Clinical Surgery*. London, J and A Churchill, 1875, p. 42.
2. Besnier E: Lupus pernio de la face. *Ann Derm Syph* 1889; 10:333.
3. Boeck C: Multipelt Benign Lud-sarcoid. *Norse Mag Laegevidensk* 1899; 14:1321.
4. Reitz ME, Seidman I, Roses DF: Sarcoidosis of the breast. *NY State J Med* 1985; 85:262–263.
5. NIH Conference: Pulmonary sarcoidosis: a disease characterized and perpetuated by activated lung T-lymphocytes. *Ann Intern Med* 1981; 94:73–94.
6. Crystal RG, Gadek JE, Ferrans VJ, Fulmer JD, Line BR, Hunninghake GW: Interstitial lung disease: current concepts of pathogenesis, staging and therapy. *Am J Med* 1981; 70:542–568.
7. NIH Conference: Idiopathic pulmonary fibrosis. Clinical, histologic, radiographic, physiologic, scintigraphic, cytologic, and biochemical aspects. *Ann Intern Med* 1976; 85:769–788.
8. Wallaert BL, Ramon P, Fournier E, Tonnel AB, Voisin C: Bronchoalveolar lavage, serum angiotensin-converting enzyme, and gallium-67 scanning in extrathoracic sarcoidosis. *Chest* 1982; 82:553–555.
9. Daniele RP, Elias JA, Epstein PE, Rossman MD: Bronchoalveolar lavage: role in the pathogenesis, diagnosis, and management of interstitial lung disease. *Ann Intern Med* 1985; 102:93–108.

ACTION ON MEDICAL LIABILITY

Among the resolutions passed by the House of Delegates of the Medical Society of the State of New York at its annual meeting in New York City, June 6–9, 1985, were the following:

Resolution 85-42, Limitation of Professional Medical Liability
Introduced by Richard J. Dwane, MD, Hospital Medical Staff Section Delegate

Resolved, That the Medical Society of the State of New York continue urgently to seek enactment of legislation that establishes a cap on awards for pain and suffering at a reasonable level; and be it further

Resolved, That MSSNY seek the enactment of legislation which would establish a cap on total awards not to exceed available policy limits; and be it further

Resolved, That MSSNY continue to seek enactment of legislation which includes limitations on attorneys contingency fees at fair and reasonable levels.

LETTERS TO THE EDITOR

This section is designed to offer rapid publication (usually within 1–3 months) of new clinical, epidemiologic, and laboratory observations, as well as of comments on articles published in the Journal *or on any topic related to the role of the physician. Letters should be typed double-spaced and should include the professional affiliation, highest academic degree, address, and signature of each author. Preference is given to letters not exceeding 450 words. All letters are personally reviewed and acknowledged by the Editor. Address correspondence to Editor,* New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042.*

Humoral hypercalcemia of malignancy (pseudohyperparathyroidism)

TO THE EDITOR: Hypercalcemia associated with a malignant lesion in the absence of skeletal metastases or a parathyroid adenoma has been designated humoral hypercalcemia of malignancy (pseudohyperparathyroidism).[1] Two recent reports[2,3] of 22 patients with this syndrome showed no evidence of elevated serum levels of immunoreactive parathyroid hormone. This is a report of a 57-year-old woman with hypercalcemia associated with elevated parathyroid hormone levels secondary to a pancreatic carcinoma.

Case Report: A 57-year-old woman was admitted to Kingsbrook Jewish Medical Center with complaints of lower back pain for four weeks and epigastric pain of two weeks duration. She denied any weight loss. On physical examination, the patient was fully conscious. Her pulse rate was 76/min, and her blood pressure was 160/90 mm Hg; the heart sounds were normal. Her chest was clear to auscultation, and no breast masses were palpable. There was epigastric tenderness, but no abdominal masses were palpable. There was no significant adenopathy, the thyroid was normal, and a rectal examination was unremarkable. Pain was elicited on flexion of her lumbosacral spine.

On the third hospital day a calcium level of 13.8 mg % (normal, 8.5–10.5 mg %) measured by the SMA 11-C analyzer was noted. On the fifth hospital day the patient became confused and lethargic and was seen by me on consultation. The serum calcium value was now >15 mg %, and the serum phosphate was 2 mg %. She was treated with normal saline and intravenous furosemide therapy. Her condition improved, and calcium levels were maintained at about 11–12 mg %. Prior to initiation of hydration and furosemide therapy, her blood study results were as follows: hemogram, normal; sodium, 139 meq/L; potassium, 4.1 meq/L; chloride, 106 meq/L; CO_2 content, 24 meq/L; blood urea nitrogen, 10 mg %; creatinine, 0.9 mg %; alkaline phosphatase, 150 U/L (normal, 39–95 U/L); total protein, 6.5 g; albumin, 3.5 g. The patient was given prednisone, 40 mg per day orally for 10 days, with no significant change in calcium levels.

Protein electrophoresis was normal; a skeletal survey, bone scan, and chest film were negative for metastases. Osteoarthritic changes were noted at the lumbosacral joint. Parathyroid hormone levels, determined using antiserum to intact hormone (done by Upjohn Laboratories, Kalamazoo, MI), were elevated. The highest value was 541 pg/mL (normal, 163–347 pg/mL) at a calcium level of 12.9 mg %. During surgical exploration of the neck, 3½ parathyroid glands of normal size were removed; frozen sections were compatible with chief cell hyperplasia. Examination of the glands in permanent sections confirmed the diagnosis of primary chief cell hyperplasia. On the first day after surgery the serum calcium level was 11.1 mg%. The patient became lethargic and confused on the fourth day after surgery; the serum calcium level was 13.8 mg %. Normal saline and intravenous furosemide therapy were restarted. The parathyroid hormone level at this time was 437 pg/mL. She was transferred to State University Hospital on the eighth day after surgery. Selective venography was done. Samples of blood from the superior vena cava, innominate veins, and internal jugular veins were measured for immunoreactive parathyroid hormone using an antiserum to the N-terminal fraction. A significant gradient of hormone was not demonstrated, strongly suggesting the absence of a parathyroid adenoma in the mediastinum. Skeletal survey, bone scan, liver scan, abdominal sonogram, and computerized tomographic scan of the abdomen were all negative. At laparotomy, numerous small nodules were found on the liver. Biopsy of the nodules revealed an adenocarcinoma; the site of origin was most likely the pancreas. The patient remained hypercalcemic until her death. An autopsy was refused.

Discussion: Sharp et al,[3] in a report on nine patients with humoral hypercalcemia of malignancy (pseudohyperparathyroidism) were unable to detect significant levels of circulating immunoreactive parathyroid hormone. Similarly Stewart et al,[2] utilizing four region-specific antisera, did not demonstrate increased levels of parathyroid hormone in 13 patients with this syndrome. The high levels of parathyroid hormone in this patient were not due to a coincidental adenomatous fifth parathyroid gland, since no hormonal gradients were demonstrated by selective venography. The production of a parathyrotrophic factor by the tumor was highly unlikely since hypercalcemia persisted after removal of 3½ parathyroid glands. Primary chief cell hyperplasia[4] was noted in this patient, as in the nine cases reported by Sharp et al.[3] She was not cachectic at the time of neck exploration; therefore, her symptoms do not fit the hypothesis proposed for chief cell hyperplasia in this syndrome.[5]

The production of increased parathyroid hormone levels by malignant tumors must be considered as a potential cause of humoral hypercalcemia of malignancy.

SEBASTIAN A. PETER, MD
Medical Coordinator
Long-Term Care Medicine
Kingsbrook Jewish Medical Center
585 Schenectady Ave
Brooklyn, NY 11203.

1. Lafferty FW: Pseudohyperparathyroidism. *Medicine* 1966; 45:247–260.
2. Stewart AF, Horst R, Deftos LJ, Cadman EC, Lang R, Broadus AE: Biochemical evaluation of patients with cancer-associated hypercalcemia. *N Engl J Med* 1980; 303:1377–1383.
3. Sharp CF, Rude RK, Terry R, Singer FR: Abnormal bone and parathyroid histology in carcinoma patients with pseudohyperparathyroidism. *Cancer* 1982; 49:1449–1455.
4. Black WC, Haff RC: The surgical pathology of parathyroid chief cell hyperplasia. *Amer J Clin Path* 1970; 53:565–579.
5. Azzopardi JC, Whittaker RS: Bronchial carcinoma and hypercalcaemia. *J Clin Path* 1969; 22:718–724.

See also pp 235–236.

Endolymphatic stromal myosis—a misconception

TO THE EDITOR: Endolymphatic stromal myosis is a low-grade uterine sarcoma which has perplexed gynecologists and oncologists for years due to confusing terminology and the lesion's benign histologic appearance yet malignant clinical behavior. We describe the management of a patient with this disorder.

Case Report: A 66-year old woman sought treatment for her first episode of post-menopausal bleeding in October 1983. She had gone through menopause 16 years earlier and had not taken any estrogen supplements for eight years prior to this admission. She had had an ovarian cystectomy and appendectomy at age 26 and a Manchester procedure (for uterine prolapse) at age 47. The patient denied any weight loss, anorexia, and abdominal or pelvic pain.

She underwent a dilation and curettage procedure that revealed a uterine cavity measuring 7 cm and yielded a moderate amount of yellowish endometrial tissue. The initial diagnosis was that of a low-grade endometrial stromal sarcoma. Routine blood studies, an electrocardiogram, and a chest film were within normal limits. An intravenous pyelogram and a barium enema were unremarkable. The patient underwent an examination under anesthesia, and exploratory laparotomy, a total abdominal hysterectomy and bilateral salpingo-oophorectomy, and a para-aortic lymphadenectomy.

Pelvic and paracolic cytologic washings were obtained at surgery and contained no malignant cells. The examination of the pelvis and upper abdomen disclosed no abnormalities. A 10-g section of tumor was analyzed for estrogen and progesterone receptors. The uterus measured 8 × 6 × 4 cm and weighted 82 g. Within the endometrial cavity and occupying the fundus to the serosal surface was an intramural myoma that measured 6 × 4 × 4 cm. On histologic analysis, this endometrial tumor showed irregular nests and sheets of abnormal stromal cells invading the full depth of the myometrium and protruding into vascular and lymphatic spaces (Fig 1). Mitotic figures were rare, with less than one per 10 high powered field. The remainder of the endometrium showed mild adenomatous hyperplasia, and the cervix was unremarkable. The fallopian tubes, ovaries, and para-aortic lymph nodes contained no metastatic disease. The receptor assay analysis was positive for the presence of estrogen and progesterone receptors. After surgery the patient was treated with 5,000 rad of total pelvic radiotherapy, and megestrol acetate (40 mg three times a day) was administered. To date, the patient remains without evidence of the disease.

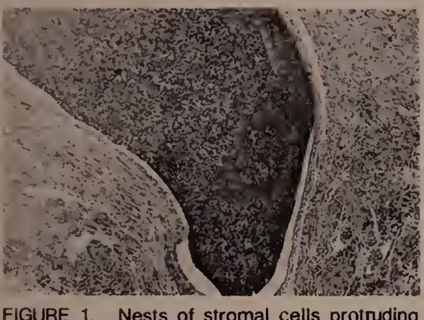


FIGURE 1. Nests of stromal cells protruding into a capillary-like space. (original magnification ×80)

Discussion: In 1908 Doran and Lockyer[1] described the first case of endolymphatic stromal myosis in the English literature and called it uterine stromatosis. In 1946, Henderson[2] suggested the name endolymphatic stromal myosis for this entity, based on the pathologic feature of growth within dilated venous and lymphatic channels. Telinde,[3] reviewing Henderson's paper, felt a more appropriate term would be stromatosis. With these original descriptions came a myriad of terms: proliferative stromatosis, stromal adenomyosis, malignant stromatosis, endometriosis interstitiale, stromatoid mural sarcoma, endometrial stromatosis, and, finally, low-grade endometrial stromal sarcoma. This last term would appear to be the most appropriate name, because of the lesion's benign appearance on histologic study and low mitotic rate. However, because of its extension beyond the uterus in nearly 40% of patients, a recurrence rate of almost 50% with both local and distant metastases, and reports of death, the lesion must be considered as having the potential for malignant behavior.[2,4-7] This patient was unusual in that the diagnosis of a lesion was made before exploratory surgery, as a result of a dilation and curettage procedure. In Gusberg's series, only 4% of lesions were diagnosed before surgery; most commonly, the patient underwent surgery for a pelvic mass or uterine leiomyoma.[6] Seventy percent to 80% of patients with this disorder are initially seen with Stage I disease, although extension beyond the uterus into contiguous structures is found in about 30% of patients. Recurrent disease occurs in nearly 50% of patients, usually five years after their initial surgery, however, recurrences as late as 25 years have been reported.[8]

Ideal treatment should consist of an exploratory laparotomy and surgical staging. Generally, this would consist of a total abdominal hysterectomy, a bilateral salpingo-oophorectomy, and resection of extra-uterine disease. Adjuvant therapy should consist of pelvic radiotherapy to prevent the local recurrence that appears to be so common, and patients should be placed on progestin therapy, since there is recent evidence to suggest that this lesion is a hormonally dependent tumor. Several reports of complete response to progestin therapy have been published.[9,10]

Only with a greater awareness of the potential malignant nature of endolymphatic stromal myosis can its origin and pathophysiology be better understood.

JOHN H. MALFETANO, MD
NICOLAS K. SALIBA, MD
Division of Gynecologic Oncology
Albany Medical College
M-809
Albany, NY 12208

1. Doran, AHG, Lockyer C: Two cases of uterine fibroids showing peritheliomatous changes; long immunity from recurrence after operation. *Proc R Soc Med* 1908; 2:25–39.
2. Henderson DN: Endolymphatic stromal myosis. *Am J Gynecol* 1946; 52:1000–1012.
3. Telinde RW: Discussion of Henderson: Endolymphatic stromal myosis. *Am J Obstet Gynecol* 1946; 52:1012.
4. Kempson RL, Bari W: Uterine sarcomas: classification, diagnosis and prognosis. *Human Pathol* 1970; 1:331–349.
5. Norris HJ, Taylor HB: Mesenchymal tumors of the uterus I. A clinical and pathologic study of 53 endometrial stromal tumors. *Cancer* 1966; 19:755–766.
6. Hart WR, Yoonessi M: Endometrial stromatosis of the uterus. *Obstet Gynecol* 1977; 49:393–403.
7. Piver MS, Rutledge RN, Copeland L, Webster K, Blumenson L, Suh O: Uterine endolymphatic stromal myosis: a collaborative study. *Obstet Gynecol* 1984; 64:173–178.
8. Kreiger PD, Gusberg SB: Endolymphatic stromal myosis—a grade I endometrial sarcoma. *Gynecologic Oncology* 1973; 1:299–313.
9. Pellillo D: Proliferative stromatosis of the uterus with pulmonary metastases. Remission following treatment with a longacting synthetic progestin: a case report. *Obstet Gynec* 1968; 31:33–39.
10. Gloor E, Schnyder P, Cikes M, et al: Endolymphatic stromal myosis: surgical and hormonal treatment of extensive abdominal recurrence 20 years after hysterectomy. *Cancer* 1982; 50:1888–1893.

See also pp 237–238.

The impact of third-generation cephalosporins on clinical outcome of gram-negative meningitis in a municipal hospital

TO THE EDITOR: Gram-negative bacillary meningitis is associated with only a fair to a poor prognosis, as shown by a review of bacteriologically confirmed cases of such meningitis in New York City during a seven-year period.[1] Of the 158 patients treated prior to the introduction of third-generation cephalosporin antibiotics, only 29% were rid of the infection. Most patients received chloramphenicol or parenteral aminoglycoside therapy which was supplemented, in rare instances, with erratic intrathecal aminoglycoside therapy.

The recent introduction of two new cephalosporin antibiotics (cefotaxime and moxalactam) has apparently changed the outlook on gram-negative meningitis, and cure rates of over 90% have been reported.[2-4] We have examined the records of one city hospital before and after the introduction of the third-generation cephalosporins in the treatment of this previously fatal disease.

From 1972 to 1982, patients seen on consultation by the Infectious Disease Service at Metropolitan Hospital were reviewed. Nine patients were seen prior to the introduction of third-generation cephalosporins (1972–1979). The infecting organisms included one isolate of *Pseudomonas aeruginosa,* two isolates of *Esherichia coli,* one of *Salmonella,* two of *Klebsiella pneumoniae,* one of *Enterobacter cloacae,* and two of *Serratia marcescens.* The three antibiotics used to treat these nine patients included ampicillin, gentamicin, and chloramphenicol. The only bacteriologic and clinical successes were obtained in two cases in which the infection was caused by *P aeruginosa* or *E coli.* The successful treatment of the patient with a *P aeruginosa* infection was unexpected. Parenteral carbenicillin and gentamicin given both intrathecally and parenterally proved adequate therapy. The other patient in which a successful outcome was observed had an infection with *E coli.* The organism was susceptible to ampicillin, and therapy with high doses of this beta-lactam and gentamicin was effective. The other seven cases of meningitis involved organisms susceptible to chloramphenicol, but chloramphenicol therapy proved ineffective.

The four patients with gram-negative bacillary meningitis who were seen after the advent of the third-generation cephalosporins (1980–1982) had similar underlying diseases as those patients of the preceding years who had meningitis. Three of the four patients initially received chloramphenicol with no clinical improvement and, in fact, the infecting organism was reisolated during the chloramphenicol therapy in each patient. Three of the patients had infections with *K pneumoniae,* and one patient had an *E coli* infection. Three patients were treated with cefotaxime, while one patient received moxalactam. The minimum inhibitory concentrations of the organisms were as follows: *K pneumoniae,* 0.03 μg/mL cefotaxime; *K pneumoniae,* 0.01 μg/mL cefotaxime; *E coli,* 0.125 μg/mL moxalactam. No minimum inhibitory concentration was available for the *K pneumoniae* isolated from one patient. All four patients were cleared of infecting bacteria, with sterile cerebrospinal fluid samples obtained within 24 hours of initiation of therapy with the beta-lactam antibiotics. The cerebrospinal fluid bactericidal activity of three patients was greater than 1:16.

The only complication of therapy was seen in a patient with thrombocytopenia, who, when treated with moxalactam, experienced massive lower gastrointestinal bleeding. These successes contrast markedly with the dismal results obtained prior to the introduction of these new agents, in which only two of nine patients had bacteriologic cure of their meningitis (determined by chi-squared with Yates correction; $P < 0.01$). Although this series is small, the data illustrate the impact of the third-generation cephalosporins on the treatment of this disease. With widespread use of the third-generation cephalosporins and the potential for resistance development, whether these agents will continue to be successful in treating this previously fatal infection is yet to be determined.

CHARLES E. CHERUBIN, MD
Bayley Seton Hospital
Staten Island, NY 10304

SHLOMO MAAYAN, MD
Metropolitan Hospital
New York, NY 10029

ROBERT H. K. ENG, MD
SHARON M. SMITH, PhD
Veterans Administration Medical Center
East Orange, NJ 07019

1. Cherubin CE, Marr JS, Sierra MF, Becker S: Listeria and gram-negative bacillary meningitis in New York City 1972–1979: frequent causes of meningitis in adults as well as children. *Am J Med* 1981; 71:199–209.
2. Cherubin CE, Corrado ML, Nair SR, Gombert M, Landesman S, Humbert G: The treatment of gram-negative meningitis: role of the new cephalosporin antibiotics. *Rev Infect Dis* 1982; 4(suppl): S453–S464.
3. Rahal Jr JJ: Moxalactam therapy for gram-negative bacillary meningitis. *Rev Infect Dis* 1982; 4(suppl):S606–609.
4. Corrado ML, Gombert ME, Cherubin CE: Designing appropriate therapy in the treatment of gram-negative bacillary meningitis. *JAMA* 1982; 248:71–74.

Ethical double standard

TO THE EDITOR: Two years ago, in a letter written to the editor of another journal,[1] I expressed outrage at the fact that physicians who were apparently guilty of fraudulently conducting biomedical research were "punished" by being relegated to the practice of medicine, as though medical practice somehow demanded a lower level of ethical behavior than does laboratory research. Others have also commented, from the point of view of the non-physician research scientist. One such comment seems to express envy: "the scientist who tampers with data will lose his job and his career. The MD in the same circumstance can enter into private practice, which need not be an economic penalty."[2]

My sense of outrage at the attitude of the medical powers that be, implicit in the treatment of fraud committed by physicians in the course of laboratory research, has not abated. To banish persons who have falsified data in research laboratories from their careers in the laboratory is appropriate. To relegate them, then, to the practice of medicine is to demonstrate that medical practice requires a lower level of ethical practice than does scientific research.

Since physicians, more than most other persons in this society, exercise a high degree of power over other people, it should follow that they ought to be held to the highest standards of ethical conduct. The profession of medicine is not—and its practitioners ought not permit it to be—open to persons who have demonstrated a lack of ethical standards in the pursuit of other occupations, related or unrelated to medicine.

STANLEY L. LEE, MD
Chief
Division of Hematology and Oncology
The Brookdale Hospital Medical Center
Linden Blvd at Brookdale Plaza
Brooklyn, NY 11212

1. Lee SL: Fraud in medical research. (letter) *JAMA* 1984; 251:215.
2. Kuttner RE: Fraud in science. *Science* 1985; 227:466.

The relationship of thrombocytopenia to the onset of persistent pulmonary hypertension of the newborn in the meconium aspiration syndrome

MICHAEL J. HORGAN, MD, NOEL JM CARRASCO, MD, HERMAN RISEMBERG, MD

ABSTRACT. We performed a prospective study of 48 full-term infants who were at high risk for persistent pulmonary hypertension of the newborn (PPHN) to define an association between PPHN and thrombocytopenia. Twenty-seven infants had meconium aspiration syndrome (MAS), four had diaphragmatic hernias, 16 had asphyxia as the primary diagnosis, and one infant had respiratory distress syndrome. Six of the 27 infants with MAS developed PPHN. The only infants with PPHN and thrombocytopenia were those with MAS (five out of six). Between 12 and 24 hours of age, the infants with MAS, PPHN, and thrombocytopenia had abnormal platelet counts. No significant differences among the infants could be detected in Apgar scores, gestational age, birth weight, maternal medication use, or in the patients' coagulation profile. More than 90% of the infants in whom PPHN developed were boys. We conclude that male infants with MAS and thrombocytopenia are at especially high risk for the development of PPHN.

(NY State J Med 1985; 85:245–247)

INTRODUCTION

Meconium staining is a relatively common occurrence in full-term infants, with an incidence of 8% to 10%.[1,2] Meconium aspiration occurs in approximately 20% of the infants noted to have meconium staining.[1] Aspiration has been associated with a variety of disorders ranging from mild respiratory distress that requires little or no supplemental oxygen, to severe respiratory distress with hypoxemia that is unresponsive to mechanical ventilation.[3,4]

In a retrospective study in 1980 Segall, et al[5] reported on 12 full-term infants with perinatal aspiration syndrome in conjunction with persistent pulmonary hypertension of the newborn (PPHN) and thrombocytopenia. In 1983, Levin, et al[6] performed a retrospective study of a similar group of newborns—some of whom had evidence of meconium aspiration syndrome—who had unresponsive pulmonary hypertension in association with thrombocytopenia. The postmortem examinations of these infants revealed diffuse pulmonary microthrombi.

We have recently completed a prospective study on a group of full-term infants at high risk for developing PPHN in an attempt to show an association between PPHN and thrombocytopenia. This report discusses those infants who developed PPHN and thrombocytopenia.

PATIENTS AND METHODS

During a 15-month period from August 1981 to November 1982, 535 full-term infants were admitted to the Albany Medical Center Hospital Newborn Intensive Care Unit (NICU). The study population of 48 infants represents 9% of the full-term infants that were admitted to the NICU during that time period. An infant was considered eligible for inclusion into the study if the following criteria were met: a gestational age of 37 weeks or greater derived from the maternal history of the last menstrual period and confirmed by clinical assessment of the infant[7]; the absence of known congenital heart disease; admission to the NICU for at least 12 hours; and the absence of sepsis. In addition, infants admitted to the study had to have a clinical picture consistent with either meconium aspiration syndrome (MAS), perinatal asphyxia, or persistent pulmonary hypertension of the newborn.

The symptoms of meconium aspiration syndrome were respiratory distress, the presence of meconium below the vocal cords, and a chest film[8] compatible with the diagnosis. Persistent pulmonary hypertension was defined as hypoxemia unresponsive to mechanical ventilation with a FIO_2 of 1.0, a negative hyperoxia response, and at least one of the following: an increased pulmonic component of the second heart sound (seen in 10 infants), an increased pulmonary artery pressure at cardiac catheterization (one infant),[9] or an increased ratio of right ventricular pre-ejection period to right ventricular ejection period ratio on echocardiography (nine infants).[10] Perinatal asphyxia was defined as a five-minute Apgar score of 6 or less.[11]

None of the mothers of the infants with thrombocytopenia were known to have a medical condition that might affect the platelet count, nor did they use any medication associated with platelet abnormalities during pregnancy.

The administration of fluids, blood products, antibiotics, and electrolyte solutions was done as part of the routine care in the NICU. Appropriate cultures, including urine, blood and cerebrospinal fluid, were obtained when needed, and antibiotics were administered at the discretion of the attending neonatologist. Platelet counts for each patient were obtained upon admission to the NICU and then once daily for the next three to five days or as long as indicated. Platelet counts of less than 100,000/μL were considered evidence of significant thrombocy-

---

From the Division of Neonatology, Department of Pediatrics, Albany Medical College of Union University, Albany.

Address correspondence to Dr Horgan, Instructor, Department of Pediatrics, Albany Medical College, Albany, NY 12208.

topenia,[12] and the test was repeated every 12 hours. Sampling for the platelet determinations was done by micropipette or venipuncture, or through an indwelling non-heparinized catheter. A decreased platelet count obtained by micropipette was verified by a venipuncture sample. Heparin was not used with any of the samplings. Upon admission, the prothrombin time, the partial thromboplastin time, and the presence of fibrin split products were also determined. These studies were repeated if the platelet count fell below $100,000/\mu L$, or if there was evidence of disseminated intravascular coagulation (DIC). Arterial blood gases were obtained upon admission and as indicated, at the discretion of the attending neonatologist. Only those infants with a diagnosis of PPHN were given a trial dose of tolazoline as described by Goetzman, et al.[13] Further use of tolazoline, based on the infant's response to the trial dose, was determined as outlined by Stevenson, et al.[14]

Comparisons between groups of infants were made by using multiple regression analysis, Student's T-test, and chi-square test. Comparisons among the groups of infants were done by analysis of variance.[15]

Parental permission was obtained for all infants, and the study was approved by the committee on research involving human subjects.

RESULTS

A total of 48 infants were entered into the study. The infants were placed into one of four groups depending on their clinical course: Group I, six infants (six boys) all with MAS and PPHN; Group II, 21 infants (14 boys) all with MAS without evidence of PPHN; Group III, 16 infants (14 boys) all with asphyxia as the primary diagnosis; Group IV, five infants (four boys) all with the diagnosis of PPHN without evidence of aspiration (four infants with diaphragmatic hernias and one infant with respiratory distress syndrome). Although 76% of the infants in the study were boys, there was no significant difference between the sexes in relation to group membership.

Thrombocytopenia (a platelet count of less than $100,000/\mu L$) was found in five of the six infants in Group I. The only other infant noted to have thrombocytopenia was from Group III, and it occurred in association with DIC. The thrombocytopenia observed in Group I was first noted between 12 and 24 hours of life ($P < 0.01$) at a mean age of 17.4 ± 5.2 hours and persisted for approximately 96 hours (Table I). PPHN was noted in the infants from Group I at a mean age of 25 hours +/− 8.5 hours. The time of onset of thrombocytopenia was significantly less than that of PPHN ($P < 0.02$). The frequency of thrombocytopenia in Group I was significantly greater than in Groups II, III, and IV ($P < 0.01$). The platelet counts during the 12 to 84 hour period from Group I were significantly lower than either the initial or final (96 hour) values ($P < 0.01$). There were no significant differences in the values for 12 to 84 hours. The infants with MAS and PPHN had significantly lower platelet counts from 12 to 84 hours of age (Table I).

Apart from the infant with DIC, there were no episodes of abnormal bleeding among the infants with thrombocytopenia. The prothrombin and partial thromboplastin times did not differ significantly among the groups. There were no positive cultures of blood, urine, or cerebrospinal fluid obtained from any of the infants. There were no significant differences in the gestational age, chronological age, birthweight, and 1-minute or 5-minute Apgar scores between the groups.

TABLE I. Platelet Counts (× 10^3 ± standard deviation) of Each Group at 12 Hour Intervals

Hours of Age	I	II	III	IV
0–12	151 ± 72	233 ± 70	194 ± 67	206 ± 44
12–24	100 ± 52*	193 ± 76	187 ± 85	175 ± 44
24–36	64.5 ± 29*	191 ± 65	183 ± 46	158 ± 38
36–48	78.5 ± 38*	215 ± 81	193 ± 96	108
48–60	57.5 ± 30*	266 ± 101	179 ± 90	115
60–72	59 ± 28*	242 ± 96	164 ± 90	—
72–84	57.5 ± 34*	366 ± 137	218 ± 126	—
84–96	98 ± 31	—	—	—

* $P < 0.01$, compared to Groups II, III, and IV.

Among the infants with PPHN, the response to a trial dose of tolazoline was generally poor. There were five non-responders, three marginal responders (a change in PaO_2 of 20–25 torr), and two clear responders (a change in PaO_2 of >50 torr). One infant from Group I, with an initial platelet count less than $70,000/\mu L$ and PPHN, was not given tolazoline.

The postmortem examinations of the two infants in Group I who died showed evidence of diffuse microthombi only in the lungs. This finding was not present in the postmortems from the other groups.

The predictors in order of importance for the determination of PPHN in the infants studied as determined by multiple regression analysis were the clinical group, the sex of the infant, the platelet count between 12 and 24 hours of age, and the platelet count between 24 and 36 hours of age.

DISCUSSION

This study shows that there is a subgroup of infants with MAS who develop PPHN. This subgroup can be identified prior to the onset of PPHN by noting the occurrence of thrombocytopenia within the first 24 hours of life. The postmortem examinations done on the infants who died with the triad of MAS, PPHN, and thrombocytopenia revealed evidence of pulmonary microthrombi consistent with that described by others.[6] Murphy, et al[16] noted a change in the pulmonary vascular smooth musculature in infants with PPHN and MAS who were dying. Although specific pulmonary vascular smooth muscle changes were not noted in the postmortem examinations described in this report, they were not specifically looked for by the pathologists. The two infants from Group I who died were clinically indistinguishable from the survivors of that group. This is similar to the observation of Murphy and colleagues[16] of uncertain value in the study population. The infants in this study who developed PPHN secondary to causes other than MAS, such as hypoplastic lungs and intrinsically high pulmonary vascular resistance, did not develop thrombocytopenia.

Although an association between PPHN, thrombocytopenia, and aspiration syndrome has been noted,[4–6] no clear explanation has been offered. The importance of platelets in the pathogenesis of pulmonary hypertension and hypoxemia in pulmonary embolism syndrome has been described.[17] The role of platelets involves the release of vasoconstrictors from aggregated platelets and the increase in pulmonary vascular resistance due to the aggregated platelets in the lungs.[18] Meconium has been shown to cause endothelial tissue injury in animal models.[19] In addition, meconium has known thromboplastic properties, and its inhalation can cause platelet aggregation and local microthrombi formation.[20] The role of meconium, therefore, may be to trigger the disorder, with local microthrombi progressing to diffuse disease.

The process that occurs in MAS with resultant PPHN may be similar to that which occurs in shock lung or adult respiratory distress syndrome. The features of persistent hypoxemia, thrombocytopenia, and pulmonary microthrombi are mutually evident.[21,22]

The response to vasodilator therapy (tolazoline) was not significantly different for either of the groups with

PPHN. The poor response reflects the underlying cause of the PPHN—a decreased cross-sectional area of the pulmonary vascular bed[17] and a lack of the normal number of functional pulmonary vessels[23] in Groups I and IV, respectively.

The fact that most of the infants with PPHN, MAS, and thrombocytopenia survived suggests that a spectrum of microthrombi diseases must exist. There were no clinical differences between survivors and non-survivors. However, one may speculate that if a larger number of infants is studied, a difference in response to vasodilator therapy or length of hypoxemia, for example, may become apparent. In addition, whereas the degree of microembolization may not be evident, the use of platelet kinetic studies such as those done with adult respiratory distress syndrome[24] may show the degree of pulmonary microembolization, which could be correlated with the clinical findings.

At the present time, there is no successful treatment for pulmonary microthrombi. Treatment methods have focused on relieving the vasoconstriction caused by the release of vasoactive substances from aggregated platelets.[13] Agents that block platelet aggregation (PGE_1, PGI_2),[25] and agents that promote platelet disaggregation (PGI_2)[26] have all been used. Unfortunately, none of these agents has proven to be safe or effective in newborns with PPHN.

There was an unusually high proportion of male infants in this study population. This has not been previously reported. Although male infants have greater morbidity and mortality rates than female infants of the same birth weight and gestational age,[27] there is no known association between the sex of the infant and the incidence of PPHN, thrombocytopenia, or respiratory distress. Further confirmation of a sex predominance and its role in the development of PPHN is needed.

The concomitant appearance of MAS and thrombocytopenia, and the subsequent development of PPHN may not be coincidental. We suggest that PPHN associated with MAS may be related to microthrombotic injury in infants noted to have thrombocytopenia.

Acknowledgments. Drs A. Bartoletti and A. Geis offered encouragement and critical review of the manuscript, Jaclyn Brilling provided editorial assistance, and Lorette Smith provided secretarial assistance.

REFERENCES

1. Gregory GA, Gooding CA, Phibbs RH, Tooley WH: Meconium aspiration in infants—a prospective study. *J Pediatr* 1974; 85:848–852.
2. Marshall R, Tyrala E, McAlister W, Sheehan M: Meconium aspiration syndrome: Neonatal and follow-up study. *Am J Obstet Gynecol* 1978; 131:672–676.
3. Wesenberg RL: *The Newborn Chest*. New York, Harper & Row Publishers Inc, 1973, pp 45, 121.
4. Fox WW, Gewitz MH, Dinwiddie R, Drummond WH, Peckham GJ: Pulmonary hypertension in the perinatal aspiration syndromes. *Pediatrics* 1977; 59:205–211.
5. Segall ML, Goetzman BW, Schick JB: Thrombocytopenia and pulmonary hypertension with the perinatal aspiration syndromes. *J Pediatr* 1980; 96:727–730.
6. Levin DL, Weinberg AG, Perkin RM: Pulmonary microthrombi syndrome in newborn infants with unresponsive persistent pulmonary hypertension. *J Pediatr* 1983; 102:299–303.
7. Dubowitz LM, Dubowitz V, Goldberg C: Clinical assessment of gestational age in the newborn infant. *J Pediat* 1970; 77:1–10.
8. Op. cit., Wesenberg, p 119.
9. Peckham GJ, Fox WW: Physiologic factors affecting pulmonary artery pressure in infants with persistent pulmonary hypertension. *J Pediatr* 1978; 93:1005–1010.
10. Riggs T, Hirschfeld S, Fanaroff A, Liebman J, Fletcher B, Meyer R: Persistence of fetal circulation syndrome: an echocardiographic study. *J Pediatr* 1977; 91:626–631.
11. Dorand RD: Neonatal asphyxia: an approach to physiology and management. *Pediatr Clin North Am* 1977; 27:455–461.
12. Oski FA, Naiman JL: Hematologic problems in the newborn, in Markowitz M (ed): *Major Problems in Clinical Pediatrics*, vol. 4. Philadelphia, WB Saunders Company, 1982, p 181.
13. Goetzman BW, Sunshine P, Johnson JD, et al: Neonatal hypoxia and pulmonary vasospasm: response to tolazoline. *J Pediatr* 1976; 89:617–621.
14. Stevenson DK, Kasting DS, Darnall Jr RA, et al: Refractory hypoxemia associated with neonatal pulmonary disease: The use and limitations of tolazoline. *J Pediatr* 1979; 95:595–599.
15. *SPSS-X User's Guide*, SPSS, Inc, New York, McGraw-Hill Book Co, 1983.
16. Murphy JD, Rabinovitch M, Goldstein JD, Reid LM: The structural basis of persistent pulmonary hypertension of the newborn infant. *J Pediatr* 1981; 98:962–967.
17. Cade JF: Platelets, pulmonary hypertension and pulmonary embolism, in Hirsh J, Cade JF, Gallus AS (eds): *Platelets, Drugs and Thrombosis Symposium, Hamilton, 1972*. Basel, Switzerland, S Karger AG, 1975, pp 191–199.
18. Bo G, Hognestad J: Pulmonary vascular effects of suddenly induced unilateral blood platelet aggregation. *Acta Physiol Scand* 1974; 90:237.
19. Tyler DC, Murphy J, Cheney FW: Mechanical and chemical damage to lung tissue caused by meconium aspiration. *Pediatrics* 1978; 62:454–459.
20. Suzuki S, Wake N, Yoshiki K: New neonatal problems of blood coagulation and fibrinolysis. II. Thromboplastic effect of amniotic fluid and its relation to lung maturity. *J Perinat Med* 1976; 4:221–226.
21. Rinaldo JE, Rogers RM: Adult respiratory-distress syndrome: changing concepts of lung injury and repair. *N Engl J Med* 1982; 306:900–909.
22. Perkin RM, Levin DL: Shock in the pediatric patient. *J Pediatr* 1982; 101:163–169, 319–332.
23. Levin DL, Rudolph AM, Heymann MA, Phibbs RH: Morphological development of the pulmonary vascular bed in fetal lambs. *Circulation* 1976; 53:144–151.
24. Schneider RC, Zapol WM, Carvalho AC: Platelet consumption and sequestration in severe acute respiratory failure. *Am Rev Respir Dis* 1980; 122:455–451.
25. Lock JE, Olley PM, Coceani F, Swyer PR, Rowe RD: Use of prostacyclin in persistent fetal circulation. *Lancet* 1979; 1:1343.
26. Gorman RR: Modulation of human platelet function by prostacyclin and thromboxane A_2. *Fed Proc* 1979; 38:83–88.
27. Williams RL, Creasy RK, Cunningham GC, Hawes WE, Norris FD, Tashiro M: Fetal growth and perinatal viability in California. *Obstet Gynecol* 1982; 59:624–632.

Humoral abnormalities in three patients with idiopathic chronic polyneuropathy

P. K. COYLE, MD, STEVEN E. SCHUTZER, MD, A. B. STERMAN, MD,
NANCY PERESS, MD, FREDERICK MILLER, MD

ABSTRACT. Three patients with chronic polyneuropathy of unknown origin were found to have abnormalities in humoral immunity characterized by anti-nerve antibodies, cold-reactive anti-lymphocyte antibodies, and circulating immune complexes. In two patients, clinical improvement coincided with plasmapheresis and removal of the anti-nerve antibodies. Certain patients with idiopathic peripheral neuropathy may benefit from therapy directed at correcting immunologic abnormalities.

(NY State J Med 1985; 85:248–250)

INTRODUCTION

Peripheral neuropathy is a common diagnostic problem confronting family practitioners, internists, and neurologists. Despite extensive investigation, a cause is found in only 76% of cases.[1] This is particularly true when the neuropathy is chronic and progressive. We describe three patients with idiopathic chronic polyneuropathy who shared similar abnormalities on both nerve biopsy and serologic immune testing. Two of them underwent plasmapheresis with good clinical responses.

METHODS AND RESULTS

Sural nerve biopsies were processed for routine light and electron microscopy. Fluorescence studies were carried out on normal sural nerve as well as patient biopsies. For direct immunofluorescence, biopsy slides were stained with optimal (as determined by chessboard titration) dilutions of fluorescein isothiocyanate-conjugated mono-specific goat antisera to human IgG, IgM, IgA, IgE, complement components, and fibrinogen (Kent Labs, Redwood, WA). Fluorescence was read using a Zeiss Axiomat NDC immunofluorescence microscope with a quartz halogen lamp as a light source. Indirect immunofluorescence on normal nerve was performed using optimal dilutions of the patient's serum (1:10 to 1:40); control serum was used at 1:2, 1:10, and 1:100 dilutions. Further staining was carried out in a similar manner.

Immune complexes were measured using a modified Raji cell assay[2] and solid phase C1q assay.[3] Cold-reactive anti-lymphocyte antibodies were measured by counting the percent of immunoglobulin-coated non-adherent lymphocytes at 4°C minus the percent at 37°C. Immunoglobulin coating was determined by a rosette assay using latex beads coated with rabbit anti-human heavy and light chain antibody.[4]

Case 1. A 44-year-old man complained of weakness and distal burning paresthesias following an episode of atypical pneumonia. He had had a non-caseating granuloma that was diagnosed on cervical lymph node biopsy, although he had never shown clinical or systemic signs of sarcoid. An initial examination revealed bilateral hand weakness of grade 4/5 (Medical Research Council), mild proximal weakness (5-/5), absent Achilles reflexes, and decreased pinprick response in a glove distribution. Within a three-week period, his condition deteriorated to the point where he required a wheelchair. Cold agglutinins were detected in his serum at a level of 1 to 256, as well as substantial levels of circulating immune complexes and cold-reactive anti-lymphocyte antibodies. The number of B-cells (25%) was elevated; the total number of T-cells, helper-inducer, and cytotoxic-suppressor cells were within normal limits. Blood chemistry studies, including tests for hematocrit, white blood cell count and differential, sedimentation rate, creatine phosphokinase, immunoelectrophoresis, serum protein electrophoresis, Bence-Jones protein using IEF, cryoglobulins, antinuclear antibody, syphilis, mononucleosis, thyroid functions, drug and heavy metal levels were all within normal limits. Skin testing was noncontributory. A lumbar puncture revealed an elevated albumin level (60 mg/dL) without cells. Electrophoresis of a spinal fluid sample showed a heavy band in the immunoglobulin region which could not be absorbed out with antigens to peripheral nerve, myelin basic protein, galactocerebroside, or mycoplasma. Electromyography and nerve conduction testing showed marked slowing of all nerve responses tested (median, 22.7 m/sec; ulnar, 19.1 m/sec; peroneal, 15.2 m/sec). Light and electron microscopic analysis of a sural nerve biopsy showed extensive demyelination, significant axonal loss with onion bulb formation, and a patchy inflammatory process. Macrophage infiltrates were seen in a perifascicular and subperineurial pattern, along with intrafascicular, multifocal lymphocyte infiltrates encircling the microvasculature. Quantitative analysis of unmyelinated fibers disclosed 53% with diameters less than 0.6 microns; this compared to 15% in a control nerve sample. Direct immunofluorescence of the sural nerve biopsy showed deposition of IgM and complement along the perineurium, and diffuse IgG staining within the endoneurium. There was no deposition of IgA, fibrinogen, or albumin. Indirect immunofluorescence identified IgG and IgM in the patient's serum; a control nerve sample stained in a similar pattern. His cerebrospinal fluid contained IgG, which was also found in a control nerve sample.

The patient was started on 60 mg of prednisone daily and had an initial response with increased ability in grip strength and arm extension (Fig 1). When it appeared that his improvement had reached a plateau, a course of eight two-liter plasma exchanges was done during a three-week period. There was a short-lived deterioration in his condition followed by marked improvement. Circulating immune complexes and anti-nerve antibodies disappeared during the plasmapheresis (Fig 1); two exchanges removed the antibody, and six exchanges cleared the circulating immune complexes. The patient was discharged from the hospital; he was able to walk with a walker. His condition has remained stable, and he has been free of anti-nerve antibody and immune complexes for more than two years.

Case 2. A 52-year-old man sought treatment for leg weakness that had progressed during a six-month period, with rapid deterioration during the preceding week. A history of asthma and multiple allergies was noted. An initial examination revealed bilateral foot drop, 3/5 weakness of both legs, 3/5 distal weakness in both arms, loss of reflexes in the lower extremities, and decreased vibration in

---

From the Departments of Neurology (Drs Coyle and Sterman) and Pathology (Drs Peress and Miller), Health Sciences Center, SUNY at Stony Brook, Stony Brook, NY, and the Department of Bacteriology and Immunology (Dr Schutzer), Rockefeller University, New York.

Address correspondence to Dr Coyle, Assistant Professor, Department of Neurology, HSC T-12, SUNY at Stony Brook, Stony Brook, NY 11794.

This research was supported by grants from the National Multiple Sclerosis Society (RG1360A2) and the Veterans Administration. Dr Coyle is the recipient of Teacher Investigator Development Award NS-00790 from the NINCDS and Dr Sterman is the recipient of New Investigator Research Award ES-02650 from the NIEHS.

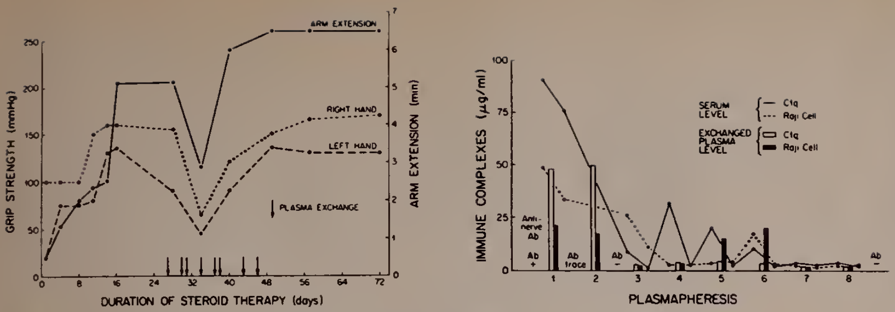


FIGURE 1. Left, effect of therapy on grip strength and arm extension; right, effect of the eight plasma exchanges on circulating immune complexes and antinerve antibody. Immune complex levels were measured in serum before and after exchange, as well as in the plasma removed. Anti-nerve antibody levels were measured in serum using indirect immunofluorescence.

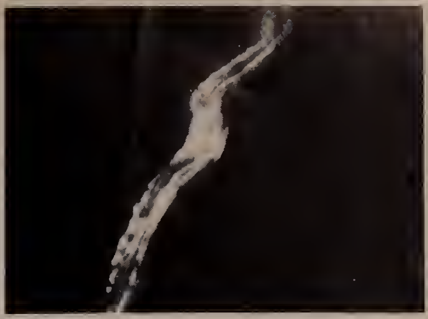


FIGURE 2. Direct immunofluorescence of sural nerve from patient 1 shows heavy granular staining of perineurium by IgM. (original magnification X 25)

the distal lower extremities.

Blood chemistry studies were within normal limits except for the level of circulating immune complexes and cold-reactive anti-lymphocyte antibodies; a polyclonal increase of immunoglobulins was found on immunoelectrophoresis. Electromyography and nerve conduction testing documented slowed nerve conduction. A sural nerve biopsy revealed focal demyelination of the nerve with perivascular collections of mononuclear cells (predominantly lymphocytes) in the perineurial connective tissue. Direct immunofluorescence showed linear deposition of IgM along the perineurium and fine granular stippling of IgG within the endoneurium. There was no fibrinogen, IgA, or albumin staining. Indirect immunofluorescence confirmed that the patient's serum contained circulating IgM and IgG that stained normal nerve in a similar pattern.

The patient was started on therapy with steroids. He showed initial improvement, but then relapsed to the point of being bed-ridden. He was begun on a course of plasmapheresis three times each week. Coincident with this, he improved dramatically to the point of being able to walk with a cane. He has been maintained for a year on steroid therapy (every other day) and weekly plasmapheresis. Anti-nerve antibodies and immune complexes disappeared following plasmapheresis.

Case 3. A 49-year-old man complained of difficulty walking and distal, burning paresthesias of three months' duration. The patient had had psoriasis previously. An examination revealed mild diffuse weakness (5-/5), absent Achilles reflexes, and decreased vibration in the distal lower extremities. Blood chemistry studies were within normal limits except for a polyclonal increase of IgG noted on immunoelectrophoresis, a positive direct and indirect Coombs test, a false positive syphilis test, circulating immune complexes, and cold-reactive lymphocyte antibody test. Electromyography and nerve conduction testing showed borderline ulnar motor conduction velocity and distal sensory latency, with diffuse mild motor unit loss in leg muscles consistent with a neuropathic process. Analysis of cerebrospinal fluid showed elevated albumin (61 mg/dL) without white cells. Extensive studies for metastatic cancer and infections, including tuberculosis and fungal cultures, viral cultures, bone marrow studies, gallium scan, computerized axial tomography and liver-spleen scans, and lymphangiogram were all negative for evidence of chronic infection or malignancy. A sural nerve biopsy showed evidence of demyelination. Direct immunofluorescence revealed heavy IgM staining of the perineurium. Similar antibodies were present in the patient's serum on indirect immunofluorescence. The patient was admitted to an outlying hospital and has been lost to follow-up.

Discussion

Three patients with chronic neuropathy of unknown etiology initially came to our attention because they shared a striking and unique abnormality on sural nerve biopsy. Immunofluorescent staining revealed autoantibodies deposited on the nerve. More importantly, these antibodies were found to be circulating in the serum of all three patients as well as in the cerebrospinal fluid of the first patient. The significance of anti-nerve antibodies is unclear since immunoglobulins, particularly of the IgG and IgM classes, have been reported to bind to nerve.[5] However, investigators have recently found multiple antibodies against peripheral nerve and myelin in certain patients with chronic polyneuropathy.[6,7] Latov[6] and others have found certain patients with plasma cell dyscrasias who show a demyelinating nueropathy along with IgM antibody directed against myelin-associated glycoprotein, or, more rarely, an axonal neuropathy along with antibody directed against chondroitin sulfate.[8,9] No information on the immune status of any of these patients was given. From an immunologic perspective the patients described in this report had evidence of abnormal humoral immunity (Table). In addition to anti-nerve antibodies, they had circulating immune complexes, cold-reactive antibodies directed against 12% to 20% of peripheral lymphocytes, cold agglutinins, false positive syphilis tests, positive Coombs tests, and elevated immunoglobulin levels. The two patients placed on steroids benefited temporarily. With the addition of plasmapheresis and coincidental removal of the autoantibodies and immune complexes, further improvement occurred in both patients and was particularly dramatic in one. We hypothesize that the removal of anti-nerve antibody correlated with clinical improvement. This is supported by a recent study of patients with Guillain-Barré syndrome,[10] as well as a case report of a patient with chronic polyneuropathy that shows a repeated association between removal of IgG anti-nerve antibody and clinical benefit.[11] The presence of circulating anti-nerve antibodies in patients with chronic neuropathy may be an indication for early and aggressive plasmapheresis therapy.

The nature and specificity of these anti-nerve antibodies remains to be determined. They bound to all normal human peripheral nerves tested. These cases were the only clear positives out of 108 sera examined from patients with a variety of neurologic diseases, including 30 patients with peripheral neuropathy and three patients undergoing plasmapheresis (one control serum was weakly positive). All three of these patients had IgM staining of the perineurium (Fig 2), as has been reported in certain

Humoral Abnormalities Detected in Three Patients with Chronic Neuropathy

Patient	(age/sex)	Clinical Features	Sural Nerve Biopsy	Humoral Abnormalities
1.	44/M	Weakness in all extremities Absent Achilles reflex Decreased pin in hands Progression over four mo. Slowed nerve conduction CSF cytoalbuminologic dissociation	Demyelination, inflammation, axonal damage IgM and complement (C3, C4, properdin) deposition along perineurium IgG deposition within endoneurium	Anti-nerve antibody in serum (IgM, IgG) and CSF (IgG) Cold reactive anti-lymphocyte antibody (20% of lymphocytes) Immune complexes (serum and CSF) Cold agglutinins
2.	52/M	Weakness in all extremities Absent reflexes in legs Decreased vibration in legs Progression over seven mo. Slowed nerve conduction CSF not examined	Demyelination, inflammation IgM deposition along perineurium IgG deposition within endoneurium	Anti-nerve antibody in serum (IgM, IgG) Cold-reactive anti-lymphocyte antibody (12%) Serum immune complexes Polyclonal immunoglobulin increase
3.	49/M	Weakness in all extremities Absent Achilles reflex Decreased vibration, hyperesthesia to pin in legs Progression over three mo. Motor unit loss CSF cytoalbuminologic dissociation	Demyelination IgM deposition along perineurium	Anti-nerve antibody in serum (IgM) Cold-reactive anti-lymphocyte antibody (12%) Serum immune complexes FP-VDRL Coombs positive (direct and indirect) Polyclonal immunoglobulin increase

cases of Guillain-Barré syndrome,[12] chronic paraproteinemic neuropathy,[13] and chronic axonal neuropathy.[14] Non-specific perineurial staining has also been reported.[14] In those patients however, and unlike those described here, other serum components were deposited along with IgM in the perineurium.

The perineurium is partly responsible for maintaining the integrity of nerve fibers in the endoneurium and for maintaining the proper ionic microenvironment.[15] It forms a diffusion barrier to the passage of materials into and out of the nerve fascicle. Mechanical damage to the perineurium, such as cutting a window, has been used as a model to produce nerve demyelination.[16] Were antibodies to attack this structure and allow leakage of serum components into the nerve fascicle, demyelination and disrupted nerve conduction might result. The first patient had complement deposited in the sural nerve along with IgM; he was the only one who showed extensive axonal damage in addition to demyelination. The first two patients also had evidence of diffuse deposits of IgG within the endoneurium. This seemed to correlate with inflammatory infiltrates, but further cases need to be studied.

The three patients are presented as evidence that humoral factors may be active in some chronic neuropathies. Sural nerve biopsy, with direct and indirect immunofluorescence, may prove useful in the evaluation of such patients. Documentation of the incidence and significance of specific perineurial antibody should be sought. Although idiopathic chronic polyneuropathy is a heterogeneous disorder, future immunologic studies may help identify a subgroup of patients with humoral abnormalities who may be candidates for plasmapheresis.

REFERENCES

1. Dyck PJ, Oviatt KF, Lambert EH: Intensive evaluation of referred unclassified neuropathies yields improved diagnosis. *Ann Neurol* 1981; 10:222-226.
2. Coyle PK, Banks N, Schutzer SE: A micro-ELISA Raji cell assay to detect immune complexes. *J Immunol Methods* 1984; 74:191-197.
3. Hunt JS, Kennedy MP, Barber KE, McGiven AR: A microplate adaptation of the solid-phase C1q immune complex assay. *J Immunol Methods* 1980; 33:267-275.
4. Coyle PK, Brooks BR, Hirsch RL, et al: Cerebrospinal-fluid lymphocyte populations and immune complexes in active multiple sclerosis. *Lancet* 1980; 2:229-232.
5. Abrams GM, Latov N, Hays AP, Sherman W, Zimmerman EA: Immunocytochemical studies of human peripheral nerve with serum from patients with polyneuropathy and paraproteinemia. *Neurology* 1982; 32:821-826.
6. Latov N, Gross RB, Kastelman J, et al: Complement-fixing antiperipheral nerve myelin antibodies in patients with inflammatory polyneuritis and with polyneuropathy and paraproteinemia. *Neurology* 1981; 31:1530-1534.
7. Panitch H, Hooper C, Hafler D: Antibodies to multiple central and peripheral myelin proteins in chronic demyelinating peripheral neuropathies. (abstract) *Neurology* 1983; 33:82.
8. Nemni R, Galassi G, Latov N, Sherman WH, Olarte MR, Hays AP: Polyneuropathy in nonmalignant IgM plasma cell dyscrasia: a morphological study. *Ann Neurol* 1983; 14:43-54.
9. Nobile-Orazio E, Vietorisz T, Messito M-J, Sherman WH, Latov N: Anti-MAG IgM antibodies in patients with neuropathy and IgM M proteins: detection by ELISA. *Neurology* 1983; 33:939-942.
10. Nyland H, Vedeler CA, Matre R, et al: Plasma exchange with Guillain-Barré-syndrome: Clinical improvement in patients with serum IgG antibodies to peripheral nerve tissue. *Int J Art Organs* 1984; 7:133-136.
11. Toyka KV, Augspach R, Wiethölter H, et al: Plasma exchange in chronic inflammatory polyneuropathy: evidence suggestive of a pathogenic humoral factor. *Muscle Nerve* 1982; 5:479-484.
12. Schott B, Chazot G, Kopp N, et al: Subperineurial space and immunoglobulin deposits in inflammatory and dysglobulinemic neuropathies, in Canal N, Pozza G (eds): *Peripheral Neuropathies.* Amsterdam, Elsevier, 1978, pp 421-426.
13. Smith IS, Kahn SN, Lacey BW, et al: Chronic demyelinating neuropathy associated with benign IgM paraproteinaemia. *Brain* 1983; 106:169-195.
14. Van Lis JM, Jennekens FG: Plasma proteins in human peripheral nerve. *J Neurol Sci* 1977; 34:329-341.
15. Ochs S: Calcium requirements for axoplasmic transport and the role of the perineurial sheath, in Jewett DL, McCarroll HR (eds): *Nerve Repair and Regeneration: Its Clinical and Experimental Basis.* St. Louis, CV Mosby, 1980, pp 77-88.
16. Spencer PS, Weinberg HJ, Raine CS, et al: The perineurial window—a new model of focal demyelination and remyelination. *Brain Res* 1975; 96:323-329.

ESSAYS IN PEDIATRICS

The "Essays in Pediatrics" series includes articles that focus on clinical management of the diseases of children and adolescents and discusses recent advances in basic science, as they relate to the needs of the practitioner. The series emphasizes changing trends, new directions in research, and current controversies in diagnosis and treatment.

Proposals for manuscripts for "Essays in Pediatrics" should be submitted to Carl Pochedly, MD, Director, Department of Pediatric Hematology/Oncology, Children's Hospital Medical Center, 910 Blackford St, Chattanooga, TN 37401.

Management of diabetes mellitus in children

IRENE N. SILLS, MD

The care of children with diabetes has changed greatly during the past 20 years. Improvements in the quality of insulin, self blood glucose monitoring, and the use of glycosylated hemoglobin to measure the success of regulation have upgraded the therapy offered to children with diabetes. Spurred on by the knowledge of the devastating complications in people with long-term diabetes, clinicians and researchers have tried newer, varied routes of insulin administration that attempt to mimic more closely the physiologic increases in insulin levels that accompany meals and the basal levels between meals.

The successful management of a child with diabetes, however, remains a challenge. The daily, unceasing demands of diabetes management may result in psychological and social difficulties for the child and the family. The focus for the physician must be on the total well-being of the child, and the challenge is to make the management plan optimal in both the medical and psychological senses.

SETTING THE GOALS

It is helpful to view diabetes management in terms of the attainment of goals. These goals must be individualized for each child and family, and are often influenced by the educational level, values, support system, and economic situation of a family. The goals also should not be static, but should be modified as a child grows older. In general, there are seven goals.

Growth. The child with diabetes should grow at a normal rate (5 cm annually after four years of age until puberty, at which time growth rates may approach 10 cm per year). The child must have a meal plan that provides the necessary calories for this growth (Table I).

Glucose regulation. Levels of blood glucose should be kept as close to normal as possible. The goal is to maintain preprandial blood glucose levels between 60 and 90 mg%, with peak postprandial blood glucose levels below 140 mg%.[1] Not all patients are candidates for this intensive therapy since striving to maintain a level of 60 to 90 mg% is likely to lead to intermittent lower and potentially dangerous glucose concentrations. Toddlers who may be at risk for permanent brain damage from hypoglycemia, and patients with long-term diabetes who might have impaired ability to sense hypoglycemic symptoms are not good candidates for intensive control. Preprandial glucose concentrations of 90 to 140 mg% are more realistic goals for these patients. Children who do not adhere to the management plan need to be motivated to follow the general principles of regular meal times and routine insulin administration before intensive management can be presented to them.

Prevention of hypoglycemia. Episodes of severe, unexplained hypoglycemia and those that interfere with a child's functioning at school or play should be evaluated. Occasional, mild hypoglycemic symptoms may be acceptable, especially in an older child. Maintenance of appropriate goals for glucose regulation, routine monitoring of blood glucose, and pre-exercise snacks may prevent such episodes.

Education. Successful management of diabetes can only come about if the child and family have a thorough understanding of the pathophysiology and treatment of diabetes. An important goal in diabetes management must be to provide ongoing education to the family (Table II).

TABLE I. Recommended Energy Intake

	Age (yr)	Weight (kg)	Energy needs (range) (kcal)
Children	1-3	13	1,300 (900-1,800)
	4-6	20	1,700 (1,300-2,300)
	7-10	28	2,400 (1,650-3,300)
Men	11-14	45	2,700 (2,000-3,700)
	15-18	66	2,800 (2,100-3,900)
Women	11-14	45	2,200 (1,500-3,000)
	15-18	55	2,100 (1,200-3,000)

Address correspondence to Dr Sills, Director, Children's Diabetes Center, The Children's Hospital at Buffalo, 219 Bryant St, Buffalo, NY 14222.

TABLE II. Diabetes Education Checklist

Basic pathophysiology of insulin-dependent diabetes mellitus and how it differs from non-insulin-dependent diabetes mellitus
Performance of urine testing for glucose and ketones
Technique of self-monitoring of blood glucose
Insulin storage and administration
Onset, peak, duration of action of child's insulin
Injection site rotation
Signs, symptoms, and prevention of ketoacidosis
Guidelines for administration of insulin and fluids during intercurrent illness
Signs, symptoms, and treatment of hypoglycemia
Use of glucagon in the event of unconsciousness due to hypoglycemia
Phenomenon of hypoglycemia and rebound hyperglycemia ("Somogyii" effect)
Meal planning
Exercise and its effect on blood glucose levels
Insulin dosage adjustment guidelines
Foot care
Importance of wearing medical identification
Traveling
Contraception
Smoking
Alcohol
Dental hygiene
Long-term complications

Management of intercurrent illnesses and stress. Metabolic decompensation may occur in a child with diabetes who is affected by an infectious illness, trauma, or emotional stress. The family must know the warning signs of impending decompensation and must understand how to adjust the treatment under these circumstances.

Psychological adjustment. The child's maturation must keep pace with the normal developmental stages of childhood and adolescence. Parental concerns about diabetes and the regimentation required to treat the illness should not inhibit this growth. A child should develop confidence and a positive self-image that will allow him or her to grow into adulthood and accept the responsibilities of maintaining good health habits. Emotional stress may affect the success of the diabetes regulation and, conversely, the success of the regulation may affect the psychological wellbeing of the child.

Prevention of long-term complications. A decade ago there were advocates of both "tight" and "loose" glucose control. Although the evidence is not conclusive, many diabetologists agree that maintenance of normal or near normal blood sugar levels in people with diabetes will delay or even prevent the long-term renal, ophthalmologic, neurologic, and vascular complications associated with diabetes. The National Institutes of Health has recently begun the Diabetes Control and Complications Trial, a multicenter, randomized, and controlled clinical study to determine if intensive management in patients with insulin dependent diabetes is feasible and beneficial with respect to preventing or delaying complications associated with diabetes.

Studies have shown that tight control is associated with improvements in metabolic and endocrine system functioning.[2] Maintenance of euglycemia has also been shown to lead to decreased retinal vessel permeability, decreased renal protein leakage, and improved motor nerve conduction velocity.[2] However, improvement in diabetes control has not been shown to reverse the changes of established diabetic nephropathy or to slow the progression of diabetic retinopathy.

ACHIEVING THE GOALS

Cooperation between the child's general pediatrician and a multidisciplinary medical team is essential in the management of diabetes in children. The team should be headed by a pediatrician with special expertise in diabetes management. The other members of the team should include a nurse educator, a dietician, and a counselor. Families should be able to contact a member of the diabetes team on a 24-hour basis. The child's general pediatrician should be kept well informed of the recommendations of the diabetes team.

Insulin. Insulin may vary according to the species of origin, the purity of the product, the potency and concentration, and the duration of action. It may be extracted from beef or pork pancreas or may be identical to human insulin. Beef insulin differs from human insulin by two amino acids, whereas pork insulin differs by one. Human insulin may be derived either from pork insulin by chemically replacing the differing amino acid or it may be synthesized by recombinant DNA technology by the bacterium *Escherichia coli*.

Animal insulin is antigenic when injected into humans, and patients with diabetes quickly develop insulin antibodies. The role of these antibodies is not known, but they may affect the biologic action of insulin. The prediction has been that the newer human insulins will result in a lesser immune response, and a recent study has confirmed this.[3]

Insulin may vary according to purity. Traditionally, when insulin was extracted from the pancreas, pancreatic byproducts were also extracted, such as glucagon, somatostatin, pancreatic polypeptide, vasoactive intestinal polypeptide, and proinsulin. Not only did these byproducts have undesirable biologic effects (glucagon raises the blood glucose, for example), but as foreign proteins, they served as stimuli for antibody production. In 1972, gel filtration chromatography and ion exchange chromatography were employed to improve the purity of insulin. Today, animal insulin may be called purified if it has less than 10 parts per million of proinsulin, but even those insulins not labelled purified have less than 50 parts per million of proinsulin, which is a thousand times more pure than was achieved prior to 1972.

Following are the current recommendations for using purified insulin: in patients recently found to have diabetes; in patients with demonstrated insulin allergy or resistance; in patients who may receive insulin intermittently to avoid an anamnestic antibody response; and in patients who develop lipoatrophy at the injection site.

Insulin is available in different strengths, but the most commonly used is U100, or 100 units per cc insulin. Insulin also has different durations of action (Table III), and it is important to note that the duration may vary in each particular patient.

Insulin regimens. There are several insulin regimens available. The choice of which one to use depends on the individual child, according to the biologic effect of the insulin in each child, the schedule of each child, the way the

TABLE III. Types of Insulin

Source	Brand	Manufacturer
Rapid Acting Insulin		
Regular (onset ½ hr, peak two to four hr, duration five to seven hr)		
beef-pork	Iletin I	Lilly
purified beef	Iletin II beef	Lilly
purified pork	Iletin II pork	Lilly
	Actrapid	Squibb Novo
	Velosulin	Nordisk
human	Humulin	Lilly
	Actrapid	Squibb Novo
Semilente (onset ½ hr, peak two to four hr, duration 12 to 16 hr)		
beef-pork	Iletin I	Lilly
purified pork	Semitard	Squibb Novo
Intermediate Acting Insulin		
NPH (onset 1½ hr, peak 6 to 12 hr, duration 18 to 24 hr)		
beef-pork	Iletin I	Lilly
purified beef	Iletin II beef	Lilly
purified pork	Iletin II pork	Lilly
	Insulatard	Nordisk
human	Humulin	Lilly
Lente (onset 1½ hr, peak 6 to 12 hr, duration 24 to 28 hr)		
beef-pork	Iletin I	Lilly
purified beef-pork	Lentard	Squibb Novo
purified beef	Iletin II beef	Lilly
purified pork	Iletin II pork	Lilly
	Monotard	Squibb Novo
human	Monotard	Squibb Novo
Prolonged Acting Insulin		
Ultralente (onset 6 to 10 hr, peak 18 to 24 hr, duration 36 hr)		
beef-pork	Iletin I	Lilly
purified beef	Ultratard	Squibb Novo

meal plan is constructed, and the willingness of a child and family to comply with a particular regimen.

Blood glucose levels rise with each meal, and insulin should be administered to best mimic the basal level of insulin between meals and the increase with meals. Insulin may be given once, twice, or several times daily (Table IV).

At The Children's Hospital at Buffalo, one preferred method for instituting insulin therapy is to try a twice-daily injection of purified pork or human short-acting and intermediate-acting insulin. The insulin sensitivity of a patient is first assessed during a 24-hour period by administering short-acting insulin (0.15-0.25 U/kg) subcutaneously before meals and a midnight snack. Judging from the requirements of the first 24 hours, the patient is then switched to the twice daily injection of short-acting and intermediate-acting insulin.[1] Most children accept two injections when this regimen is started, whereas management plans of more than two injections require special dedication.

TABLE IV. Insulin Regimens

Pre-breakfast	Pre-lunch	Pre-supper	Bedtime
Short/intermediate			
Short/intermediate		Short/intermediate	
Short/intermediate		Short	Intermediate
Short	Short	Short	Intermediate
Short	Short	Short	Prolonged

Short = short acting.
Intermediate = intermediate acting.
Prolonged = prolonged acting.

Since 1978, when the first case report on the use of the continuous subcutaneous insulin infusion (CSII) pump was published, there has been much hope that CSII would offer an improved way to deliver insulin, since the pump can supply both a steady basal level of insulin and preprandial boluses that mimic normal metabolism.

The infusion pumps that are currently in use are programmed to deliver short-acting insulin at a rate set by the user ("open loop pumps"). The pump is unable simultaneously to measure blood glucose and to infuse a pre-calculated amount of insulin based on the glucose level. The user must monitor blood glucose levels frequently and must be meticulous in following a meal plan in order to judge the amount of insulin needed for infusion.

Some clinicians have found that CSII provides flexibility in scheduling as well as improvement in glucose regulation; many of the benefits of "tight" regulation have been demonstrated in patients on CSII. Other studies have shown that comparable levels of regulation may be achieved with either CSII or multiple subcutaneous insulin injections.[4]

Concern over the safety of pump therapy was raised after the Centers for Disease Control reported on 11 patients who died while receiving insulin pump treatment.[5] Many of the deaths were unrelated to insulin therapy, and none could be attributed to pump malfunction. A subsequent investigation[6] concluded that the use of CSII pumps was not associated with a greater than expected number of deaths in patients with insulin dependent diabetes, but caution was expressed that candidates for CSII be chosen carefully.

CSII has limited potential use in children. The extra burden of wearing a machine and caring for it requires an exceptionally well motivated, mature child and excellent family support. If the same success can be achieved with multiple injections of insulin, there is even less reason to advocate CSII. If CSII is contemplated for a child with poor regulation, it is important to determine that poor compliance or psychosocial stress is not the basis of the poor regulation.

Meal planning. The meal plan should be designed so as to include a caloric intake, including essential nutrients and vitamins, sufficient to support normal growth and development. The meals are timed on a regular schedule based on the patient's lifestyle, physical activity, and insulin schedule. Most children eat three meals and two snacks daily. The meal plan should be written for each child by a dietician who is also responsible for educating the patient and family about nutrition.

The composition of the diet is usually 50% to 60% carbohydrate, 15% to 20% protein, and 25% to 35% fat. It was only in 1979 that the percentage of carbohydrate was increased in an attempt to reduce fat consumption and blood lipid levels. The recommendation was to use complex carbohydrates as opposed to simple sugars. The benefits of including high fiber as the carbohydrate have been studied, but there is no proven advantage in glucose control for children with insulin-dependent diabetes.[7] High fiber diets are unpalatable to many children, thus limiting the usefulness of such diets.

Since 1950, meal planning for persons with diabetes has been based on the concept of exchange lists: foods are grouped into six categories according to similarities in composition (milk, meat, vegetable, fruit, bread, and fat). In 1976, further refinements divided the milk and meat sections according to their fat content, as part of the attempt to limit saturated fat and cholesterol intake. Patients have meal plans that list the quantities of foods that should be selected from the food groups at meals and snacks, thereby theoretically standardizing intake from day to day.

More recent research has shown that foods listed together in a particular group may result in different elevations in blood glucose levels. The so-called "glycemic index" (rise in blood glucose after eating the food) may be larger for a baked potato than for an equivalent serving of boiled rice.[8] It has been realized that much depends on the texture and digestibility of the food, whether it is cooked or raw, and with what other food stuffs it is combined.

Much publicity has been given to the glycemic index of a "forbidden" sweet like ice cream versus a "permitted" food like a baked potato, since the ice cream has the lower glycemic index. Nutritionists are cautioning that persons with diabetes should not substitute ice cream for potatoes, since there is nutritional value in the potato that may not be obtained from the ice cream and since the ice cream also contains cholesterol and saturated fats that may affect the development of atherosclerosis.

The use of artificial sweeteners by children with diabetes must be viewed with caution. The American Diabetes Association has urged that saccharin not be removed from the market because of its usefulness to people with diabetes. The safety of the most recent addition to this market group, aspartame, has been questioned, but the Food and Drug Administration has found it safe for consumption. Ideally, one would like to proscribe the use of artificial sweeteners, since there still lurk unanswered questions about their long-term safety. In practice, however, since children in our society are constantly tempted by sweets, we do recommend use of the artificial sweeteners.

Exercise. Exercise is important in diabetes regulation since it leads to a lowering of blood glucose levels. Exercise does carry with it the risk of hypoglycemia. In addition, studies have shown that while exercise is beneficial in patients with regulated, non-ketotic diabetes, it may have deleterious effects in the poorly controlled patient.[9]

The benefits of regular exercise on overall glucose regulation in diabetes have not been proven, although training has been shown to increase insulin sensitivity.[10] Exercise still remains an important part of diabetes management, because it improves cardiovascular fitness and lipid profiles, and it promotes a sense of wellbeing.

For children with diabetes who enjoy exercise, it is important that they participate in appropriate activities and that they are taught how to exercise safely without risking hypoglycemia. Children should consume an additional snack before exercise, with the size and type of snack dependent on the strenuousness and duration of the exercise. For those children who are overweight, it is a better idea to exercise regularly at the same time of day and to lower the insulin dose that would be acting at that time of day rather than increasing caloric intake.

Illness and stress. Illness and other stresses, including emotional upset, cause secretion of counter-regulatory hormones (growth hormone, catecholamines, glucagon, cortisol) that result in an elevation in blood glucose. The meal plan must be adjusted if a child is unable to eat solid foods; the plan should include liquids to provide both calories and fluids.

Monitoring of blood glucose and urinary ketones should be done every three to four hours to determine the need for additional insulin[1,11] and to avoid the development of ketoacidosis. Vomiting in a child with diabetes is an important sign, since such a child may become rapidly dehydrated due to an inability of the kidneys to conserve fluid in the face of glucose-induced diuresis; emesis may also be a sign of ketoacidosis.

Home monitoring and adjustment of insulin dosage. Measurement of glycosylated hemoglobin is currently the best overall estimate of the degree of hyperglycemia during the preceding eight to 12 weeks.[12] Hemoglobin A is glycosylated in a non-enzymatic reaction and remains glycosylated for the 120-day lifespan of the red blood cell. Glycosylated hemoglobin is not, however, an indicator of how the treatment plan should be modified. For that, the child with diabetes must do continuous, daily monitoring.

In the past, testing urine for glucose was the only way to monitor the effect of a particular insulin dose and meal plan. The serum glucose was estimated from knowing the concentration of glucose in a double voided urine specimen. In 1976, Malone and colleagues[13] reported that simultaneous determinations of plasma glucose and urinary glucose concentrations gave a range of plasma glucose levels for each urinary glucose concentration, indicating that an individual urine test may not be a good predictor of the coincident plasma glucose level. The concentration of glucose in the urine is what is being tested; therefore without knowledge of the volume of urine, the same amount of glucose may be present in vastly different concentrations.

The availability of self-glucose monitoring has revolutionized diabetes care.[14] The ability of the patient to know his or her blood glucose level to an accuracy of 10% to 15% has eliminated the vagaries of urinary glucose testing. The procedure is done by pricking the side of a finger using an automatic lancet. The blood is squeezed into a drop on a strip that is measured either visually or with the use of a reflectance meter. Ideally, the tests are done four times a day—before meals and a bedtime snack.

The results are recorded and are used to adjust the insulin dose or to vary the meal plan. Changes are made in response to patterns of plasma glucose levels and not in response to a single blood glucose test. The doses of insulin are adjusted in increments of 10% based on the readings that reflect the action of the particular insulin.[1]

Children who are introduced to the concept of self-monitoring of glucose at the onset of their diabetes respond quite well to the procedure. However, like all aspects of diabetes management, the physician must be prepared to modify the frequency of such testing based on a particular child's tolerance for the procedure.

In adjusting insulin dosages, it is important to remember that children with newly diagnosed diabetes often experience a temporary remission or "honeymoon phase" of their diabetes weeks after diagnosis. This must be anticipated, so that the insulin dose is lowered accordingly.

The physician also needs to be aware of the phenomenon of hypoglycemia precipitating hyperglycemia ("Somogyii effect"). Hypoglycemia may not always be accompanied by symptoms, especially if it occurs during sleep. However, counter-regulatory hormones will cause a rise in blood glucose when they are stimulated by hypoglycemia, and in a child with diabetes the glucose level will rise far above normal. If the insulin dose is increased when the morning blood glucose level is checked and is found to be elevated, there will be additional episodes of hypoglycemia causing rebound hyperglycemia. This may be occurring if there is no lowering of elevated glucose levels with increases of insulin of 20% to 30% with doses totaling more than 1 U/kg to 1.5 U/kg in a child who is compliant with the meal plan. The insulin dose should then be decreased.

Psychosocial Aspects of Diabetes

Diabetes is a chronic disease that places many demands on the child and the family. The difficulties in adhering to a daily routine, the need to resist conforming to certain peer standards, the disruption in intrafamilial relationships, and the fear of long-term complications if the medical attention is not optimal may lead to potential psychosocial difficulties and adjustment problems. The child should be included at all stages of the treatment to the extent of his developmental capabilities.[15] A child with diabetes needs to develop a positive self-image. The use of terms like "cheat" to describe the non-adherence to the meal plan makes a child with diabetes feel less trustworthy. Such disparaging terms should be avoided. The child with diabetes should be cared for with an optimistic outlook. Questions about long-term complications should be answered truthfully, but the eventuality of complications should not be used to threaten a child who is not compliant.

Family counselling and individual counselling with the team counsellor should be suggested to all families, with the encouragement that shock, guilt, denial, anger, and depression are normal stages towards acceptance of the diagnosis. Counselling may assure families that their distress is part of a usual reaction to the diagnosis, and it does not mean that they are in some way weak or incapable. Some families will have more extreme, pathologic reactions that require more intensive intervention.

Adjustment to diabetes is assisted by participation in peer support groups, both for the child and the parents. Many communities have such groups, in which parents and children may discuss their fears and concerns with empathetic others. Since the establishment of the first camp for children with diabetes in 1925, summer camping has played a significant role in helping children with diabetes learn about their disease, care for themselves without parental supervision, and meet other children who share the same medical problem.

Monitoring the Long-Term Outcome

More than 95% of people with diabetes of 15 years' duration will have background diabetic retinopathy. After 30 years, half will proceed to the more severe proliferative retinopathy; blindness will occur in 3% to 5% of patients with insulin-dependent diabetes. Laser therapy and vitrectomy have reduced the incidence of new blindness by as much as 60%. The Early Treatment Diabetic Retinopathy Study is being undertaken to evaluate the effects of treatment with laser and aspirin therapy before the proliferative stage.

However, since there are specific therapies available, it becomes even more important to be aware of any eye changes. Children with diabetes of five to seven years' duration should have annual eye examinations performed by an ophthalmologist. Because eye changes may occur with a shorter duration of the disease,[16] especially in adolescents, it is wise to have this population checked at five years' duration.

Macroproteinuria that can lead to renal failure from diabetic glomerulosclerosis will develop in 40% of patients who have had diabetes for 25 years.[17] The only way to prevent this is to try to achieve the most meticulous care possible at an early stage. Measurements of 24-hour urinary protein will help to quantitate significant protein excretion (>500 mg/24 hr). Hypertension may exacerbate the nephropathy, and it has been shown that antihypertensive therapy may reduce the proteinuria.[18] Routine measurements of blood pressure are indicated in the management of children with diabetes.

In The Future

The future looks hopeful for persons with insulin-dependent diabetes. Researchers are looking for new ways to give insulin without injections, such as administering it in the form of a nasal spray. Subcutaneous insulin pumps have become smaller and more responsive to the needs of the individual; implantable pumps are being developed. Work is progressing on an implantable glucose sensor that will be most like an artificial pancreas.

Segmental pancreatic transplantation has been less successful than transplantation of other organs. Researchers are now perfecting the ability to transplant individual islet cells. The procedure includes pretreating the islet cells so that they are less antigenic; thus the transplantation may not require the concomitant immunosuppression that makes transplantation risky.

The ultimate hope is that one day the pathophysiologic processes that result in pancreatic islet cell destruction will be prevented, and that diabetes will become a disease of the past.

References

1. Skyler JS, Skyler DL, Seigler DE, O'Sullivan MJ: Algorithms for adjustment of insulin dosage by patients who monitor blood glucose. *Diabetes Care* 1981; 4:311–318.
2. Tamborlane WV, Sherwin RS: Diabetes control and complications: new strategies and insights. *J Pediatr* 1983; 102:805–813.
3. Fineberg SE, Galloway JA, Fineberg NS, Rathbun MJ, Hufferd S: Immunogenicity of recombinant DNA human insulin. *Diabetologia* 1983; 25:465–469.
4. Schiffrin A, Belmonte MM: Comparison between continuous subcutaneous insulin infusion and multiple injections of insulin. *Diabetes* 1982; 31:255–264.
5. Centers for Disease Control: Deaths among patients using continuous subcutaneous insulin infusion pumps—United States. *MMWR* 1982; 31:80–87.
6. Teutsch SM, Herman WH, Dwyer DM, Lane JM: Mortality among diabetic patients using continuous subcutaneous insulin infusion pumps. *N Engl J Med* 1984; 310:361–368.
7. Lindsay AN, Hardy S, Jarrett L, Rallison ML: High carbohydrate, high fiber diet in children with type 1 diabetes mellitus. *Diabetes Care* 1984; 7:63–67.
8. Jenkins DJ, Wolever TM, Taylor RH, et al: Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr* 1981; 34:362–366.
9. Berger M, Berchtold P, Cuppers HJ, et al: Metabolic and hormonal effects of muscular exercise in juvenile type diabetics. *Diabetologia* 1977; 13:355–365.
10. Wallberg-Henriksson H, Gunnarsson R, Henriksson J, et al: Increased pe-

ripheral insulin sensitivity and muscle mitochondrial enzymes but unchanged blood glucose control in type I diabetics after physical training. *Diabetes* 1982; 31:1044-1050.

11. Daneman D, Frank M: Managing intercurrent illness in the child with diabetes. *Clin Diabetes* 1984; 2:1-7.

12. Goldstein DE, Parker KM, England JD, et al: Clinical application of glycosylated hemoglobin measurements. *Diabetes* 1982; 31 (suppl 3):70-78.

13. Malone JI, Rosenbloom AL, Grgic A, Weber FT: The role of urine sugar in diabetic management. *Am J Dis Child* 1976; 130:1324-1327.

14. Self monitoring methods for blood glucose. *Med Lett Drugs Ther* 1983; 25:42.

15. Kaiser S: Infancy through adolescence: a developmental approach, in Van Son A (ed): *Diabetes and Patient Education: A Daily Nursing Challenge*. New York, Appleton-Century-Crofts, 1982, pp 69-84.

16. Frank RN, Hoffman WH, Podgor MJ, et al: Retinopathy in juvenile-onset type I diabetes of short duration. *Diabetes* 1982; 31:874-882.

17. Viberti G, Keen H: The patterns of proteinuria in diabetes mellitus. *Diabetes* 1984; 33:686-692.

18. Parving H, Andersen AR, Smidt UM, Friisberg B, Svendsen PA: Reduced albuminuria during early and aggressive antihypertensive treatment of insulin-dependent diabetic patients with diabetic nephropathy. *Diabetes Care* 1981; 4:459-463.

FROM THE LIBRARY

PROGNOSIS AND TREATMENT OF DIABETES MELLITUS

John Rollo, an English surgeon, in the year 1797, was the first to call attention to the diet in the treatment of diabetes mellitus. He observed the action of animal foods and fats in this disease, and advised their use. He also noticed, when carbohydrates were given, that the symptoms became aggravated, and hence counseled against them. He, however, was too severe in excluding vegetable food of every kind, but wisely saw the use of the administration of alkalies and laxatives.

Since Rollo's time the question of diet in diabetes has been studied by clinicians, more so during the past two decades by such men as Cantani, Pavy, Seegen, Külz, Ebstein, Naunyn, von Noorden, Williamson, Hirschfeld and a host of others who have given this subject the closest attention. So that to-day this important therapeutic measurement has been thoroughly systematized. Rubner has given us in tables the caloric requirements of individuals of different weights and occupations; and Koenig has analyzed the different foods, dividing them into their component parts of albumins, carbohydrates and fats, so that their exact caloric equivalents can be exactly computed.

Knowing the exact excretion of sugar of a diabetic and his weight, his nutritive needs are readily ascertained from these tables.

Chemists have come to our aid in preparing aleuronat and almond flour, which, notwithstanding the disagreeable taste of the former, in some measure replace wheaten flour, and they have given us such comparatively harmless coal-tar products as saccharine and crystallose, to replace the sugars, the two foods so harmful in diabetes and for which patients so longingly crave. From almond meal very palatable cakes, containing a very small quantity of carbohydrates, can be produced, and champagnes and wines containing one-half per cent or less of sugar can be found on sale. The formula of an artificial milk containing but a minute quantity of lactose was first published by Williamson in England. So that today the diet of a diabetic can be made so various and palatable that, excluding those who must be placed on a very rigid diet, the majority are thoroughly content with their menus, an advantage which both patients and practitioners will recognize.

Inasmuch as the exact cause of diabetes mellitus is still an unsettled question, our chief endeavor has been and is to combat the symptoms as they present themselves. Where syphilis or neurasthenia can be defined as the cause, special treatment must be directed against them, but whatever the etiological factor or factors may be, our whole aim must be to increase the power of the system for oxidizing carbohydrates and to diminish the hyperglycaemia—in other words, to make our patient aglycosuric, or sugar-free.

To do so, no method which excludes diatetic treatment is of any avail. I am a believer in the efficacy of medicines in this disease; to the dietary they are important adjuncts.

ABRAHAM MAYER, MD
(*NY State J Med* 1902; 2:42-43)

Medical cataphoresis

Electrical experimentation and 19th century therapeutics

John S. Haller, Jr

Throughout the latter half of the 19th century, physicians and electrical specialists experimented with magnetism or metallo-therapy, the galvanic and the faradic forms of free electricity moving as a current, and the frictional or static electricity known as Franklinism. Initially, these enthusiastic panegyrists relied on electricity to treat certain nervous states and diseases, and for diagnostic and pathologic purposes. Broader acceptance of such therapy came with the fashionable use of electricity in intra-uterine electrolysis, galvano-puncture, and the galvanic treatment of paralysis, impotence, and insanity. Rheumatism, arthritis, and the vagaries of neurasthenia also seemed ripe for this new type of therapeutics. For a brief period of time, the power and prestige of electrical treatment knew few bounds among advocates, even though much of the reasoning remained empirical. Although proponents of electricity did their best to create an environment supportive of its application, their efforts remained more focused than they sometimes believed and thus limited the impact of electrical medicine to a narrow group of urban specialists.[1,2] (Figs 1–3)

EARLY YEARS

That medicines could be introduced into the human body through the skin had long been known and practiced through such forms as anointing, inunction, moxa, actual cautery, and massage. By the mid-19th century, intradermic injection into the dermis, diadermic or hypodermic injection into the subcutaneous connective tissues, and parenchymatous injection into the muscular masses had become popular alternatives to topical and endermic medication. That medicines could be introduced "by intercalating the part to be treated in the external circuit of the galvanic battery" [3,4] was less known than any of the former methods. Physicians and electrical experimenters identified this latter technique with such terms as medical cataphoresis, anodal diffusion, cataphoric action, Porret's phenomenon, electro-vection, electro-filtration, voltaic narcotism, electric osmose, and electric endosmose. Despite varying terminology, they implied by these names "the introduction of drugs or medicaments into the human body by means of electric current." [3,4]

Reuss of Moscow was one of the first to notice the passage of electric current through the body tissue.[5] Later, Porrett observed that as electric current passed through muscular tissue, the fiber at the negative pole exhibited a swelling.[5] Porret concluded that fluids could be transported through animal tissue, and he identified the process as electric endosmosis. This discovery, which he announced in *Annals of Philosophy* in 1815, became known for a brief period as "Porret's phenomenon." [6]

Medical cataphoresis was based on the principle of osmosis: an electric current could be passed through two liquids (one liquid was a medicament and the other was the body fluid) across a porous wall or septum which separated them. The skin and tissue served as the porous wall and the movement of liquid took place in the direction of the current: the level of fluid in the body was raised on the side of the negative electrode and lowered on the side of the positive electrode. Not comprehending fully the phenomenon before them, physicians tended to explain the process with less than scientific rigor. As one explained it, "the drug to be administered is forcibly pushed in through the skin by the normal flow of the current from the positive to the negative pole." [3]

Early developers of electrical cataphoresis included Fabre-Palaprat who, in 1833, claimed that iodine could travel from one arm to the other by the aid of electricity, and Napier who, in 1845, noted that "the endosmotic current will follow the electric, although in a contrary direction to that which it would take were there no electric current passing, showing that, under these circumstances, it has its origin in the passage of the electric force." [3] In 1846, Lenke announced the cure of several intractable diseases by the galvanic introduction of medicines into the system.[7–9] A few years later, in 1858, Clemens[7–9] claimed success with the cataphoric use of iodine, while Richardson[10] of London proposed a combination of electricity and narcotics. In an article entitled "On voltaic narcotism," published in 1859, Richardson reported on tests on the anesthetic qualities of aconite in combination with chloroform for the extraction of teeth. In preparing for cataphoric treatment, Richardson passed current through a fine wire anode that he placed around the tooth which was packed in cotton and soaked in the aconite and chloroform mixture; this technique effectively anesthetized the wound area in about five minutes. He first demonstrated

---

Address correspondence to Mr Haller, Professor, Department of History, California State University at Long Beach, 1250 Bellflower Blvd, Long Beach, CA 90840.

Figures are courtesy of California State University, Long Beach.

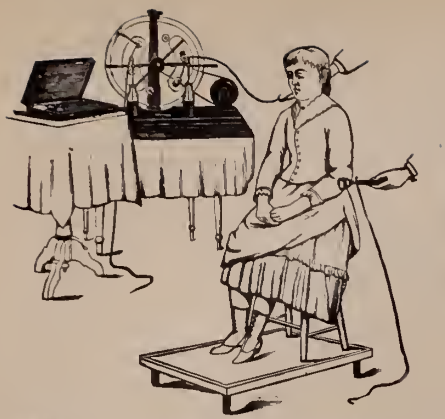


FIGURE 1. Franklinization or static treatment.

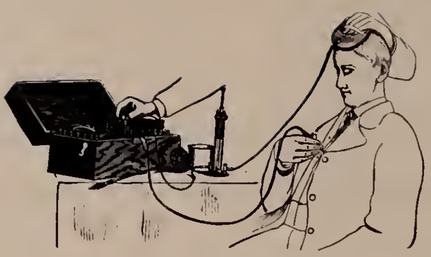


FIGURE 2. Central galvinization.

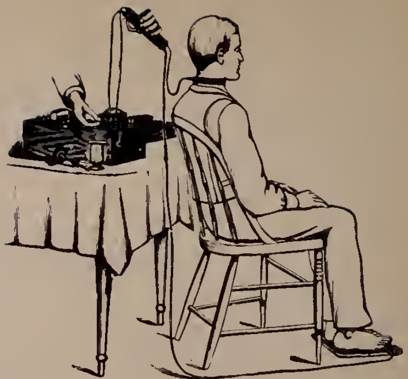


FIGURE 3. General faradization.

the success of his treatment on dogs and rabbits and later employed the method successfully on humans, treating them for strangulated hernia, tumor, and staphyloma of the cornea. Richardson chose to abandon his experimentation altogether due to professional opposition and the introduction of general anesthesia.[11]

Cataphoric Revival

As a medical technique, cataphoresis remained undeveloped for some 25 years until it was revived by Wagner and Adamkiewicz in 1886.[15] A year later, Reynolds[15] introduced the cataphoric application of cocaine in his dermatologic practice and, in 1888, Peterson[11] inaugurated a series of more than a hundred experiments. Demonstrations of cataphoric application to dentistry were noted by McGrath in 1888, and by Westlake in a paper before the New York State Dental Society in 1892.[12,13] An article written by Peterson in 1889 and entitled "Electric cataphoresis as a therapeutic measure," served as the catalyst to a revival in interest among American physicians.[12,13] In the same year, Lawrence and Harris described the cataphoric method of medication before the Society of Arts; again in 1889, Cagney used the process to treat labyrinthine deafness, lead palsy, and syphilitic gummata.[9] Three years later, Gillette presented his experiments in a paper before the American Dental Association. And Jack,[9] writing in 1902, noted success in over 260 cases using a dry cell battery, an ammeter to measure current strength, and a Williams controller to regulate the electric irritation (Figs 4, 5).

That medicines in their entirety could penetrate the skin through cataphoresis was accepted without question by those physicians and specialists experimenting with the process. To what extent they were agreed on its practical utility, however, was not sufficiently determined. To be sure, cataphoresis could not compete with the hypodermic syringe or with oral medication. Moreover, certain procedures were necessary, some quite complicated. While cocaine anesthesia could effectively be applied by electricity, the problems of battery maintenance, of determining the amount of electric current necessary, and of measuring the dose with any accuracy remained. Nevertheless,

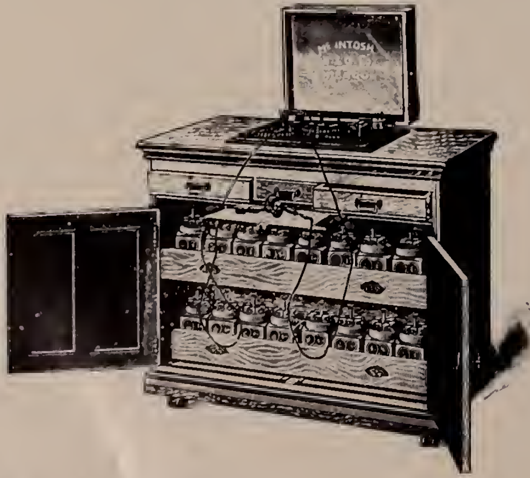


FIGURE 4. Galvanic cabinet.

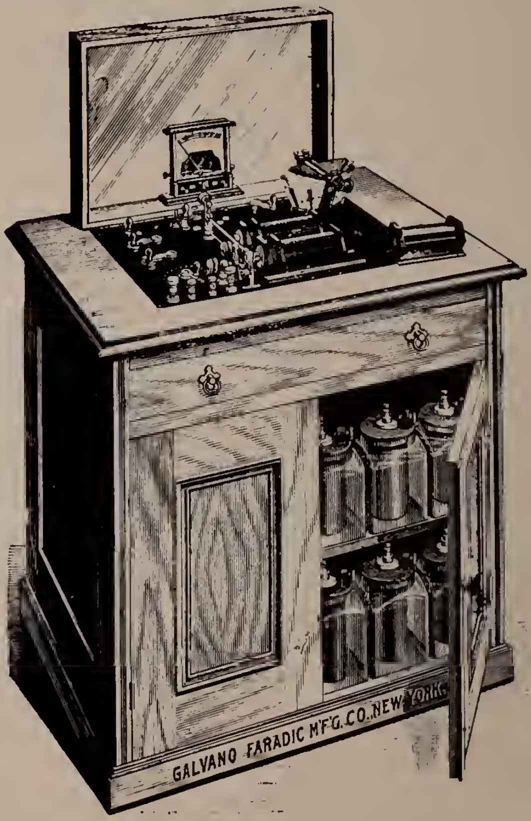

GALVANO FARADIC M'F'G. CO. NEW YORK

FIGURE 5. Galvanic office cabinet containing 50 cells, meter, rheostat, and faradic coils.

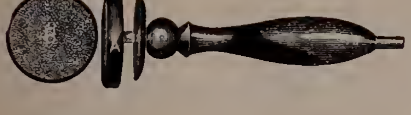


FIGURE 6. Carbon electrode.

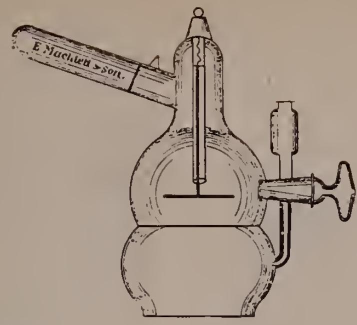

E. Machlett & Son.

FIGURE 7. Vacuum electrode.

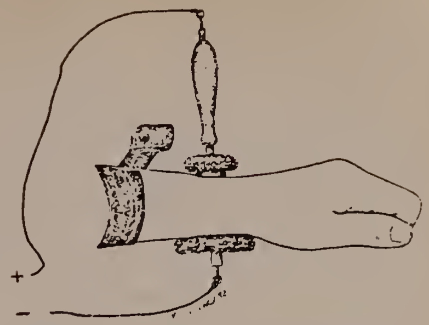


FIGURE 8. Anemic cataphoresis with Esmarch's bandage.

the advantages, both theoretical and practical, were evident in the desire to have a particular drug act on a superficial and local tissue.[14]

Physicians and physicists agreed that cataphoresis was an example of the phenomenon of osmosis, and that electric osmose was by no means thoroughly understood. The use of electric current combined with the "complexity arising from the effects of vitality, of modified nutrition, [and] of alterations in the contractility of the tissues," not only modified cataphoresis as understood by the physicist, but modified it from the standpoint of the therapeutist as well. "The fundamental causes of the phenomenon of electric endomose in non-living matter are still involved in dispute," commented Houston in 1892.[3] "The causes of the phenomenon of cataphoresis, or electric endosmose in living matter must, therefore, of necessity be involved in considerable obscurity." [3]

For the electro-physiologist and electro-therapeutist in the late 19th century, the principles established by previous experimenters had allowed the successful introduction of liquid medicaments into the body by electric endosmose. In such situations one electrode acted as the "therapeutic electrode," while the other became the "indifferent electrode." As for the cataphoric action itself, the distribution of the fluids into specific body areas depended on the condition of the skin, the strength of the current, and the accompanying electrolytic decomposition.[3]

Physicians at the turn of the century employed two particular methods for introducing medicines by cataphoresis. In the first method, medicines were applied to block tin electrodes covered by large sponges that were placed on each side of the inflamed joint. In this treatment the applicator maintained a current of 15 to 20 milliamperes for approximately 20 minutes.[14] One of the drawbacks was the difficulty in regulating and measuring the amount of drug that diffused through the skin. Waite and Bartlett[13,16] attempted to resolve this by replacing the sponges covering the electrodes with an electrode disk in which the metal surface was overlaid with a thin disk of platinum edged by a narrow rim of soft rubber. They then took a piece of tissue paper that was cut to fit the platinum disk within the rubber rim and they wet it with an exact amount of drug. They applied this to the skin, maintaining the current until the medicated disk dried. Physicians reasoned that specific doses of chloroform, methyl chloride, ether, cocaine, hellebore, iodide of potassium, corrosive sublimate, aconitine, and other drugs could be accurately and effectively applied to the body. According to Peterson, medicated cataphoretic disks could be prepared in advance by pharmacists. A physician had only to add several measured drops of water to the disk in order to ready the drug for administering by electricity.[13,16]

In the second method, Morton, a specialist in diseases of the mind and nervous system and in electro-therapeutics at the New York Post Graduate Medical School and Hospital, introduced a plaster made from gelatin, carbon, and medicine mixed in a measured dosage. When moistened, the plaster could be applied to the desired point and attached to a positive electrode. Like the tissue disk, medicated plasters were available from New York pharmacists (Fig 6).[4]

A variation of the cataphoric method involved an application of the old cupping-glass principle combined with the vacuum electrode (Fig 7). Produced by E. Machlett and Son of New York City, the vacuum electrode proved highly popular with John C. Lester, MD:

> A cotton tipped applicator is saturated with ninety-five percent alcohol. This is lighted with an ordinary alcohol lamp and immediately introduced into the cupping-glass attachment and repeatedly swept around the inside of the cupping-glass. In this manner sufficient alcohol is thrown off on the inside of the glass to continue burning until the electrode can be pressed upon the part to be treated. . . . After the electrode has been attached this way, any medication desirable can be introduced through the tube by turning the stop-cock and allowing it to flow down from a receptacle at the top. . . . In the manner described, I have employed such drugs as iodine, cocaine, holocane, and solutions of menthol.[17]

In order to maintain the local effect of cataphoresis and to prevent the drug from finding its way into the blood stream, physicians Peterson and Morton resorted to what they called "anemic cataphoresis," which meant creating a stasis of blood in the treated part by using an Esmarch bandage or rubber ring to ensure that the drug acted *in loco dolenti*. Both physicians preferred anemic cataphoresis in treating gouty and rheumatic joints—the wrist, elbow, ankle and knee being the most easily treated. They believed the anemic treatment to be far more effective than existing constitutional methods or local treatments that used actual cautery or blisters (Fig 8).[4,8,14]

In instances where physicians desired local anesthesia instead of general anesthesia, or where localized pains warranted the use of anodynes, cataphoresis seemed both a wise and a useful treatment. The cataphoric use of cocaine became popular for minor surgical operations such as the opening of abscesses, furuncles, carbuncles, and felons; the removal of sebaceous cysts, lipomas and moles; and in instances of severe localized pain from muscular spasms.[2,18] Morton claimed success with cocaine anesthesia as well as with the use of tincture of iodine over the spinal cervical centers to arrest progressive muscular atrophy.[14]

An alternative form of cataphoresis, known as the electric bath, involved immersion. The physician placed a positive electrode in the bath while attaching the negative electrode to a part of the body such as the sternum. Occasionally physicians enhanced the treatment with massage "to encourage the absorption of exudates." [19,20] For Begg[8] and Gibson,[8] this latter form of cataphoresis was most useful in treating subacute and chronic gout and almost all forms of arthritis. In preparing the treatment, both Begg and Gibson used a bath of carbonate, chloride, and bromide. Morton[14] devised a variation of the electric bath method using a cataphoric mouth electrode; medicines held in the mouth were thought to enter the circulation by way of mucous membranes, thus avoiding stomach fluids. A rectal electrode performed a similar function.

At the International Congress held in Berlin in 1890, Bayles read a communication from Thomas A. Edison, the inventor, on the successful use of the cataphoric electric bath in treating several cases of gout. In subsequent experiments, Kennelly, the chief electrician at the Edison Laboratories, expanded on Edison's work by devising a more systemic medicated electric bath. Vergnes, another exponent of the electric bath, introduced medicinal substances such as phosphate of iron in a solution with nitric acid and quinine into the body. Gartner and Ehrmann treated syphilis using a medicated bath of corrosive sublimate. By reversing the current of his "electrochemical bath," Royal claimed to have successfully treated lead and mercury poisoning by cataphorically removing the metals from the body.[1,6,14,16]

DENTAL CATAPHORESIS

In a paper before the Academy of Medicine of Cincinnati in 1896, Buxbaum, a dentist, reported success in anesthetizing dental pulps according to experiments he had earlier observed in New York.[21] By applying cocaine to the teeth using cataphoresis, he found a local rather than a systemic effect. "No dentist of scruple," he remarked, "can give pain without in a measure feeling it himself, and after a year of close application his nervous system will begin to show signs of weakening." As a result of his success with the cataphoric method, patients that had once entered his office "with a fear of that torturing pain so soon to follow," now greeted him with smiling faces "having full confidence in that wonderful power and drug which will carry them through an operation as painlessly as chloroform or ether will under the surgeon's knife." [21]

In a discussion of the value of cataphoresis at a meeting of the Academy of Stomatology of Philadelphia in 1902, Woodward recounted using cataphoresis on 346 individuals during the period from 1896 to 1902.[22] Not including the time required to place the rubber dam and adjust the electrode, the actual application of current took 15 minutes. "I have nothing which will produce so deep an impression upon the dentine," he noted, "as cocaine cataphorically applied." Woodward noted variations in response time to the electrical current extending in some cases from a few minutes to more than 40 minutes.

Whereas some dentists preferred to place one of the cathodes on the back of the patient's hand, others preferred to attach it to the cheek. But as one dentist recalled, the results were not always successful. "I have had blisters on the back of the hand from cataphoresis and the scars did not disappear for months," remarked Roberts in 1902.[22] "For that reason I would apply the cathode only to the palm of the hand, where the skin is better able to resist burning; also, the sponge should be kept well wet."

Dentists who employed cataphoresis in their practice purchased cataphoric outfits that used 30 primary cells producing about 40 volts. However, not all dentists were convinced of its efficacy. "I commenced the treatment with great hope and confidence," noted Rodolph in 1897, "but I have since come to the conclusion that [the successes] were greatly due to the fact that I operated first upon patients who had reached about thirty years of age and upward." [23] He concluded that age, along with the location of the cavity, affected the success of the treatment. Children were as discomforted by the pain produced by the current as by the drill. Furthermore, the child "was required to remain in the chair perfectly quiet, gagged with that despised rubber-dam, mouth wide open, and from ten to thirty minutes longer than would be required by the usual method." The need to insulate adjoining fillings, the time needed to prepare the tooth for application, and the unequal results seemed to reduce the effectiveness and overall applicability of the treatment.

DISPUTED THEORIES

Physicians and dentists remained unclear as to the laws that governed medical cataphoresis. Whether the medicament was transported into the tissue as the result of a simple mechanical transportation, an incidental or fundamental part of electrolysis, electric osmosis, or perhaps a compound phenomenon involving two or more actions remained unresolved. By the end of the century, the accepted theories had been reduced to three. The first theory suggested that any effect produced in passing an electric current through a medicament, as in cataphoresis, was produced by the current rather than by the medicament. In this regard, Du Bois-Reymond in 1843 discovered that passing a constant galvanic current through a nerve produced what he identified as electrotonus: "the resulting nerve-current was either increased or diminished, according to the direction of the constant current." [24] In 1859, Pfluger showed that there was a definite change in the irritability of the nerve that resulted from the passage of a constant galvanic current.[24] Dentists who employed electric current in this manner referred to "short-circuiting the nerve" as a means of producing a condition of anesthesia. In effect, no medicament was needed as the results were solely from the electric current.[24]

The second theory, that of electric osmose, held that the medicament applied under the electrode was the true agent of anesthesia, being carried to the treated part by a physical force ("just as a stream of water carries sediment with it, so the electric current carries the ingredients in solution with it through the solvent and through the tissue"). This remained the predominant theory among the advocates of medical cataphoresis, the most outspoken being physicians Morton, Peterson, and Phillips in the United States. As for the laws governing electric osmose, Price believed that when an electric current was applied to tissues "with an interposing layer of some medicament under the positive pole," the medicament was carried into the tissue by the "physical force possessed by the current." By itself, without the support of current, osmosis could not produce sufficient movement of drug through cellular tissue. Moreover, not all substances could pass through cellular tissue and porous partitions. Price concluded from his own experiments that osmotic pressure alone was not forceful enough to explain the transmission of cocaine through dentine into the pulp.[24]

Finally, a third theory suggested that passing an electric current through a medicament resulted in electrolysis and the migration of ions to the electrodes, rather than the transmission of matter. "I do not believe the medicine was in any case carried in as the original chemical species," noted Price, "but was changed by electrolysis; and further, with the conditions under which we use cataphoresis, I believe the forces upon which we are dependent are the dissociation of the molecules and the increased energy of these dissociated products." [1,24]

THE DEMISE OF CATAPHORESIS

Despite its popularity among certain members of the profession, medical cataphoresis was never broadly accepted or used. Articles on the subject remained limited to a few journals, principally those that advocated electrical experimentation. Although advocates were gratified by their successes, critics remained disappointed in the uncertainty of the method. One major reason for its lack of popularity lay in the confusion surrounding the nature of the process. That, coupled with the cost of the equipment, the disadvantage of preparation time, and the disappointing results meant that few were willing to give it a fair trial. Finally, the popularity of the syringe ensured the early demise of cataphoric medication. No method of intradermic medication proved as effective, as inexpensive, and as immediate in its results as the hypodermic syringe. Although there continued to be physicians and dentists who advocated electric osmose, it was relegated to the fringe of therapeutics. Only for a few short years did it tempt the medical profession with the breadth of its applicability.

REFERENCES

1. Haynes CM: *Elementary Principles of Electro-Therapeutics for the Use of Physicians and Students.* Chicago, McIntosh Galvanic and Faradic Publishing Co, 1884, pp 36, 46.
2. Monell SH: *The Treatment of Disease by Electric Currents.* New York, William Beverley Harison, 1897, pp 433–452, 561–583, 728–738.
3. Houston EJ: Cataphoresis. *Transactions.* American Electro-Therapeutic Association. 1982; 2:51–62.
4. Morton, WJ: Note on a new method of cataphoresis. *NY Med J* 1891; 53:473–475.
5. Anon: Remarks on the phenomena of galvanism. *Ann Philosophy* 1815; 5:430–439.
6. Althaus J: *A Treatise on Medical Electricity.* Philadelphia, Lindsay and Blakiston, 1873, pp 45–46.
7. Begg C: Cataphoresis in the treatment of gout, rheumatism, and rheumatoid arthritis. *Edinburgh Med J* 1902; 12:45–49.
8. Jack L: The value of cataphoresis. *International Dental J* 1902; 23:781–784.
9. Hebert T: The cataphoresis of drugs as a remedial and pallative therapeutical measure—a few cases. *Transactions.* Louisiana State Medical Society. 1894; 15:335–345.
10. Richardson BW: On voltaic narcotism. *Medical Times and Gazette.* 1859; 39:156, 203, 647.
11. Peterson F: *Cataphoresis, Anodal Diffusion, Electrical Osmosis, or Voltaic Narcotism.* Philadelphia, F.A. Davis Co, 1894, C-3, C-4.
12. Peterson F: Electrical cataphoresis as a therapeutic measure. *NY Med J* 1889; 49:449–453.
13. Peterson FL: Note on a new system of exact dosage in the cataphoretic use of drugs. *NY Med J* 1890; 52:543.
14. Morton WJ: Electric cataphoresis; its uses in general medicine. *Transactions.* American Electro-Therapeutic Association. 1892; 2:62–73.
15. Reynolds: Local anaesthesia of the skin. *JAMA* 1887; 9:235.
16. Steavenson WE, Jones HL: *Medical Electricity: A Practical Handbook for Students and Practitioners.* Philadelphia, P. Blakistan & Co, 1892, pp 220–221.
17. Lester JC: Improved vacuum electrode for cataphoresis. *Medical Record* 1906; 49:735–736.
18. Gill JC: Cataphoresis. *J Advanced Therapeutics* 1904; 22:347–349.
19. Jacoby GW: *A System of Physiologic Therapeutics,* vol 1. Philadelphia, P. Blakiston's Son & Co, 1904, pp 202–204.
20. Beard GM, Rockwell AD: *On the Medical and Surgical Uses of Electricity.* New York, William Wood & Co, 1891, pp 408–411.
21. Buxbaum AIF: Cataphoresis. *Cincinnati Lancet-Clinic* 1896; 37:539–541.
22. Discussion of March 25, 1902 meeting of the Academy of Stomatology, reported by Inglis OE. *International Dental J* 1902; 23:833–840.
23. Rodolph GW: Some observations on cataphoresis. *Pacific Stomatolotigal Gazette* 1897; 5:241–243.
24. Price WA: The phenomena of cataphoresis. *Dental Cosmos* 1897; 39:703–718.

FROM THE LIBRARY

THE PRESENT STATE OF ELECTRO-THERAPEUTICS

. . . it is customary to place the feet of the patient upon a metallic plate to which the negative pole is attached, while the positive pole is applied to the surface, generally. . . . For a number of years it has been customary with us to substitute our hand for the ordinary sponge electrode, especially when operating about the neck and head. No artificial electrode that can be devised equals the human hand in flexibility and power of adaptation to the inequality of surface in the face and neck; and one needs only to accustom himself to the passage of the current through his hands and fingers to become practically aware of the immense advantage given him in treating all such sensitive parts. . . .

Soon after my first essays to use the faradic current through my own person, I observed an increased size and hardness of the muscles of the arm. Muscular development gradually increased until within a year the circumference of the arm and fore-arm was markedly greater. An interesting fact was observed in the more decided development of the left arm.

This is explained by the more powerful influence of the negative electricity as I invariably grasped the positive pole with my right hand.

A. D. ROCKWELL, MD
(*Trans Med Soc State of NY,* 1872, pp 148–149)

Sarcoidosis of the breast

MARY ELLEN REITZ, MD, IRVING SEIDMAN, MD, DANIEL F. ROSES, MD

Sarcoidosis is a systemic disease of uncertain etiology and protean clinical manifestations. The granulomatous lesions of sarcoidosis usually occur in the lymph nodes, lungs, and skin, but they have been described in virtually every organ.[1-11] Sarcoidosis in breast tissue, however, is very rare. In a review of the literature, we read of 11 cases of sarcoidosis involving the breast; several of these cases were presumed to be sarcoidosis by clinical presentation, without proof by microscopic examination. This report describes a patient with a breast mass that was thought to be carcinoma on clinical examination, but was shown to be sarcoidosis on microscopic examination of the biopsy specimen.

CASE REPORT

A 39-year-old black woman sought treatment for a mass in the left breast that she first noted three weeks earlier. She stated that once before she had had a mass in the left breast, but that the mass had disappeared. The patient had had sarcoidosis 10 years earlier that occurred at the time of her first pregnancy. At that time she demonstrated bilateral ankle and knee arthritis, a widened mediastinum on chest films, and non-tender, non-pruritic cutaneous lesions on her right arm that were diagnosed as sarcoidosis following a biopsy. The arthritis was controlled with aspirin and subsided after her pregnancy. The patient remained without complaints until her second pregnancy two years later, when she had a flare-up of the arthritis and skin lesions; this was controlled with prednisone. Biopsy of an enlarged right axillary lymph node at that time again showed sarcoidosis. Chloroquine was used for three years following this pregnancy to control persistent cutaneous lesions. The patient was fine for several months following an elective decision to discontinue chloroquine when she was admitted to the hospital with streptococcal pharyngitis. A chest film at this time confirmed hilar lymphadenopathy, but results of pulmonary function tests were normal. Skin tests were negative for purified protein derivative and *Trichophyton*, with positive *Candida* and streptokinase-streptodornase findings. Laboratory values were remarkable for a negative antinuclear antibody test result, increased gamma globulin (2.61) on serum protein electrophoresis, and a 24-hour urine calcium of 382 mg. Other values were within normal limits.

The patient continued to receive no therapy for her sarcoidosis until two years later when she was seen for vesicular, mildly pruritic lesions of sarcoidosis on her torso, arms, neck, and face. When there was no response to other drugs, she underwent a liver biopsy and methotrexate therapy was started with good control of the skin lesions. A chest film at the time of the biopsy showed new, slight prominence of bilateral interstitial markings.

A repeat liver biopsy for evaluation of a 3-cm hepatomegaly and a lactate dehydrogenase value of 241 was performed two years later. Chest films revealed new, nodular infiltrates in the right apex. The liver biopsy showed sarcoid granulomas in the liver. No further studies were carried out for sarcoidosis. It was at this time that the mass in the left breast was discovered, and she was referred for a breast biopsy.

Physical examination revealed diffuse nodules in both breasts. There was a firm mass, 1 cm in diameter, in the left breast at the 3 to 4 o'clock position in the periareolar region. Examination of the left axilla disclosed no abnormalities. The right axilla was normal to palpation. Mammography demonstrated atrophic glandular elements, with no findings suggestive of malignancy. An excisional biopsy was performed.

The excised mass was a 1-cm, hard, tan-grey nodule with irregular borders that grossly suggested carcinoma. Microscopic examination of the mass showed ducts and ductules surrounded by confluent granulomas with numerous multinucleate giant cells (Fig 1). Stains for acid-fast bacilli were negative. A diagnosis of sarcoidosis was made. There was no necrosis throughout most of the specimen; however, rare granulomas contained central, acellular areas of fibrillar, eosinophilic material with scattered lymphocytes. Smaller granulomas were found clustered around ducts in portions of the specimen that had appeared grossly to be uninvolved parenchyma.

DISCUSSION

The etiology of sarcoidosis is not known, but impaired cellular immunity appears to play a significant role in its pathogenesis; skin tests are often negative after challenge for a number of bacterial, fungal, and viral antigens, and T lymphocytes usually have a diminished mitotic response to phytohemagglutinin. On the other hand, B-cell activity is enhanced, and there is often an increase in the level of circulating immunoglobulins. Hypercalcemia and hypercalciuria are common, and the serum angiotensin-converting enzyme level is elevated in about 60% of the patients with active disease.

The increase of both occult and clinically evident sarcoidosis involving a breast is rare. The medical literature contains descriptions of about 11 cases, some of which were diagnosed on clinical grounds and not confirmed by biopsy. Two series totalling 195 patients with sarcoidosis included three with mammary involvement, but in each instance the diagnosis was made without confirmation by biopsy. The clinical presen-

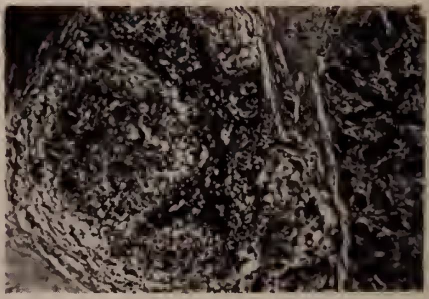


FIGURE 1. Non-caseating granulomas with giant cells in breast parenchyma. (hematoxylin and eosin stain, original magnification ×100)

From the Departments of Pathology (Drs Reitz and Seidman) and Surgery (Dr Roses), New York University Medical Center, New York.

Address correspondence to Dr Seidman, Associate Professor, Department of Pathology, New York University Medical Center, 550 First Avenue, New York, NY 10016.

tations in these three cases varied from multiple mobile masses, which regressed spontaneously, to a hard, immobile lesion that suggested carcinoma.[1,2]

Scott[3] reported the first case of sarcoidosis of the breast in 1938. The patient had a mobile, well-defined mammary mass suggestive of fibrocystic disease, as well as enlarged axillary and cervical lymph nodes suggestive of carcinoma. Biopsy of both the breast mass and a lymph node revealed sarcoidosis. The breast, therefore, was the site of the first clinical manifestation of the underlying disease.[3,4] Indeed, in several of the other reported cases, the breast was also the first clinical site of sarcoidosis.[5-10]

In two other reports of biopsy-proved mammary sarcoidosis, the clinical impression was that of a benign lesion, either fibrocystic disease[6] or fibroadenoma.[10] Several previously reported cases, however, were clinically suspicious for carcinoma. In one instance the breast contained a large, fixed mass with changes in the overlying skin. Enlarged axillary lymph nodes were present, and an "obvious" clinical diagnosis of carcinoma was made. Needle aspiration, however, revealed a granuloma, and excisional biopsy confirmed the diagnosis of sarcoidosis.[5] Another woman underwent a radical mastectomy for an unbiopsied 5 × 3-cm mass in the breast with overlying skin changes and enlarged axillary lymph nodes. The microscopic examination showed only sarcoidosis.[9]

The repeated finding of axillary lymphadenopathy in these cases, and in other patients with sarcoidosis, raises the possibility of carcinoma of the breast, despite the absence of a clinically suspicious breast mass.[3,4,5,7-9] However, the history of documented, generalized sarcoidosis does not preclude the need to examine significant breast nodules; in one patient with generalized lymphadenopathy due to sarcoidosis, biopsy of a mammary mass showed carcinoma. Lymph node biopsy specimens and the axillary lymph nodes from the mastectomy specimen showed only sarcoidosis. In addition to the adenocarcinoma, the breast also showed diffuse, microscopic involvement by sarcoidosis.[8]

Although rare, sarcoidosis should be included in the differential diagnosis of a mass in the breast, even in a patient without a history of sarcoidosis. Clinically, sarcoidosis can simulate more common breast diseases, ranging from fibrocystic disease to carcinoma. Especially worrisome is the frequent lymphadenopathy in patients with sarcoidosis. Mammography, used in one patient in the literature[6] as well as in this patient, showed no evidence of malignancy, suggesting that this technique may have diagnostic value in cases of sarcoidosis. Needle aspiration proved its diagnostic use in another case.[5] Certainly, in a patient with known sarcoidosis, biopsy remains essential to preclude carcinoma of the breast.

REFERENCES

1. Longcope WT, Freiman DG: A study of sarcoidosis. *Medicine* 1952; 31:1-132.
2. Reisner D: Boeck's sarcoid and systemic sarcoidosis. *Am Rev Tuberc* 1944; 49:289-307, 437-462.
3. Scott RB: The sarcoidosis of Boeck. *Br Med J* 1938;2:777-781.
4. Stallard HB, Tait CBV: Boeck's sarcoidosis. *Lancet* 1939; 1:440-442.
5. Bodo M, Döbrössy L, Sugár J: Boeck's sarcoidosis of the breast: cytologic findings with aspiration biopsy cytology. A case clinically mimicking carcinoma. *Acta Cytol* 1978; 22:1-2.
6. Bonecchi L, Nanni MR: Sarcoide di Boeck della mammella. *Radiol Med* 1981; 67:656-657.
7. Dalmark G: Lymphogranulomatose benigne. *Acta Chir Scand* 1942; 86:168-178.
8. Haagensen CD: *Diseases of the Breast*, 2nd ed. Philadelphia, WB Saunders Co, 1971, pp 339-340.
9. Masaaki S, Shinhachiro N, Kazuro I, Takateru I: A case of breast sarcoid. *Arch Jap Chir* 1979; 48: 404-410.
10. Scadding JG: *Sarcoidosis*. London, Eyre and Spottiswoode, 1967, pp 335-336.
11. Rigden B: Sarcoid lesion in breast after probable sarcoidosis in lung. *Br Med J* 1978; 2:1533-1534.

See also pp 240-241.

Intrathymic parathyroid adenoma in a child

RICHARD H. K. WU, MD, RAKESH SHREEDHAR, MD, B. NARAYAN PAI, MD, JULIO LEVY, MD, PAUL H. GERST, MD

Primary hyperparathyroidism in childhood is a relatively rare finding. Between the first case report by Pemberton and Geddie[1] in 1930, and the review by Girard et al[2] in 1982, a total of 86 childhood cases of primary hyperparathyroidism have been described. Allo et al[3] added another 26 cases in 1982. We describe a 10-year-old patient with hyperparathyroidism in whom an intrathymic parathyroid adenoma was resected.

CASE REPORT

A 10-year-old boy was seen at a local health care facility because of symptoms of an upper respiratory tract infection, malaise, and pain in the mandible, all of recent onset. A history of occasional polyuria was noted, but this finding was not persistent. There was no history of arthritis, pathologic fractures, bone pain, or nephropathy. He took no medications except for a daily dose of cod liver oil. There was no family history of parathyroid disease or other endocrinopathy. The mandibular pain was subsequently attributed to a dental abscess, which was treated by his dentist. A serum chemistry profile revealed elevated serum calcium and low phosphorus levels, and the patient was referred to the pediatric endocrinology service of the Bronx-Lebanon Hospital Center.

When seen by us, the patient was alert with normal orientation. His blood pressure was 112/70 mm Hg; heart rate, 80/min; weight, 44.5 kg; and height, 135 cm. He had no café au lait spots on the skin and no joint pain or limitation. He had Tanner stage 2 pubic hair development.[4] The rest of the physical examination, including neurologic studies, was within normal limits.

A blood chemistry analysis revealed a serum calcium level of 11.2 mg/dL (normal, 8.5 to 10.6 mg/dL) and a phosphorous level of 2.7 mg/dL (normal, 3.5 to 6.0 mg/dL). Subsequent calcium levels were 12.4 to 13.0 mg/dL. The ionized serum calcium value was 6.12 mg/dL (normal, 4.2 to 4.7 mg/dL), and the serum magnesium level was 1.8 mg/dL (normal, 1.6 to 2.6 mg/dL). Parathyroid hormone level determined by N-terminal immunoassay was 54 pg/mL (Ni-

---

From the Departments of Pediatrics (Dr Wu), Surgery (Drs Shreedhar, Pai, and Gerst), and Pathology (Dr Levy), Bronx-Lebanon Hospital Center and Albert Einstein College of Medicine, Bronx.

Address correspondence to Dr Wu, Associate Professor, Department of Pediatrics, Bronx-Lebanon Hospital Center, Bronx, NY 10457.

chols Laboratories, Los Angeles, CA) (normal, 18 to 40 pg/mL). The C-terminal parathyroid hormone level was 111 μLEq/mL (Smith-Kline, Lake Success, NY and Nichols Laboratories, Los Angeles, CA) (normal, 40 to 100 μLEq/mL).

Because the boy had taken cod liver oil, the level of 25-hydroxy vitamin D was measured and was found to be 15 ng/mL (Smith-Kline, Lake Success, NY) (normal, 5 to 65 ng/mL). Radiographic studies of the bones showed no demineralization.

The calcium to creatinine clearance ratio was 0.022, and the urinary calcium excretion over a 24-hour period was 5.48 mg/kg body weight. The clearance ratio of 0.022 is twice the normal value established by Marx et al.[5] Values below 0.01 would suggest familial hypercalciuric hypercalcemia. The calcium excreted in the urine was also greater than the normal value of 4 mg/kg body weight.[6] Four family members—the mother and three brothers—were screened for hypercalcemia, and all had serum calcium levels within normal limits. These data would suggest that the patient did not have benign familial hypercalciuric hypercalcemia.

Since all of the laboratory data pointed toward primary hyperparathyroidism, the patient underwent neck exploration. During surgery, two parathyroid glands (the two upper glands) were found in their usual position behind the thyroid gland. The lower glands could not be identified behind or near the thyroid. However, two thymic remnants were located below the thyroid (one on each side of the neck) extending into the mediastinum. The right thymic pole was resected and was found to contain a normal parathyroid gland. The left thymic pole was found to contain a parathyroid adenoma (Figs 1, 2).

The patient tolerated the procedure well. His immediate postoperative calcium level was 8.4 mg/dL, without symptoms of hypocalcemia. Four days after surgery, the N-terminal parathyroid hormone level was 18 pg/mL, (normal, 11 to 24 pg/mL), and the C-terminal parathyroid hormone level was 74 μLEq/mL (normal, 40 to 100 μLEq/mL). Four months after surgery he was asymptomatic, with a serum calcium level of 9.6 mg/dL.

Discussion

Primary hyperparathyroidism in children tends to be recognized because of symptoms attributable to hypercalcemia, or because of bone or renal disease. In the past, most patients have sought treatment for bone pain or fractures, or manifestations of renal disease. Weakness, anorexia, vomiting or constipation also are noted.[3,7] Joint involvement has occasionally been prominent, while among other patients abdominal pain was the only symptom.[8] Abdominal pain has been attributed to acute pancreatitis, a recognized complication of hyperparathyroidism. Three patients had an associated duodenal ulcer.[7]

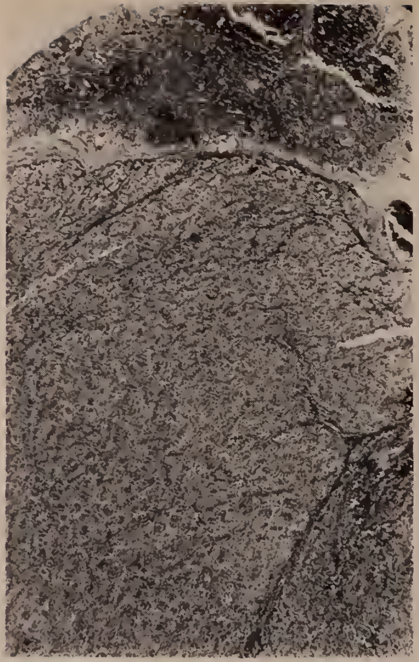


FIGURE 1. A low-power photomicrographic section of the parathyroid adenoma, with the thymus at the upper portion of the picture (original magnification ×56).

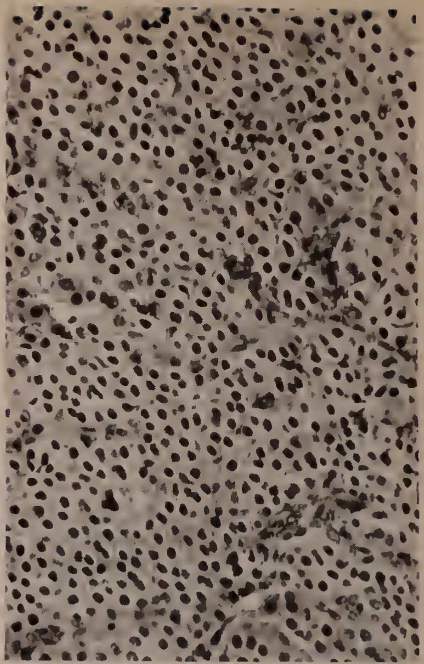


FIGURE 2. Photomicrographic magnification of the adenoma. Note presence of clear cells. (original magnification ×350)

The diagnosis usually is suspected from the clinical features and the finding of persistent hypercalcemia. The plasma inorganic phosphate concentration is characteristically low. Familial hyperparathyroidism and familial benign hypocalciuric hypercalcemia should be excluded by screening other family members. If the parathyroid hormone level is clearly elevated, the diagnosis is confirmed. The cortisone suppression test has been found to be unreliable, as have tests of calcium and phosphate metabolism such as tubular reabsorption of calcium and phosphate excretion in diagnosing primary hyperparathyroidism. This patient had a modestly elevated parathyroid hormone level, high urinary calcium excretion, and a higher than normal calcium to creatinine clearance ratio. Together, these data would rule against hypocalciuric hypercalcemia. The absence of elevated plasma calcium levels in the family supports the diagnosis of primary hyperparathyroidism.

Selective venous sampling has been used to locate parathyroid adenomas in a large series of adults.[9] Its use in children is more limited, although successful localization has been reported.[3] High resolution ultrasonography has also been used to locate parathyroid tumors, and in one series it was used successfully to distinguish adenomas from parathyroid enlargement found in benign familial hypocalciuric hypercalcemia.[10]

Intrathymic adenoma is a rare condition. Although Wang[11] reported, in a review of 645 normal parathyroid glands in 160 cadavers, that 39% of the parathyroid glands were located in the thymic tongue, Girard et al[2] found only two (3.6%) adenomas in the thymus, on review of 55 patients with parathyroid tumors. The difference in these two observations needs to be clarified.

In children, as in adults, the disease is more frequently caused by solitary adenomas than by multi-glandular hyperplasia. The ratio of adenomas to hyperplasia reported by Mannix[12] in 1975 was five to one, which corresponds to the findings in adult patients. In general, hyperplasia is found in patients with a family history of hyperparathyroidism, while adenomas are the common finding in non-familial cases.

The prognosis after parathyroidectomy in children, as in adults, is good, and normocalcemia is the rule. Delayed diagnosis may lead to renal or neurologic damage; therefore, it is important that the diagnosis not be overlooked, and that the finding of even mild or asymptomatic hypercalcemia in children not be ignored.

Acknowledgment. Dorothy Zapakin and Olivia Mosby helped in the preparation of the manuscript, and Barbra Saeed, MD, referred the patient.

References

1. Pemberton J de J, Geddie KH: Hyperparathyroidism. *Ann Surg* 1930; 92:202-211.
2. Girard RM, Belanger A, Hazel B: Primary hyperparathyroidism in children. *Can J Surg* 1982; 25:11-13, 32.
3. Allo M, Thompson NW, Harness JK, Nishiyama RH: Primary hyperparathyroidism in children, adolescents, and young adults. *World J Surg* 1982; 6:771-776.
4. Marshall WA, Tanner JM: Variations in the pattern of pubertal changes in boys. *Arch Dis Child* 1970; 45:13-23.
5. Marx SJ, Spiegel AM, Brown EM, et al: Divalent cation metabolism. Familial hypocalciuric hypercalcemia versus typical primary hyperparathyroidism. *Am J Med* 1978; 65:235-242.
6. Greenhill A, Gruskin AB: Laboratory evaluation of renal function. in, *Pediatric Nephrology.* Philadelphia, Saunders, 1976, pp 678.
7. Bjernulf A, Hall K, Sjorgren L, Werner I: Primary hyperparathyroidism in children. Brief review of the literature and a case report. *Acta Paediat Scand* 1970; 59:249-258.
8. Frier BM, Marrian VJ: Uncomplicated hyperparathyroidism. *Arch Dis Child* 1974; 49:808-810.
9. Aurbach GD: Hyperparathyroidism: recent studies. *Ann Intern Med* 1973; 79:566-581.
10. Law WM, James EM, Charboneau JW, Purnell DC, Heath H: High-resolution parathyroid ultrasonography in familial benign hypercalcemia (familial hypocalciuric hypercalcemia). *Mayo Clin Proc* 1984; 59:153-155.
11. Wang CA: The anatomical basis of parathyroid surgery. *Ann Surg* 1976; 183:271-275.
12. Mannix H: Primary hyperparathyroidism in children. *Am J Surg* 1975; 129:528-531.

See also p 237.

Amniotic fluid embolism

Margaret E. Bates, MD, Uma L. Verma, MD, Nergesh A. Tejani, MD, Daniel J. DiTroia, MD

Amniotic fluid embolism, which is associated with cardiovascular collapse and an 80% to 90% mortality rate is difficult to diagnosis and to treat.[1] A definitive diagnosis is usually made at autopsy with the demonstration of amniotic fluid debris in the pulmonary circulation.[1-3] With few exceptions, the diagnosis among survivors is based purely on clinical evidence. A rapid and reliable means of diagnosis would improve survival rates by allowing prompt and aggressive therapy. We describe a patient with this condition who survived.

Case Report

A 25-year-old primigravida woman was admitted to the hospital in active labor. She was in the 42nd week of gestation and had had an uncomplicated antepartum course. Physical examination disclosed an intrauterine pregnancy consistent with dates and a cephalic presentation. The cervix was 6 cm dilated and 100% effaced; the membranes were intact. The presenting part of the fetus was at 0 station. One hour later, the cervix measured 7 cm, the membranes were artificially ruptured, and continuous monitoring with a fetal scalp electrode and an internal uterine catheter was started. Uterine contractions occurred every two to three minutes; they lasted one minute and were of 40 to 50 mm in intensity. Forty minutes later, with the cervix at full dilation and the vertex at +2 station, there was a sudden and persistent fetal bradycardia. The patient was moved to the delivery room, where she complained of dizziness, became tachypneic and cyanotic and suffered cardiopulmonary arrest. Resuscitation efforts with endotracheal intubation and positive pressure ventilation were initiated immediately, and a low forceps delivery of a female infant weighing 2.7 kg was performed.

The Apgar score was 9 and 10 at one and five minutes, respectively. Following the spontaneous expulsion of the placenta, repair of a fourth degree perineal laceration was performed. The patient lost profuse amounts (1,500-2,000 mL) of unclotted blood through the vagina. Exploration of the uterus did not reveal any retained placenta or membrane. Dopamine infusion therapy was started because of prolonged hypotension. An electrocardiogram taken immediately after resuscitation showed sinus tachycardia with ST elevations in leads V_1 to V_4. The patient's hemoglobin level was 13.3 g/dL; platelet count, 21,000/mm^3; prothrombin time, 24.9 sec (control, 19.4 sec); partial thromboplastin time, >110 sec (control, 33.1 sec); and fibrinogen, <70 mg/dL. Arterial blood gases were within normal limits. A central venous pressure (CVP) line was inserted, with an initial reading of 7 cm H_2O. Blood drawn from the right atrium via the CVP catheter and stained with 1% nile blue showed clumps of fetal squamae (Fig 1) and lanugo hair.

After the patient's condition stabilized, she was transferred to the intensive care unit where she was placed on a ventilator with positive and expiratory pressure of −5 cm of water and 60% O_2. A Swan-Ganz catheter was inserted through the CVP line; the initial pulmonary artery pressure was 34/19 mm Hg (normal, 15-25/8-15 mm Hg), with a pulmonary capillary wedge pressure of 14 mm Hg (normal, 6-12 mm Hg). A chest film disclosed no abnormalities. Measurements of hemodynamic functioning during the next 60 hours are shown in Figure 2. Two hours after the cardiopulmonary arrest, the patient had a hematocrit of 26%: fibrinogen, <10 mg/dL; prothrombin time, 28.9 sec (control, 10.1 sec); partial thromboplastin time, 110 sec (control, 33.1 sec); and platelet count, 21,000/mm^3. The subsequent hematologic data gathered during the next 60 hours are shown in Figure 3.

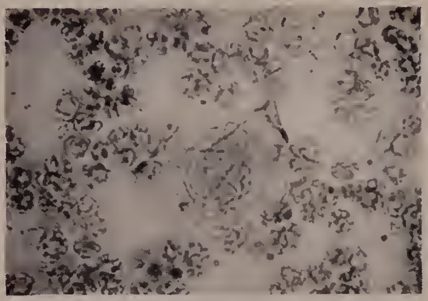


FIGURE 1. Blood from the maternal pulmonary circulation shows clumps of fetal squamae.

The patient continued to have moderate to heavy vaginal bleeding for three hours after delivery in spite of replacement therapy with platelets, cryoprecipitate, fresh frozen plasma, and red blood cells. Since a continuing source of coagulopathy was postulated, heparin infusion with 5,000 units intravenous bolus followed by an infusion of 1,000 units/hr was started. Measurements of clotting ability began to show some improvement, and the vaginal bleeding decreased. Six hours later, the vaginal bleeding had stopped, but there was profuse rectal bleeding. A pelvic examination failed to re-

---

From the Perinatal Service, Department of Obstetrics and Gynecology, Nassau County Medical Center, East Meadow, NY, and the Health Sciences Center, State University of New York at Stony Brook.

Address correspondence to Dr Verma, Chief, Service of Perinatal Medicine, Nassau County Medical Center, 2201 Hempstead Turnpike, East Meadow, NY 11554.

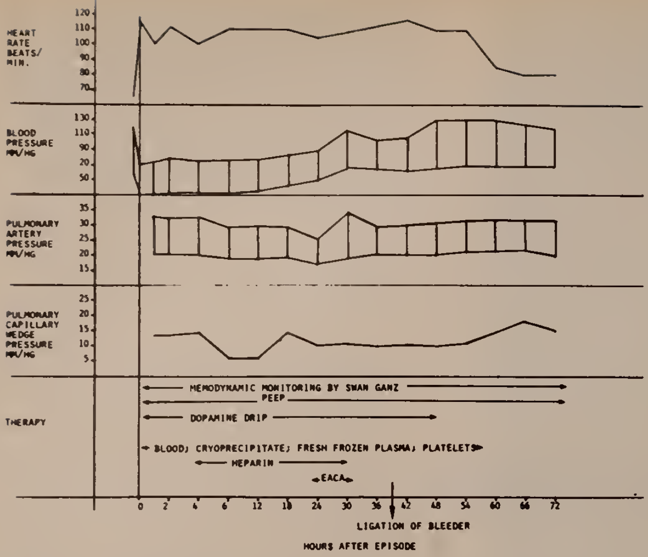


FIGURE 2. Hemodynamic parameters seen during the first 72 hours after the amniotic fluid embolism.

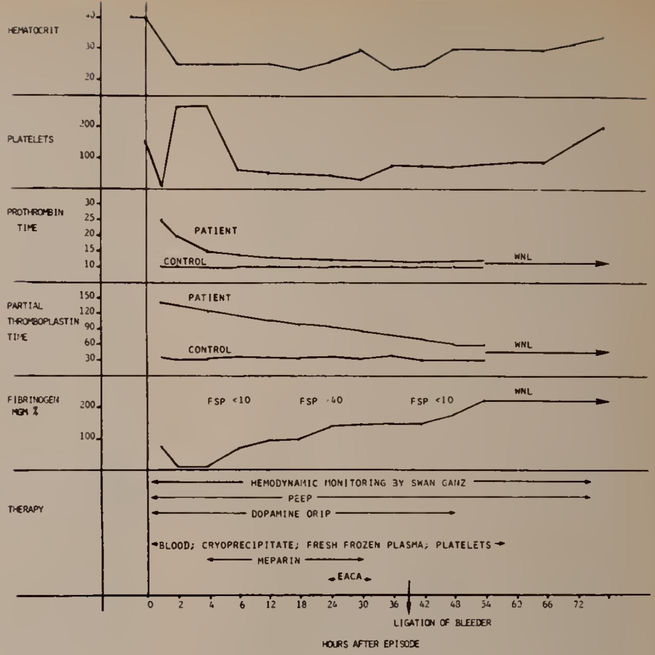


FIGURE 3. Hematological data obtained during the first 72 hours after the AFE.

veal any abnormality. Sigmoidoscopy was performed and the colon was found to be distended with blood, but no source of active bleeding could be identified. Since the fibrin split products were greater than 40 ug/min, aminocaproic acid was given for six hours at 1 g/hr.

The patient continued to have intermittent bouts of rectal bleeding for approximately 36 hours after the initial episode. Since the bleeding was from the rectum and the coagulation parameters were within normal limits, the need for surgery was suspected. A mesenteric angiogram showed bleeding from the middle rectal artery. Exploration of the perineum under local anesthesia showed the vaginal repair to be intact, with an arterial bleeder beyond the apex of the tear that was bleeding into the rectum. Hemostasis was achieved and the perineal laceration was resutured. There was no further rectal or vaginal bleeding during her hospital stay.

The total duration of the cardiovascular collapse, disseminated intravascular coagulation (DIC), and subsequent rectal bleeding was 42 hours, during which time the patient received 47 units of blood, 20 units of platelets, 20 units of fresh frozen plasma, 30 units of cryoprecipitate, and 10 units of 50% albumin. Dopamine infusion had been started to maintain blood pressure and could have been discontinued within 24 to 36 hours. The infusion was continued for an additional 12 to 24 hours to maintain and enhance renal perfusion. The endotracheal tube was removed 36 hours after bleeding was controlled. Since the electrocardiogram performed on postpartum days four and five showed a small sub-endocardial infarction (as seen by poor R wave prognosis, inverted T waves in leads V_1 through V_5, ST segment depression in leads III, AVF, V_5, and V_6, and a pyrophosphate scan showing discrete uptake pattern), the patient was observed for two days in the intensive care unit and then in the medical ward, and she was discharged from the hospital in good condition on the 15th day. She has since been seen three to four times in the medical clinic, and she has no cardiac or respiratory disability three months after the episode of amniotic fluid embolism.

DISCUSSION

The differential diagnosis of acute cardiopulmonary collapse during labor and delivery includes pulmonary embolism, eclampsia, aspiration pneumonitis, acute myocardial infarction, gram-negative sepsis, drug reactions, uterine rupture, and acute inversion of the uterus. When consumptive coagulopathy follows cardiopulmonary shock, the presumptive diagnosis is amniotic fluid embolism.[1,3] In this patient, the diagnosis was confirmed by the presence of fetal squamae and lanugo hair in blood obtained from the right side of the heart.

Gross and Benz[4] in 1947 first showed that blood aspirated from the right ventricle on postmortem examination could be stained to document amniotic fluid embolism in patients in whom autopsy was refused. The literature is uniform in emphasizing the rarity of survival following this condition.[1,3] In addition, the diagnosis among survivors, with few exceptions, is based on presumptive clinical evidence. Tuck[5] in 1972 diagnosed amniotic fluid embolism in a postpartum patient with respiratory distress by demonstrating the presence of fetal squamae in maternal sputum. However, this is a rare finding, and its reliability is questionable because of contamination of cells from the air passages.

The demonstration of the presence of amniotic fluid debris in the maternal pulmonary circulation was first reported by Resnik et al[6] in 1975 in a patient with a characteristic clinical course, cardiopulmonary collapse, and DIC. Schaerf and colleagues[7] and Masson et al[8] each reported similar cases in which, again, the diagnosis was confirmed by the presence of amniotic fluid debris in the maternal pulmonary circulation. The survival of these three patients and the patient described in this report emphasizes the importance of a rapid and accurate diagnosis.

It can be argued that amniotic fluid debris in maternal circulation and lungs may be a physiologic occurrence similar to the presence of trophoblastic tissue. In 1958, Sparr and Pritchard[9] injected labelled red blood cells into the amniotic sacs of volunteers and failed to detect any evidence of radioactivity in the maternal circulation; they concluded that amniotic fluid embolism is not a simple exaggeration of a physiologic phenomenon but is always pathologic. Roche and Norris,[10] showed that the presence of amniotic fluid in maternal lungs is presumptive evidence of this condition and is not seen in other patients.

The high mortality rate associated with amniotic fluid embolism has been attributed to cardiopulmonary collapse and DIC. Any efforts to decrease fatal outcomes must include prompt and aggressive hemodynamic support so as to maintain adequate tissue perfusion and oxygenation. Use of the Swan-Ganz catheter is indispensible for accurate hemodynamic support. DIC occurs in 40% to 50% of patients who survive the first hour following the embolism. In 12% of patients described by Morgan,[1] excessive bleeding was the first indication of this disorder. It is also conceivable that a percentage of patients who die of postpartum hemorrhage suffered an undiagnosed amniotic fluid embolism. The defibrination is due to the release of thromboplastic substances which activate intravascular clotting, deplete coagulation factors, and increase fibrin split products. The latter further accelerate the bleeding, and a vicious cycle is started. Heparin therapy has the advantage of controlling both the clotting and the fibrinolytic phases by inhibiting early stages of the coagulation cascade. Heparin has been used successfully in several instances of documented and presumptive cases of amniotic fluid embolism without complications.[11] The DIC seen in this patient was probably different from the DIC seen in other obstetric emergencies because it persisted for more than five hours after removal of the conceptus and resolved only after therapy with heparin and aminocaproic acid. It is of interest to note the presence of amniotic fluid debris in decreasing amounts in each of the five days that the Swan-Ganz catheter was in place. This suggests a trapping effect of the debris which might contribute to the continuing DIC.

Although a number of preventive measures have been suggested, there is no known method of assessing the risk of amniotic fluid embolism or preventing its occurrence. A prompt diagnosis and aggressive therapy based on hemodynamic monitoring hold the key to improved chances of survival.

REFERENCES

1. Morgan M: Amniotic fluid embolism. *Anaesthesia* 1979; 34:20–32.
2. Steiner EP, Lushbaugh CC: Maternal pulmonary embolism by amniotic fluid as cause of obstetrical shock and unexpected deaths in obstetrics. *JAMA* 1941; 117:1245–1254, 1340–1345.
3. Courtney LD: Amniotic fluid embolism. *Obstet Gynecol Surv* 1974; 29:169–177.
4. Gross P, Benz EJ: Pulmonary embolism by amniotic fluid. Report of three cases with a new diagnostic procedure. *Surg Gynecol Obstet* 1947; 85:315–320.
5. Tuck CS: Amniotic fluid embolus. *Proc R Soc Med* 1972; 65:94–95.
6. Resnik R, Swartz WH, Plumer MH, Benirschke K, Stratthaus ME: Amniotic fluid embolism with survival. *Obstet Gynecol* 1976; 47:295—298.
7. Schaerf RHM, de Campo T, Civetta JM: Hemodynamic alterations and rapid diagnosis in a case of amniotic-fluid embolus. *Anesthesiology* 1977; 46:155–157.
8. Masson RG, Ruggieri J, Siddiqui MM: Amniotic fluid embolism: definitive diagnosis in a survivor. *Am Rev Resp Dis* 1979; 120:187–192.
9. Sparr RA, Pritchard JA: Studies to detect the escape of amniotic fluid into the maternal circulation during partuition. *Surg Gynecol Obstet* 1958; 107:560–564.
10. Roche Jr WD, Norris HJ: Detection and significance of maternal pulmonary amniotic fluid embolism. *Obstet Gynecol* 1974; 43:729–731.
11. Chung AF, Merkatz IR: Survival following amniotic fluid embolism with early heparinization. *Obstet Gynecol* 1973; 42:809–814.

See also pp 238–240.

Amniotic fluid embolism

PERVEZ AHMED, MD, CHARLES TRAUBE, MD, OYA FRESKO, MD

Amniotic fluid embolism is a rare condition, although its occurrence is a common cause of obstetric maternal mortality.[1] The following patient with amniotic fluid embolism is described because of the hemodynamic data obtained during right heart catheterization, and because of the patient's rapid response to a treatment regimen that included high doses of corticosteroids.

CASE REPORT

A 25-year-old gravida 8 para 7 woman was admitted to Brookdale Hospital Medical Center on August 8, 1980 with diffuse abdominal pain and vomiting at 36 weeks gestation. The patient had diabetes mellitus that was treated with 50 units of NPH insulin daily; her antepartum course was complicated by difficulty in controlling her blood glucose level because of poor dietary compliance. She had experienced episodes of diabetic ketoacidosis, and had undergone seven induced abortions. A physical examination was unremarkable except for occasional bouts of vomiting and a uterine fundus consistent in size with her expected date of delivery but without recordable fetal heart sounds. Her blood glucose level was 621 mg%, and the arterial pH was 7.25. Acetone was present in her serum.

During the next 24 hours the patient was treated with intravenous insulin; a subsequent blood glucose measurement was 145 mg%. Approximately 72 hours after admission the patient received oxytocin infusion, but, despite occasional contractions, the fetus was not expelled. One day later labor progressed and a dead macerated fetus was delivered. The placenta was then expelled, and it appeared intact.

Shortly after delivery the patient became dyspneic and cyanotic and complained of dizziness and tightness of the chest. She was restless and confused. Her blood pressure was 90/60 mmHg; pulse rate, 136 and regular; respiratory rate, 40/min; temperature, 35.8°C. An examination of the chest demonstrated bilateral diffuse rhonchi and bibasilar rales. Arterial blood gas studies yielded a pH of 6.89; PO_2, 50 mmHg; and PCO_2, 16 mmHg. Her blood glucose value was 328 mg%, and a trace of acetone was present. The lactic acid level was 15.8 mg/dL. A chest roentgenogram revealed alveolar infiltrates (Fig 1), and sinus tachycardia and nonspecific T wave abnormality were detected on an electrocardiogram. The white blood cell count was 32,000/mm^3 with 42% band forms. The prothrombin time was 12 sec (control, 19 sec), and the partial thromboplastin time was greater than two minutes. The fibrinogen level was 150 mg/dL, and fibrin degradation products were present. A peripheral smear revealed the presence of schistocytes and fragmented red blood cells; the platelet count was 105,000/mm^3.

Because of the relative hypotension and worsening blood gas values immediately after delivery, an endotracheal tube was inserted, and the patient was placed on a mechanical ventilator with the addition of positive end expiratory pressure. She was transferred to the intensive care unit where a Swan-Ganz catheter was inserted via the

---

From the Divisions of Cardiology (Drs Ahmed and Traube) and Pathology (Dr Fresko), Brookdale Hospital Medical Center, Brooklyn, NY.

Address correspondence to Dr Ahmed, Attending Physician, Department of Cardiology, The Brookdale Hospital Medical Center, Linden Blvd at Brookdale Plaza, Brooklyn, NY 11212.

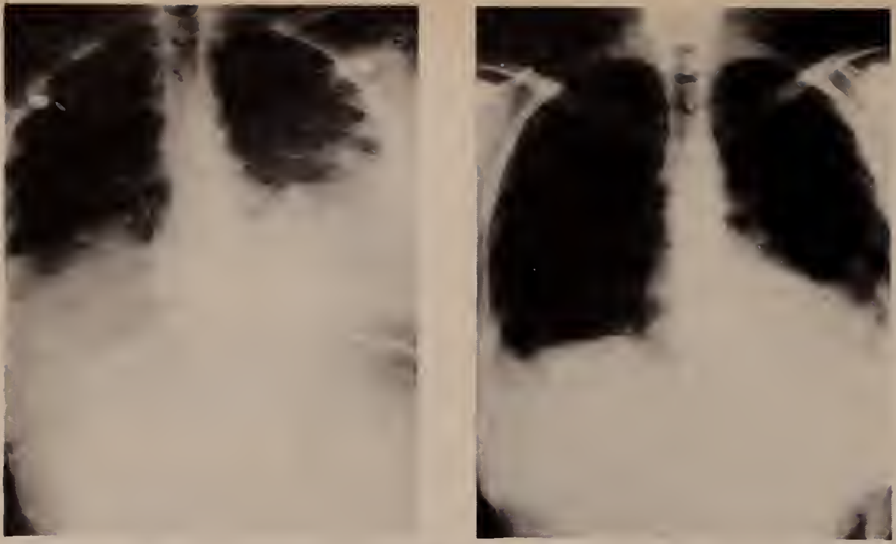


FIGURE 1. Left, chest roentgenogram taken shortly after delivery; Right, chest roentgenogram taken 48 hours after delivery.

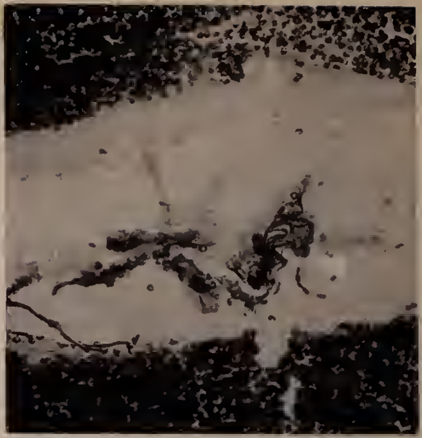


FIGURE 2. Direct smear of blood aspirated from the right atrium via a Swan-Ganz catheter. Note fetal squamae present in smear (arrow). (hematoxylin and eosin stain, original magnification × 200).

right internal jugular vein. The following pressure readings were recorded: mean right atrium, 1 mmHg (normal, 0 to 4 mmHg); pulmonary artery, 26/12 mmHg (normal, 15-30/4-12 mmHg); right ventricle, 22/1 mmHg (normal, 15-30/0-9 mmHg); mean pulmonary artery, 18 mmHg (normal, 10-15 mmHg); pulmonary capillary wedge pressure, 4 (normal mean, 4-12 mmHg); cardiac output by thermodilution, 3.2 L/min (normal, 5-6 L/min).

The patient received rapid volume expansion, which consisted of intravenous aliquots of normal saline solution beginning with 50 cc every 10 minutes and decreasing to 20 cc every 10 minutes until the pulmonary capillary wedge pressure was raised to 12 mmHg. She received a total of 2.5 liters of normal saline solution in 2½ hours. As part of the treatment for adult respiratory distress syndrome, she also received an initial dose of 200 mg of intravenous dexamethasone propionate and doses of 200 mg every six hours for 48 hours.

Within 48 hours, the patient's blood pressure was 130/80 mm Hg, signs of pulmonary edema—such as rales and radiologic signs of alveolar fluid—had decreased, and her clotting parameters were within normal limits (Fig 1). The endotracheal tube was removed. Blood obtained from the Swan-Ganz catheter revealed the presence of fetal cells in the right atrium (Fig 2). The patient recovered well and was discharged from the hospital on August 11, 1980.

DISCUSSION

Amniotic fluid embolism has been described as "the most dangerous and untreatable condition in obstetrics."[2] The syndrome involves a triad of respiratory distress, cyanosis, and shock. It is usually seen in a multigravid patient, with acute onset of the syndrome in the intrapartum or postpartum period of an active or tumultuous labor, frequently following use of uterine stimulants for the induction or progression of labor.

Clinical manifestations of this condition are similar to those of pulmonary embolism and are characterized by the sudden onset of dyspnea, chest pain, cyanosis, disseminated intravascular coagulation, and shock.

This disorder is associated with hemorrhage, hypofibrinogenemia, and almost instantaneous cardiovascular collapse.[3] Supportive measures are frequently inadequate, and death usually results. Only 25 cases of survivors of amniotic fluid embolism have been reported in English language medical journals.

Diagnosis of amniotic fluid embolism is usually made at postmortem examination by findings of squamae, lanugo hair, mucin, and fat emboli in the lungs.[4,5] In patients who survive this condition, a presumptive diagnosis of amniotic fluid embolism is made by the finding of squamae or lanugo hair in the maternal sputum, or in blood obtained from a central venous catheter.

This patient illustrates several typical features of this disorder: sudden respiratory and cardiovascular collapse, development of disseminated intravascular coagulation, and the finding of diagnostic cellular material in blood from the right atrium. The use of high doses of corticosteroids, along with other supportive measures, was effective in this patient.

Hemodynamic alterations in amniotic fluid embolus have been reported.[6] That report, however, included the finding of left heart failure. This patient had normal left-sided pressure, pulmonary hypertension, and low cardiac output. These findings are more in accordance with the hemodynamic observations of patients with adult respiratory distress syndrome.

Experimental studies have not identified the cause of the syndrome associated with amniotic fluid embolism. Hypotheses to explain the shock and intravascular coagulation range from anaphylaxis, to the effect of the quantity of material entering into the circulation, to the effect of an undefined humoral substance. The role of complement activation and its effect in causing aggregation of polymorphonuclear cells is being clarified in a number of clinical entities including adult respiratory distress syndrome with shock.[7,8] The disseminated intravascular coagulation seen with adult respiratory distress syndrome[9] in amniotic fluid embolism could be secondary to complement activation, triggered by entry of amniotic material into the maternal circulation. This in turn could initiate leukocyte aggregation with resultant occlusion of capillaries in the lung and release of perioxidase and superoxidase, subsequently causing an increase in capillary permeability and lung edema. Corticosteroids have been shown to inhibit complement aggregation of leukocytes.[7]

The hemodynamic values measured in this patient support the concept that adult respiratory distress syndrome was not due to fluid overload or left ventricular failure, but was the result of a capillary leak phenomenon. The rapid recovery of the patient may be due to the ability of steroids to inhibit complement activation, as has been reported.[7–9] Further studies to measure circulating activated complement fragment c5a, which is a potent leukocyte aggregating agent, are needed to test this hypothesis in amniotic fluid embolism.

A patient suspected of having amniotic fluid embolism should undergo hemodynamic monitoring via a Swan-Ganz catheter, as well as monitoring for cardiac output. High doses of corticosteroids are recommended in the hope that they will speed reversal of both adult respiratory distress syndrome and the disseminated intravascular coagulation associated with this condition.

REFERENCES

1. Danforth DN: *Textbook of Obstetrics and Gynecology*, 2nd ed. New York, Harper & Row Publishers, 1971, p 682.
2. Courtney LD: Amniotic fluid embolism. *J Ir Med Assoc* 1973; 66:51.
3. Stephanini M, Turpini RA: Fibrinogenopenic accident of pregnancy and delivery: a syndrome with multiple etiological mechanisms. *Ann NY Acad Sci* 1959; 75:601.
4. Schaerf RH, deCampo T, Civetta JM: Hemodynamic alterations and rapid diagnosis in a case of amniotic fluid embolus. *Anesthesiology* 1977; 46:155–157.
5. Gross P, Benz EJ: Pulmonary embolism by amniotic fluid: report of 3 cases with new diagnostic procedure. *Surg Gynec Obstet* 1947; 85:315, 320.
6. Reis RL, Pierce WS, Behrendt DM: Hemodynamic effects of amniotic fluid embolism. *Surg Gynec Obstet* 1969; 129:45–48.
7. Hammerschmidt DE, White JG, Craddock PR, Jacob HS: Corticosteroids inhibit complement-induced granulocyte aggregation. *J Clin Invest* 1979; 63:798–803.
8. Hyers TH: Pathogenesis of adult respiratory distress syndrome: current concepts. *Sem Respir Med* 1981; 11:104–107.
9. Bone RC, Francis PB, Pierce AK: Intravascular coagulation associated with adult respiratory distress syndrome. *Am J Med* 1976;61:585–589.

See also pp 238–240.

Spontaneous remission in acute nonlymphocytic leukemia

AZRA RAZA, MD, LIVELEEN M. GILL, MD, IRENE RAKOWSKI,
HARVEY D. PREISLER, MD

Spontaneous remission in acute nonlymphocytic leukemia (ANLL) is a rare but not unknown phenomenon. We describe a patient with acute promyelocytic leukemia who, on initial contact, was so acutely ill that chemotherapy had to be withheld until the overwhelming bacterial infection could be brought under control. As the infection responded, a simultaneous improvement was noted in his hematologic picture as well as a partial remission in the bone marrow.

CASE REPORT

An acutely ill 52-year-old man had had a fever and urinary tract infection for two weeks that was being treated with an oral cephalosporin. On physical examination, he was noted to have a markedly inflamed scrotum and penis with severe cellulitis extending to both inguinal areas. A laboratory profile revealed a white blood cell count of 21.2×10^9/L with 8% blast cells, 69% leukemic promyelocytes with multiple Auer rods, 3% myelocytes, 6% segmented neutrophils, 3% monocytes, and 11% lymphocytes. The hemoglobin was 8.3 g/dL, and a platelet count was 68×10^3/L. Prothrombin time was 13.5 sec compared to a control time of 12.0 sec; partial thromboplastin time was 42 sec, and fibrinogen level was 443 mg. Fibrinogen degradation products were greater than 8 and less than 64.

A diagnosis of acute promyelocytic leukemia was established from the bone marrow aspirate which showed 77% leukemic promyelocytes with markedly abnormal granules and numerous cells with multiple Auer rods (Fig 1). In addition, Fourrier's gangrene of the scrotum was diagnosed, and the patient was immediately taken to the operating room. A right orchiectomy with suprapubic cystostomy was performed, and multiple subcutaneous incisions were made throughout the perineum, with insertions of drains. He was treated with broad-spectrum antibiotics and transfusions of white blood cells. His condition deteriorated in the next 48 hours, with increasing spread of cellulitis to the umbilicus that required extensive debridement of the necrosed areas and insertion of additional subcutaneous drains. No chemotherapy could be contemplated for the treatment of the acute promyelocytic leukemia because of the poor physical condition of the patient.

The infection began to respond to the aggressive measures from the fourth hospital day, and simultaneously, the patient's white blood cell count began to fall (Table). His platelet count started to improve, as did his peripheral blood picture by the third week after surgery. At this time, he was able to pass urine per urethrum, so the suprapubic cystostomy was discontinued.

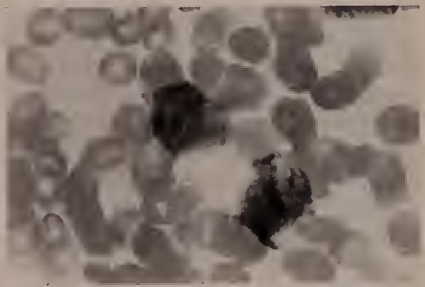


FIGURE 1. Bone marrow aspirate obtained at the time of initial presentation showing leukemic promyelocytes, one of them containing multiple Auer rods.

He was discharged from the hospital in excellent physical condition, and he continued to do well for seven months after the diagnosis of acute promyelocytic leukemia. After seven months, his bone marrow began to show replacement by leukemic promyelocytes, and his peripheral white blood cell count fell to 0.9×10^9/L. Remission induction therapy was attempted with 10 days of cytosine arabinoside at 100 mg/M^2 by continuous intravenous infusion and three days of adriamycin at 30 mg/M^2 by intravenous push. During the hypoplastic phase after administration of chemotherapy the patient died of a severe necrotizing enterocolitis. An autopsy revealed extensive infiltration of the esophagus and gastrointestinal tract by leukemic promyelocytes.

DISCUSSION

In this patient with acute promyelocytic leukemia chemotherapy could not be administered due to the severity of a life-threatening bacterial infection. After response to the infection, a simultaneous spontaneous partial remission of the leukemia was noted, with normalization of the peripheral blood picture as well as a reduction of bone marrow blasts to 3% and abnormal leukemic promyelocytes with Auer rods to 14%.

Fifteen cases of spontaneous remission in ANLL were reported between 1930 and 1954,[1] but as the criteria for diagnosis of ANLL became more stringent and the definition of complete remission became more strict, the number of patients with spontaneous remis-

---

From the Roswell Park Memorial Institute, Leukemia Service, Buffalo.

Address correspondence to Dr Raza, Assistant Professor, Leukemia Service, Roswell Park Memorial Institute, 666 Elm St, Buffalo, NY 14263.

Serial Peripheral Blood and Bone Marrow Examinations in a Patient with Acute Promyelocytic Leukemia Who Achieved a Spontaneous Partial Remission

	5/11/83		5/23/83		6/27/83		8/1/83		9/6/83		10/27/83		11/3/83		11/10/83	
	PB[‡]	BM[†]	PB	BM	PB	BM	PB	BM	PB	BM	PB	BM	PB	BM	PB	BM
WBC × 10^9/L	21.2	—	1.3	—	1.4	—	1.9	—	2.0	—	0.9	—	0.5	—	0.1	—
Hemoglobin, g/dL	8.3	—	12.4	—	11.3	—	14.5	—	15.5	—	14.6	—	13.3	—	8.9	—
Platelets × 10^3/L	68	—	77	—	271	—	198	—	142	—	196	—	93	—	16.0	—
Myeloblasts %	16	0.6	—	1.8	—	1.4	—	1.6	—	3.0	—	11.2	—	3.0	—	0.5
Promyelocytes %	51	77.4	—	44.8	—	11.0	—	13.0	—	14.0	—	62.6	—	54.0	—	70.5
Myelocytes %	13	3.4	—	22.6	—	14.4	—	10.2	—	17.0	—	2.8	—	4.0	—	0.5
Segmented neutrophils %	2	0.8	40	5.4	16	6.0	60	9.8	50	20.5	—	2.4	—	3.0	—	5.5
Eosinophils %	—	—	—	0.6	—	0.8	—	1.8	1	0.5	—	0	—	0.5	—	0
Monocytes %	—	0.6	—	0.6	—	—	8	1.2	5	1.0	—	0	—	0	—	0
Lymphocytes %	—	6.8	60	4.2	84	8.8	32	7.2	43	11.5	—	6.2	—	27.5	—	21.5
Megakaryocytes	—	N[**]	—	N	—	N	—	N	—	N	—	N	—	N	—	N
Erythroid Cells %	—	8.4	—	17	—	55.8	—	54.8	—	32.5	—	14.8	—	6.5	—	0.5
Cellularity	—	3+	—	3-4+	—	2+	—	1-2+	—	1+	—	2+	—	1+	—	1+

[†] BM, bone marrow

[‡] PB, peripheral blood

[*] Cellularity, subjectively assessed from 0 to 4+

[**] N, normal

sions became progressively fewer. As a result, only five such patients have been reported since 1955.[2] One patient, very similar to this patient, was described in this journal in 1951[3]: an 18-year-old woman had gangrenous ulceration of the palate, a swollen left hip, high fevers, and a peripheral blood and bone marrow picture consistent with the diagnosis of acute monocytic leukemia. After an incision and drainage procedure and antibiotic therapy gradual clinical improvement was achieved, and there was a concomitant improvement of her hematologic condition. Her white blood cell count at initial contact was 16.0×10^9/L, which fell to 1.6 to 3.0×10^9/L when she left the hospital, and her platelet count rose from 86×10^3/L on admission to 370×10^3/L at discharge. Serial bone marrow studies revealed that only a partial remission was achieved, and the patient had a relapse and died six months later. The disease manifestation and subsequent course in this patient are almost identical to those of the patient described in this report.

A review of the literature shows that several cases of spontaneous remission in ANLL were associated with severe life-threatening infections.[2,3] It was therefore proposed that nonspecific stimulation of the immune system by the infection might play a major role in inducing remission. Therefore, attempts were made to manipulate the immune system artificially by such techniques as injection of malarial parasites,[4] the use of *M turberculosum*[5] or methanol-extracted residue.[6] These measures have not resulted in any sustained benefit to date, and their use has been restricted to a few specialized centers.

The patient described here demonstrated a similar association of severe infection and a spontaneous partial remission due to a transient suppression of the leukemic cells. Interestingly, several cytogenetic examinations performed both on initial examination and during the course of his illness revealed only normal metaphases. A substantial number of patients with acute leukemia will not show any detectable cytogenetic abnormalities unless specialized prophase banding techniques are applied[7]; we performed only the standard G-bandings. Therefore, the absence of the classic translocation t(15;17) pathognomonic of acute promyelocytic leukemia did not rule out such a diagnosis in this patient.

The possibilities that this was only a leukemoid reaction in response to an overwhelming infection or a "maturation arrest" in a patient with an underlying preleukemic process that later evolved into acute promyelocytic leukemia were ruled out for the following reason: the patient had thrombocytopenia in addition to leukocytosis, and even though these features may be seen in sepsis with or without disseminated intravascular coagulation, the bone marrow was extremely hypercellular and was infiltrated with abnormal promyelocytes, with a majority of cells containing multiple Auer rods as well as abnormal granularity. It is interesting that the final episode was not accompanied by leukocytosis, as was the initial manifestation. This could mean either that there was indeed a leukemoid reaction along with the leukemic process because of sepsis, or that the nature of the leukemic process had undergone a biological change during the partial remission.

During therapy for the infection, the patient did not receive any vitamin or steriod therapy or any other drugs besides antibiotics and an occasional analgesic or antipyretic. Therefore, inducing differentiation and maturation by agents such as *cis*-retinoic acid is out of the question as well.

Treatment with chemotherapy resulted in death during hypoplasia, and an autopsy revealed extensive leukemic infiltration throughout the gastrointestinal tract. Nevertheless, the patient had a spontaneous partial remission of his illness that enabled him to live somewhat normally for seven months.

References

1. Lachant NA, Goldberg J, Nelson DA, Gottlieb AJ: Spontaneous remission in acute myelogenous leukemia in the adult. *Am J Med* 1979; 67:687-692.
2. Wiernik P: Conference on spontaneous regression of cancer, National Cancer Institute Monograph 44 U.S. Dept of Health, Education, and Welfare publication No (NIH) 76:1038, 1976.
3. Schwarz A, Levine L: Prolonged spontaneous remission in acute leukemia. *NY State J Med* 1951; 51:2413-2415.
4. Pelner L, Fowler GA, Nauts HC: Effects of concurrent infections and their toxins on the course of leukemia. *Acta Med Scand* 1958;162(Suppl 388):1-47.
5. Omura GA, Vogler WR, Lynn MJ: A controlled clinical trial of chemotherapy versus BCG immunotherapy vs no further therapy in remission maintenance of acute myelogenous leukemia. *Proc Am Soc Clin Oncol* 1977; 18:272.
6. Whitaker JA, Bailey-Wood R, Hutchins S: Active immunotherapy for the treatment of acute myelogenous leukemia, in Terry WD, Rosenberg SA (eds): *Immunotherapy of Cancer*. New York, Elsevier-North Holland, 1982, p 23.
7. Testa JR, Rowley JD: Chromosomes in leukemia and lymphoma with special emphasis on methodology, in Catkovsky D (ed): *The Leukemic Cell*. Edinburgh, Churchill-Livingstone, 1981, p 184.

LIFE STRESS AND CORONARY HEART DISEASE

By Ulf De Faire, MD and Tores Theorell, MD. 118 pp, illustrated. St. Louis, Warren H. Green, Inc, 1984. $18.50 (hardcover)

Two Swedish physicians, one an internist and the other a psychosociologist, have compiled a brief summary of the literature along with studies of their own concerning the relationship between life stress and coronary artery disease.

The physiology of stress, along with its cardiovascular manifestations, is presented in some detail. Building on this foundation, a temporal relationship between life stresses and coronary artery disease is established. Studies of the behavior patterns of the coronary-prone individual are discussed. Type A personality patterns are emphasized, and the origins of this type of behavior are highlighted. One chapter describes the typical patient with coronary problems and the psychosocial factors that may determine the length of time between the onset of symptoms and admission to a coronary care unit, along with the emotional factors that make for satisfactory rehabilitation. The last chapter discusses the various medical and psychosocial risk factors and interventional strategies that affect the prevention of coronary artery disease.

Each chapter is followed by an extensive bibliography. In fact, the book is an excellent introduction to the current status of the subject. Just about every aspect of the subjects discussed is fully cited.

It becomes evident from the authors' presentations that the relationship of emotional stress to human function and disease, which seems so obvious on the surface, still remains elusive when put to the scientific test. The postulates of such greats as Hans Selye and Harold Wolff are still the subject of more specific and controlled experimentation and more sophisticated statistical analysis. Yet, such phrases as "may thus be" and "seem to verify" are still common in this text, despite its wealth of scientific experiments.

The authors themselves have been deeply involved in their subject and have had considerable experimental experience in relating stressors to stress in coronary heart disease. For years they have been involved in psychosocial studies of 5,000 41 to 61-year-old construction workers in greater Stockholm. For example, they were able to relate increased stress at work with an increased risk of myocardial infarction within the year. This finding correlated well with a nationwide Swedish study which made a similar correlation retrospectively.

To one who has a sizable time perspective in relation to medicine, it is heartening to observe the persistence with which investigators continue to whittle away at this complex subject, and yet it is disappointing to find still so many ifs and maybes in these scientific discussions.

The authors allow that they have not attempted to present a comprehensive review of the subject, but have been interested mainly in bridging the gap between the disciplines of clinical medicine, psychosociology, and epidemiology. In so doing, they have provided a brief text that includes the essence of studies that associate life stress with coronary heart disease.

JOHN N. EDSON
The Long Island College Hospital
New York City 11201

PEDIATRIC RISK FACTORS FOR MAJOR CHRONIC DISEASE

By Christine L. Williams, MD, MPH. 159 pp, illustrated St. Louis, Warren H. Green, Inc, 1984. $22.50 (hardcover)

Pediatricians and family physicians should find this book about pediatric risk factors for cardiovascular disease and cancer interesting and relevant to their practices, even though the book is directed to health educators, parents, the mass media, and producers of products that are consumed by children, as stated in the foreword.

During the last decade, there has been an association between the interest of the public in physical fitness and a lessening of cardiovascular mortality. The author offers hope that continuing progress in reducing morbidity and mortality can be made by educating children in a prudent lifestyle. She thinks that there are sufficient data about the association of cardiovascular disease and the risk factors of smoking, sedentary habits, blood cholesterol, obesity, hypertension, and others to formulate an educational plan of action. Corroborating data are given, with extensive references including many from the Framingham study, the Bogalusa heart study, autopsy data from the Korean and Vietnam wars, and Scandinavian studies.

The author has been involved in the "Know Your Body" program, sponsored by the American Health Foundation in several New York City schools, and uses it as a model to show how education can be employed to reduce the major risk factors. Of the children involved in the program, 36% of the junior high school students and 33% of the first through third grade students had one or more risk factors present. The most common was an elevation of serum cholesterol followed by obesity and cigarette smoking. Using the educational approach of the "Know Your Body" program, the author outlines methods used to reduce the major risk factors. In this program, the children and their teachers underwent extensive risk factor screening, involving a medical history, cholesterol and high density lipoprotein determination, blood pressure readings, and physical measurements. A "Personal Health Passport" was given to each child and parent which summarized the risk factor status. The child's physician was also notified if necessary. A behavior oriented health education curriculum was used and was evaluated during a three-year period. The author suggests that modifications of the curriculum could be used by many school systems. The concepts could also be used in a pediatric practice. The material is presented to the children in a non-threatening way, using the principle of a "positive approach" to a lifestyle change. The approach is a logical one, but, as the study showed, education has to extend through the entire 12 grades to be effective.

I know of no other book of similar

content directed toward the proposed audience. It deserves to be read by anyone interested in "wellness" or preventive medicine, since the subject is handled in a "non-faddist," objective manner. It accomplishes its stated purpose and is a good introduction to an important subject. The expense of this hardcover book, unfortunately, may be a deterrent to its widespread use, especially by educators.

JOHN R. BREWER, MD
St. Louis University School of Medicine
St. Louis, MO 63104

THE ADDICTIVE BEHAVIORS

By Howard Shaffer, PhD, and Barry Stimmel, MD. 172 pp. New York, The Haworth Press, Inc, 1984. $22.95 (hardcover)

This book is a special hardcover issue of "Advances in Alcohol and Substance Abuse," and was essentially derived from a conference with the same title held at Harvard Medical School in October, 1982. The purpose of the book is to attempt a "description and classification of addictive human behavior" in order to look for patterns and similarities and to develop "an ecology that deals with the relationship between man and drugs viewed as specific features of our environment inexorably interwoven as part of our complex social system." In fact, the book is a collection of nine articles of uneven quality, only a few of which have some relationship to the goals of the editors.

Two of the chapters are quite good and may be worth the price of the volume. "Caffeine: A potential drug of abuse" is an excellent, useful and well written review of the literature on this subject. Included is startling information about the average caffeine consumption of small children, the negative effects of caffeine on pregnancy, and the incidence of anxiety, depression, psychophysiologic disorders, and impaired work performance associated with moderate and heavy caffeine intake.

An article by Margaret Bean, "Clinical implications of models for recovery from alcoholism," is an excellent review of the ways in which the three controversial models (the disease, the psychogenic, and the moral models) of the etiology of alcoholism affect patients and clinicians and of how they can be combined to understand and treat the disease. This is one of the clearest discussions of this subject that I have read.

The articles on methadone maintenance and on controlled opiate use are well written and would be useful to those unfamiliar with these areas, but would offer nothing new to most workers in the field.

The rest of the chapters are for the most part poorly organized and of little use. The chapter "A strategy for the biological assessment of addiction" provides no good description of biological aspects of addiction and no strategy for assessing them. The chapter "Diagnosis in the addictions: conceptual problems" uses philosophical constructs and jargon not familiar to clinicians or researchers in the field, for whom it would be essentially unreadable. A chapter on cigarette smoking in adolescents is a report of a specific study of some of the variables in smoking behavior in a small subgroup of smokers in one setting. An overview like that presented on caffeine would have been much more relevant and useful.

If the editors had solicited chapters that directly addressed the goals and objectives of the book and had taken care that all of the resulting articles were consistent with that purpose, then this volume might have been a useful addition to the literature in the field. As it is, I can recommend it only for the two chapters mentioned above.

I have one additional minor cavil. "Behavior" is the proper term to use for both the singular and plural modes; "behaviors" is not grammatically appropriate.

BERNARD BIHARI, MD
SUNY-Downstate Medical Center
New York City 11203

MANAGEMENT OF CHRONIC SCHIZOPHRENIA

Edited by Carol L. M. Caton. 210 pp. New York, Oxford University Press, 1984. $19.50 (hardcover)

The American public has been faced with the problem of coping with the great numbers of troubled and disturbed people in its midst due to the recent program of deinstitutionalization. As Dr Lawrence C. Kolb stresses in the preface to this book, the effects of this process during the past two decades were not foreseen by planners and administrators. Yet all of us have been strained by the vast shift that has taken place in the care of those who suffer from serious mental illness.

Dr Carol Caton is eminently qualified to address this problem and does so in a scientific and objective manner. The editor is a capable researcher and writes in a clear and attractive style. She reviews the history of mental institutions and how the medical profession and society established treatment processes from pre-colonial days to the present.

The desire to give freedom and better care to chronically disturbed patients, as well as the poor levels of care that existed in many state hospitals, led to the process of deinstitutionalization. This process has produced a 70% drop in the resident population in state and county mental hospitals in the United States. The psychopharmacological revolution in psychiatry, as well as economic and political factors, also propelled this process.

Caton makes clear that the lofty goals of the process have not been realized; rather, these patients are now in new life situations. They may be in sheltered care facilities, in state hospitals, or in jails and prisons. Others are homeless or have achieved independent marginal life styles. As a result, new and different problems have arisen. This book examines the effectiveness of programs aimed at dealing with this dilemma.

Enlightening and helpful sections are presented by Sheehy on chronic schizophrenia, the primary disease at the root of the problem, and how this disorder is diagnosed and treated. The section on hospital treatment, by Maxman, is informative, describing the types of hospitals and the approaches that may be taken, from therapeutic communities to custodial care units.

Caton provides objective insights on the delivery of after-care services, including discharge planning, the management of medication, and psychosocial treatments. The critical role that the family plays in the treatment of the mentally ill is also discussed, and innovations in therapy are outlined.

Caton describes steps that are being taken to foster competence in this troubled population, including the teaching of social skills and training in community living. She elaborates on the efforts that have been undertaken in gaining employment for patients as well as the use of industrial therapy. A review of housing alternatives is provided, including foster-family care, halfway houses, the community lodge, satellite housing, and board and care homes.

The treatment of chronic, deeply disturbed schizophrenic patients places real burdens on mental health professionals. Caton describes the phenomenon of burnout in such professionals and offers steps for prevention, emphasizing reasonable patient-to-staff ratios, shorter work hours, sharing of burdens, and management of intrastaff conflict. All of this must lead to a sense of creativity and fulfillment in the daily tasks of staff members. Finally, avenues of research are recommended.

This book arrives at an appropriate time, as society attempts to deal with this mentally disturbed population. The book is recommended for therapists, mental health administrators, and policy makers.

THOMAS L. DOYLE, MD
Payne Whitney Clinic
New York Hospital
New York City 10021

MAJOR DEPRESSION: PREVENTION AND TREATMENT

By Michael R. Lowry, MD, 120 pp. St. Louis, Warren H. Green, Inc, 1984. $15.00

"This text," says Lowry in chapter one of this book, "is the result of one man's effort to assess what is already known in the area of the prevention of one mental disability, ie, depression, and to point to specific gaps in that knowledge which current and future research hold promise of filling." Eighty-six pages later Lowry concludes "we are not currently prepared to offer the public or individual patients primary prevention (reduction in incidence) of depressive disorders." Effective secondary prevention (reduction in prevalence, treatment of the disorder once established), however, is available, in the form of lithium for treatment of acute mania and in prophylaxis of bipolar disorder, electroconvulsive therapy, tricyclic antidepressants, and psychotherapy in major depression.

To reach these conclusions, Lowry reviews the evidence for factors that are said to predispose a person to depression or to precipitate depression: genetic factors, with reviews of family, twin, and adoption studies; psychological factors which stress the deterministic role of childhood bereavement to depressive disorder in adults; the role of life events in the precipitation of depression. One section briefly mentions the attempt to find biochemical markers for depression (thyrotropin releasing hormone, serotonin, and methoxyhydroxyphenylglycol).

As Lowry warns, this small volume makes little scholarly pretense to study depression in depth. It is as though somewhere in his residency he reviewed the subject for himself and is summarizing his understanding and feelings with us. The book is thus simply written, offers little new knowledge to practitioners in the field, and has conclusions which are not at all surprising. For non-psychiatrists, however, the book may be of interest. The author's ideas are clearly expressed, and the book is typographically easy to read.

ARCHIE A. SILVER, MD
University of South Florida
Tampa, FL 33612

ALZHEIMER'S DISEASE: THE STANDARD REFERENCE

Edited by Barry Reisberg, MD. 464 pp. New York, Free Press, 1983. $45.00

The elderly now make up an increasingly significant percentage of the population of the United States. Recent estimates indicate that there are 25 million people aged 65 or older and that this figure will probably increase to 40 million by the turn of the century. Alzheimer's disease, a disease primarily of the elderly, is attracting an enormous amount of attention within the medical, scientific, and lay communities. It is estimated that more than 5% of the elderly will develop Alzheimer's disease. As the numbers rise, research dollars are becoming available, and the clinician is increasingly confronted with people's concerns about this disease. Thus, an up-to-date, comprehensive textbook is essential.

This book, edited by Barry Reisberg, partially fills the above needs. It is geared toward a scientifically sophisticated audience. It is divided into 13 sections that highlight theories about the pathologic and neurochemical features of the disease, its clinical presentation, and its epidemiology. Diagnostic issues, the use of laboratory animal models, and possible treatments are also discussed.

The various chapters are written by experts in their fields. Up-to-date references make the volume a good source book of information about Alzheimer's disease; more than 1,000 references are cited throughout the book.

As with any book of this type, there are a number of problems. The virtues of having chapters of only three pages in length can be defended, but perhaps it would have been better to have one chapter on neurochemistry rather than nine. There is a redundancy of introductory material, because each author has written a chapter that includes general comments already made by other authors.

A number of chapters are well beyond the ken of the general practitioner. It might have been better for each of the first 12 sections to have had an introduction that would explain, for example, how genetic factors, chromosomal factors, and histocompatibility might overlap in understanding Alzheimer's disease. The non-human models of Alzheimer's disease seem to float between a discussion about pharmacotherapy and psychotherapy. A more clinically oriented chapter on the interplay of all treatment methods should have been included.

Despite some shortcomings, the book approaches a state-of-the-art understanding of Alzheimer's disease. It will be interesting to return to this book 10 or 15 years from now to reexamine the theories associated with Alzheimer's disease.

KENNETH P. KAHANER, MD
Jewish Institute for Geriatric Care
New Hyde Park, NY 11042

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Farrow Robert Allen, MD, Bronx. Died February 22, 1985; age 87. He was a 1926 graduate of Harvard Medical School. Dr Allen was a Fellow of the American College of Surgeons, and a member of the Academy of Medicine, the American Thoracic Society, the New York County Medical Society and the Medical Society of the State of New York.

Cosimo Basirico, MD, Brooklyn. Died January 2, 1985; age 55. He was a 1962 graduate of Università degli Studi di Padova, Facoltà Medicina e Chirurgia, Padova. Dr Basirico was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Fritz Jakob Bloch, MD, Sarasota, Florida. Died February 1, 1985; age 88. Dr Bloch was a 1921 graduate of Ludwig-Maximilians-Universität Medizinische Fakultät, München. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Kenneth Francis Bott, MD, Greenville. Died January 19, 1985; age 85. Dr Bott was a 1928 alumnus of Albany Medical College of Union University. He was a member of the Medical Society of the County of Greene and the Medical Society of the State of New York.

Bruce E. Bradley, MD, New York. Died in September, 1984, age 82. He was a 1926 alumnus of the University of Virginia School of Medicine. Dr Bradley was a member of the American Occupational Medical Association, the New York County Medical Society and the Medical Society of the State of New York.

William D. Brandon, MD, Massapequa. Died February 14, 1985; age 63. He was a 1946 graduate of Jefferson Medical College of Philadelphia. Dr Brandon was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the American Geriatrics Society, the Nassau County Medical Society and the Medical Society of the State of New York.

Giovanni N. Caggiano, MD, Died August 30, 1984; age 84. Dr Caggiano was a 1923 alumnus of Università di Napoli Facoltà di Medicina e Chirurgia, Napoli. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Berthold R. Comeau, MD, Delray Beach, Florida. Died February 14, 1985; age 84. Dr Comeau was a 1928 graduate of Yale University School of Medicine, and was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Edwin Fisk Comstock, MD, Wellsville. Died February 7, 1985; age 83. Dr Comstock was a 1927 alumnus of the University of Pennsylvania School of Medicine. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Allegany and the Medical Society of the State of New York.

Gerard C. De Grandpre, MD, Plattsburgh. Died January 14, 1985; age 89. Dr De Grandpre was a 1924 alumnus of the University of Vermont College of Medicine. He was a member of the Medical Society of the County of Clinton and the Medical Society of the State of New York.

Joseph Epstein, MD, New York. Died February 3, 1985; age 86. He was a 1923 graduate of Yale University School of Medicine. Dr Epstein was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the Academy of Medicine, the New York Neurological Society, the American Psychiatric Association, the Association for Research in Nervous and Mental Disease, the American Academy of Neurology, the New York County Medical Society and the Medical Society of the State of New York.

Enid Lynn Feld, MD, New York. Died September 16, 1984; age 34. Dr Feld was a 1976 alumna of the University of California San Diego School of Medicine. She was a Diplomate of the American Board of Nuclear Medicine, and a member of the Society of Nuclear Medicine, the New York County Medical Society and the Medical Society of the State of New York.

Perry Michael Fersko, MD, Brooklyn. Died October 27, 1984; age 54. He was a 1956 alumnus of the State University of New York Downstate Medical Center. Dr Fersko was a Diplomate of the American Board of Internal Medicine, and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

John Edward Franklin, MD, New York. Died February 10, 1985; age 78. Dr Franklin was a 1932 alumnus of Harvard Medical School. He was a Diplomate of the American Board of Pediatrics, and a member of the Academy of Medicine, the American Academy of Pediatrics, the Society of Surgical Oncology, the New York County Medical Society and the Medical Society of the State of New York.

Harry Ginsberg, MD, Hollywood, Florida. Died February 6, 1985; age 84. He was a 1926 graduate of the State University of New York Downstate Medical Center. Dr Ginsberg was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Urology. He was a member of the American Urological Association, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Arthur Vose Greeley, MD, New York. Died February 20, 1985; age 82. He was a 1929 alumnus of Johns Hopkins University School of Medicine. Dr Greeley was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Academy of Medicine, the New York Obstetrical Society, the American Fertility Society, the New York County Medical Society and the Medical Society of the State of New York.

Norman Heilbrun, MD, Buffalo. Died January 21, 1985; age 78. He was a 1929 graduate of the University of Buffalo School of Medicine. Dr Heilbrun was a Fellow of the American College of Radiology and a Diplomate of the American Board of Radiology. He was a member of the American Roentgen Ray Society, the Radiological Society of North America, Inc, the Medical Society of the County of Erie and the Medical Society of the State of New York.

Francis E. Kenny, MD, Buffalo. Died January 22, 1985; age 78. Dr Kenny was a 1931 alumnus of the University of Buffalo School of Medicine. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Anne M. Kullman, MD, Kenmore. Died January 15, 1985; age 77. She was a 1934 alumna of Universität Heidelberg Medizinische Fakultät, Baden. Dr Kullman was a Fellow of the American Academy of Family Physicians. Her memberships included the American Geriatrics Society, the Medical Society of the County of Erie and the Medical Society of the State of New York.

Ethel E. Little, MD, Santa Fe, New Mexico. Died February 3, 1985; age 93. Dr Little was an alumna of the University of Illinois College of Medicine. She was a member of the Medical Society of the County of Tompkins and the Medical Society of the State of New York.

Michael J. Maggiore, MD, Boynton Beach, Florida. Died February 10, 1985; age 81. Dr Maggiore was a 1929 graduate of the University of Buffalo School of Medicine. He was a member of the New York State Society of Occupational Medicine, the Academy of Medicine, the Broome County Medical Society and the Medical Society of the State of New York.

Jack Moore, MD, Wantagh. Died January 28, 1984; age 82. Dr Moore was a 1928 graduate of Tufts University School of Medicine. He was a member of the New York State Society of Pathologists, the American Psychiatric Association, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Nicholas F. Parise, MD, Brooklyn. Died July 21, 1984; age 79. He was a 1941 graduate of Chicago Medical School. Dr Parise was a member of the American Academy of Family Physicians and the Medical Society of the State of New York.

Jesse Silsby Parker, MD, Glens Falls. Died December 21, 1984; age 80. He was a 1932 graduate of Harvard Medical School. Dr Parker was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the Medical Society of the County of Warren and the Medical Society of the State of New York.

Ira Allan Polisar, MD, Great Neck. Died February 14, 1985; age 70. Dr Polisar was a 1941 graduate of the State University of New York Downstate Medical Center. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the New York Otological Society, the American Academy of Otolaryngology, the American Laryngological, Rhinological and Otological Society, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Bernard Saul Post, MD, Kings Point. Died February 6, 1985; age 70. He was a 1940 graduate of the Licentiate of the Royal College of Physicians of Edinburgh. Dr Post was a Fellow of the American College of Physicians and a Diplomate of the American Board of Physical Medicine and Rehabilitation. His memberships included the New York Society for Physical Medicine and Rehabilitation, the Radiological Society of New York State, the Aerospace Medical Association, the American Academy of Physical Medicine and Rehabilitation, the American Academy of Compensation Medicine, Inc, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Barnet Irving Romm, MD, Brooklyn. Died December 26, 1984; age 79. Dr Romm was a 1929 alumnus of the State University of New York Downstate Medical Center. He was a Fellow of the American College of Gastroenterology, and a member of the American Gastro-Enterological Association, the American Geriatrics Society, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Seymour Schwalben, MD, Glendale. Died November 16, 1984; age 63. He was a 1945 graduate of Middlesex University School of Medicine. Dr Schwalben was a Fellow of the American Academy of Family Physicians, and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Charles Alfred Shaffer, MD, Olean. Died January 20, 1985; age 77. He was a 1936 alumnus of Rush Medical College. Dr Shaffer was a member of the Medical Society of the County of Cattaraugus and the Medical Society of the State of New York.

Ned Shnayerson, MD, Riverdale. Died in August, 1984; age 85. He was a 1922 graduate of the University of Pennsylvania School of Medicine. Dr Shnayerson was a Fellow of the American College of Surgeons, and a member of the Academy of Medicine, the American Geriatrics Society, the New York County Medical Society and the Medical Society of the State of the New York.

Jose C. Sicat, MD, Staten Island. Died January 31, 1985; age 61. He was a 1954 graduate of the University of Santo Tomas Faculty of Medicine and Surgery, Manila. Dr Sicat was a Diplomate of the American Board of Anesthesiology. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Richmond County Medical Society and the Medical Society of the State of New York.

Samuel Garson Slo-Bodkin, MD, Petah-Tivka, Israel. Died January 3, 1984; age 87. He was a 1924 alumnus of the State University of New York Downstate Medical Center. Dr Slo-Bodkin was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Bernard Hooper Smith, MD, Buffalo. Died January 24, 1985; age 68. He was a 1940 alumnus of the University of Aberdeen Faculty of Medicine. Dr Smith was a Diplomate of the American Board of Psychiatry and Neurology, and a member of the American Academy of Neurology, the Association for Research in Nervous and Mental Disease, the Medical Society of the County of Erie and the Medical Society of the State of New York.

Mario H. Solis, MD, New York. Died January 28, 1985; age 52. He was a 1959 graduate of Universidad Nacional Autónoma de México Facultad de Medicina, México, Distrito Federal. Dr Solis was a Diplomate of the American Board of Radiology, and a member of the New York County Medical Society and the Medical Society of the State of New York.

Paul Dewey Steele, MD, Garden City. Died February 13, 1985; age 86. Dr Steele was a 1925 alumnus of New York University College of Medicine. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Richard Edward Sullivan, MD, Binghamton. Died February 3, 1985; age 64. He was a 1948 alumnus of the State University of New York Upstate Medical Center. Dr Sullivan was a Fellow of the American College of Surgeons, and a member of the Academy of Medicine, the Broome County Medical Society and the Medical Society of the State of New York.

Gustav Szabo, MD, New York. Died September 12, 1984; age 74. Dr Szabo was a 1935 graduate of Deutsche Universität Medizinische Fakultät, Prague. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Arthur Vigdor, MD, Miami Beach, Florida. Died December 19, 1984; age 81. Dr Vigdor was a 1932 alumnus of New York Medical College, Flower and Fifth Avenue Hospitals. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Peter P. Vitanza, MD, Lake Park, Florida. Died November 1, 1982; age 79. He was a 1935 alumnus of the University of Buffalo School of Medicine. Dr Vitanza was a member of the American Academy of Family Physicians, the Medical Society of the County of Chautauqua and the Medical Society of the State of New York.

Walter F. Watton, MD, Falls Church, Virginia. Died January 27, 1984; age 95. Dr Watton was a 1916 alumnus of the University of Michigan Medical School. He was a Diplomate of the American Board of Pediatrics, and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Chester H. Whitney, MD, New York. Died December 9, 1984; age 78. He was a 1931 graduate of Columbia University College of Physicians and Surgeons. Dr Whitney was a Diplomate of the American Board of Internal Medicine, and a member of the New York Cardiological Society, the New York County Medical Society and the Medical Society of the State of New York.

Blandina V. Worcester, MD, Geneva. Died December 31, 1984; age 82. She was a 1927 graduate of Johns Hopkins University School of Medicine. Dr Worcester was a Diplomate of the American Board of Pediatrics. Her memberships included the Academy of Medicine, the American Academy of Pediatrics, the American Medical Women's Association, the New York County Medical Society and the Medical Society of the State of New York.

Catherine M. Zaia, MD, Schenectady. Died January 27, 1985; age 84. She was a 1929 graduate of the State University of New York Upstate Medical Center. Dr Zaia was a member of the American Academy of Family Physicians, the Medical Society of the County of Schenectady and the Medical Society of the State of New York.

Samuel Zaldin, MD, Bronx. Died February 1, 1981; age 79. He was a 1926 graduate of New York University College of Medicine. Dr Zaldin was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089–1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), an abbreviated title of no greater than 40 spaces for use as a running foot, and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear on the text. When there are six or fewer authors, all should be listed; where there are seven or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

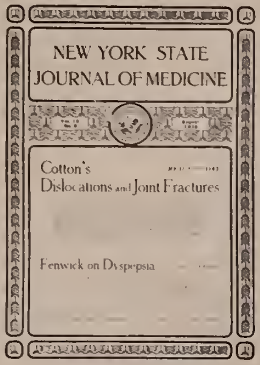
1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925-927.
2. McGuigan JE. Peptic ulcer. In: Petersdorf RG, Adams RD, Braunwald E, Isselbacher KJ, Martin JB, Wilson JD, eds. *Harrison's Principles of Internal Medicine.* New York, McGraw-Hill, 1983, pp 1697-1712.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

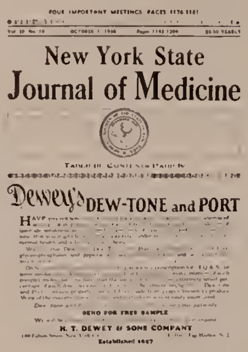
Address correspondence to Alan Blum, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

Cover to cover . . . . always good reading.


NEW YORK STATE JOURNAL OF MEDICINE

Cotton's Dislocations and Joint Fractures

Fenwick on Dyspepsia


New York State Journal of Medicine

Dewey's DEW-TONE and PORT

SEND FOR FREE SAMPLE

H. T. DEWEY & SONS COMPANY

Established 1857

NEW YORK STATE JOURNAL OF MEDICINE

JANUARY 1, 1936

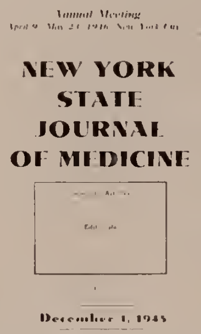
IN THIS ISSUE

TABLE OF CONTENTS PAGE 6

NEXT ANNUAL MEETING, NEW YORK CITY, APRIL 27 28 29, 1936

NEW YORK STATE JOURNAL OF MEDICINE

JANUARY 1 1938


Annual Meeting

NEW YORK STATE JOURNAL OF MEDICINE

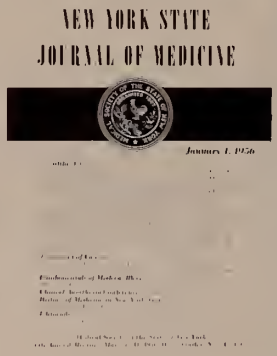
December 1, 1945

NEW YORK STATE JOURNAL OF MEDICINE

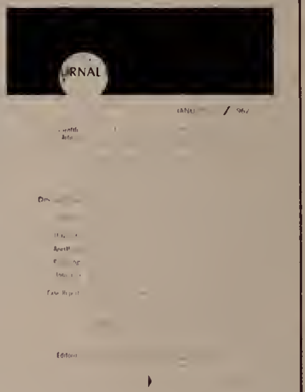
November 15, 1949

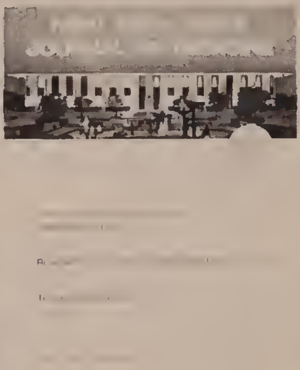
Scientific Articles

Editorials


NEW YORK STATE JOURNAL OF MEDICINE

January 1, 1956





NEW YORK STATE JOURNAL OF MEDICINE

NEW YORK STATE JOURNAL OF MEDICINE

In 1985, the *New York State Journal of Medicine* promises to provide timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news. The journal will make a welcome gift to a colleague or student in the United States or abroad.

A free copy of the December 1983 issue, a special issue on smoking, will be included with each subscription while the supply lasts.

SUBSCRIPTION ORDER FORM Clip and mail

Circulation Department
New York State Journal of Medicine
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042

Name________________________________

Address________________________________

____

	1 Year	2 Years	3 Years
U.S. & Canada	☐ $25	☐ $45	☐ $60
Foreign	☐ $30	☐ $55	☐ $75

Payment required with order.

Classified Advertising

New York State Journal of Medicine

GENERAL INFORMATION

All classified advertisements are payable in advance, except for government agencies and advertising agencies; a 15% commission to recognized advertising agencies.

Frequency: Monthly
Mailing date: 15th of the month
Closing date: 30 days before mailing date

Circulation: 27,000 (mainly physicians & medical libraries)

DISPLAY ADVERTISING

	1 time	*6 time*	*12 time*	*Width*		*Depth*
1/4-page	$450	$400	$350	3-1/2″	×	4-7/8″
1/6-page	210	190	170	2-1/8″	×	4-7/8″
1/8-page	190	170	150	3-1/2″	×	2-3/8″
1/16-page	95	85	75	3-1/2″	×	1-1/8″
1/18-page	70	60	50	2-1/8″	×	1-1/2″

PARAGRAPH ADVERTISING

The first several words are set in bold face caps.

Rates: $25.00 per insertion for 50 words or less; additional words are 30 cents each. On request, Dept. numbers will be assigned by the Journal for replies to be forwarded to the advertiser. Dept. numbers are 50 cents per insertion. There is no frequency discount for paragraph ads.

Counting Words: Two initials, each abbreviation, figures consisting of 5 numerals or less, are counted as separate words. For replies, your name and address, or telephone number should appear at the end of the ad and be counted in the total number of words.

For further information, please call the Advertising Department at 516-488-6100.

CLASSIFIED ADVERTISING ORDER FORM

New York State Journal of Medicine
Attn: Classified Advertising
420 Lakeville Rd., P.O. Box 5404
Lake Success, N.Y. 11042

Date ____________________

Name ______________________________________

Address ______________________________________

City/State/Zip ______________________________________

Payment enclosed: $____________

Number of Insertions: ____________

Assign Dept. Number: ____________

Please make check payable to New York State Journal of Medicine.

DOCTOR:

is your Medical Assistant keeping in step with you?

As medical practice becomes more complicated and more highly specialized, you need more highly trained medical assistants in your office.

Membership in the AMERICAN ASSOCIATION OF MEDICAL ASSISTANTS will help your assistants keep up-to-date and informed of new ideas and techniques. AAMA's continuing education program offers workshops and seminars that will enhance the professionalism of your office employees.

As the first professional organization for medical assistants (founded 1956), AAMA pioneered in developing the only certification program in this field. A medical assistant who successfully completes the examination is identified as a Certified Medical Assistant (CMA). More than 8,000 certificates have been earned since the first examination was given in 1963.

The AAMA pioneered in the development of curriculum standards for medical assisting programs. The American Medical Association, in collaboration with AAMA, is recognized as an official accrediting agency for such programs by the U.S. Office of Education.

On five different occasions the AMA House of Delegates has passed resolutions commending the objectives of the AAMA, endorsing its functions, and urging every physician to encourage medical assistants to join the association in order to benefit from its educational programs.

To help your medical assistants do a better job of helping you, urge them to join AAMA—the professional association dedicated to their continuing education. Fill in the attached coupon and mail it today. Your practice deserves the best!

I wish to inquire about membership for my medical assistant in the American Association of Medical Assistants, Inc. Please send more information to:

Name______________________________

Business Address______________________________

City/State/Zip______________________________

County________________Phone________________

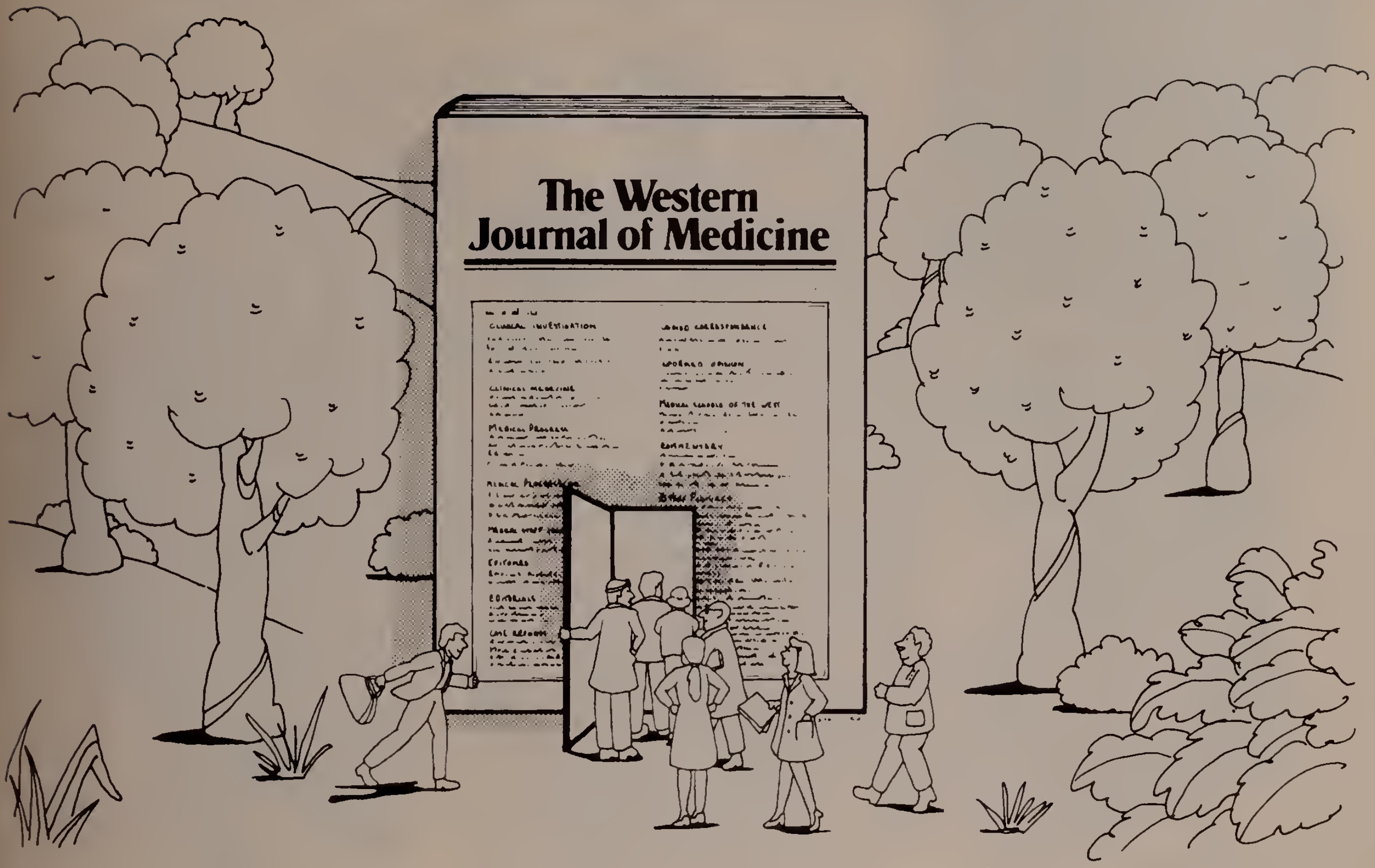
Names and Addresses of Assistants______________________________

____

____

Clip and mail to: AAMA, NYSS, Inc., c/o Department of Information, Medical Society State of New York, 420 Lakeville Road, Lake Success, New York 11042.

Inside Information


The Western Journal of Medicine

Consider a few features of **The Western Journal of Medicine:**

- **Epitomes of Progress** — Each month this section highlights the major advances in a different specialty with 15 or 20 brief epitomes by experts in the field.
- **Clinical Investigation** — Five major clinical research societies coordinate important new research findings, emphasizing clinical value.
- **Socioeconomics** — WJM is often the first to predict, examine and evaluate socioeconomic changes, trends.
- **Yearly Special Issue** — Each December's issue is devoted to a topic vital to physicians: clinical nutrition (1979), cost of care (1980), geriatrics (1981), occupational/environmental medicine (1982) cross-cultural medicine (1983), personal health maintenance (1984), high-tech medicine (1985).

The Western Journal of Medicine

Please mail this coupon with
enclosed payment to:

Circulation Department
44 Gough Street, San Francisco, California 94103

Subscriptions, 12 Issues Per Year
U.S. and Canada $20
All other countries $30

NYJ

Enclosed is $________ to cover ________ subscriptions

Subscription renewal ☐ Regular subscription ☐ Foreign subscription ☐

TO: ____________________

Street ____________________

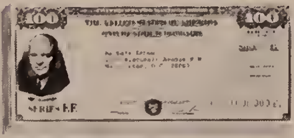
City ________ State ________ Zip ________

Specialty ____________________

Gift Card to read "From ____________________"

SOME OF THE GREATEST THINGS IN AMERICA NEVER CHANGE.

SOME DO.



Paying Better Than Ever.

Back in 1872, San Francisco's first cable cars were called "Hallidie's folly." Today, they're America's only mobile historic monument.

Some things never change.

But one great American tradition *has* changed—U.S. Savings Bonds. Now Savings Bonds pay higher variable interest rates like money market accounts. That's the kind of change anyone can appreciate.

Just hold Savings Bonds for five years and you get the new variable interest rates. Plus, you get a guaranteed return. That means you can earn a lot more, but never less than 7½%.

You can purchase Bonds at almost any financial institution. Or easier yet, through the Payroll Savings Plan where you work.

Buy Savings Bonds. Like the cable car, they're another part of our proud heritage.

For the current interest rate and more information, call toll-free 1-800-US-Bonds.

U.S. SAVINGS BONDS
Paying Better Than Ever

Variable rates apply to Bonds purchased on and after 11/1/82 and held at least 5 years. Bonds purchased before 11/1/82 earn variable rates when held beyond 10/31/87. Bonds held less than 5 years earn lower interest.

A public service of this publication.

REAL ESTATE FOR SALE OR RENT

BAY SHORE, leasing new luxurious medical building. Located near Southside Hospital. Minimum suite 740 sq. ft. T.V. Monitored hallway. Large parking area, handicapped designed. Individual gas heat, a/c-electric. (516) 665-4064, 10 A.M. to 5 P.M., Monday to Friday.

COUNTRY ESTATE: for gracious living or weekend retreat. Catskills. 100 miles from N.Y.C. 9 room ranch, large living room with fireplace, dining room, 4 bed rooms, 3 baths, superb kitchen with Jenair stove and microwave, family room, large storage cellar, separate 4 room guest house. 4 car garage, large barn, trout pond, bass pond, skiing, snowmobiling, deer, wild turkey, magnificent view. $239,500 (201) 391-4801 or (914) 351-2346.

GASTROINTESTINAL ENDOSCOPY SUITES. Rent by procedure or hour. Fully equipped with latest Olympus equipment including video. Full-time gastrointestinal assistants. "Park Avenue Brownstone" location. Endo-Center, Inc. (212) 751-0365.

WANTED: MEDICAL OFFICE BUILDINGS. As principals we are seeking "existing buildings"—fully tennanted by physicians, dentists and health care services. Contact: Paul Gellert, Gelco Associates, 155 West 68th St., N.Y., N.Y. 10023; (212) 724-7900.

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

SPACE FOR RENT in new Medical Building. Orangeburg, Rockland County. (914) 359-1877.

FOREST HILLS, Cord Meyer area, all new 7-room medical suite, two baths, available immediately, near all transportation and parking. Call owner (516) 482-1780.

OFFICE SPACE AVAILABLE, Central Bergen County, NJ. Share expenses. Call Dr. Rhine (201) 836-7722.

RIVERHEAD . . . Roanoke Avenue Professional Building, 940 Roanoke Avenue, near hospital. 470 sq. ft., previously occupied by M.D. Call owner (516) 589-8787, 8 A.M. to 5 P.M., Monday thru Friday.

DOCTOR'S HOUSE FOR SALE—Baldwin Harbor, Long Island. Spotless solid brick Split Level with 3 bedrooms, 2 baths (1 bath off master bedroom) plus maid's room. 30 ft. by 18 ft. den plus 5 room Professional Suite (including lab), with private entrance away from living quarters. $270,000, must sell immediately. Call Pfeiffer (516) 678-1131.

REAL ESTATE FOR SALE OR RENT—CONT'D

JERICHO, L.I., HOME + OFFICE. HOME: entry on fine residential street. OFFICE: entry on Broadway/Rt. 107. Contemporary decor distinguishes this residence that offers living room, dining room, bright new kitchen, big den plus family room, 3 bedrooms, 2½ baths. Spacious is the word for the 3 room + ½ bath office wing. Other features include 1/3 acre with private yard; 2 car garage; built-ins include central air, telephone intercom, etc. $385,000. Call Birch Tree (516) 496-3434.

OAKDALE SOUTH (Idlehour), Enjoy elegant rooms in this prestigious georgian colonial. French doors open to center hall with spiral staircase, formal dining room and large library with magnificent fireplace. This mint home also features a modern kitchen, sitting room off master bedroom and inground pool. Owner (516) 567-5672, $1350/mo.

FOR SALE outstanding building with 2 large offices: Nassau, N.Y.C. border. Near 3 hospitals and excellent transportation. Also well established, lucrative medical practice for sale, will introduce. Reply Dept. 390 c/o NYSJM.

MANHATTAN PIED-A-TERRE: Rent stabilized tenant seeks suburban partner for joint purchase of large studio in Parc Vendome Condominium, West 57th Street. Occupancy on time-share basis: other party would use Friday 9 A.M. through Monday 9 P.M. Permanent arrangement or progressive buy-out. Call (212) 581-1445 weeknights.

DOCTOR'S OFFICE AVAILABLE FROM JULY 1, 1985. Rockaway Park, 900 square feet, five panelled rooms, two bathrooms, space for sharing. Street level on busy boulevard, one block from ocean, subway, bus. $800.00 per month. Irene Storozynski, 115-04 Rockaway Beach Boulevard, Rockaway Park, NY 11694. (718) 474-2870.

PROFESSIONAL LOCATION on residential street. "3 fireplaces", sunken living room, dual fireplace to raised formal dining room, main level den with fireplace, 3-4 bedrooms, finished basement, garage. "Motivated seller". Century 21 Carousel of Homes, 255 Sunrise Highway, (516) 868-6620.

PHYSICIANS—rent a elegantly furnished 3½ room office suite located in a middle to high middle income area in the Bay Ridge, Bensonhurst section of Brooklyn. For information call: (718) 837-8799.

MODERN OFFICE IN ROSLYN, L I., newly furnished and equipped, time-share or rent, on major road, free parking. Close to 3 University Hospitals, suitable for any specialty. Call (718) 539-6222.

MEDICAL OFFICES AVAILABLE. Prime Downtown Manhattan Area—ground floor opposite NY Telephone Building—1 block to all subways and buses. Excellent opportunities, new family area, ideal for internists and all specialties. Call days (212) 966-2496, evenings (718) 263-5498.

FOREST HILLS, NEW YORK, Original owner, custom built office and home. Ten room house plus six rooms fully equipped office of former internist. Ideal location, accessible to subway and buses. Call after 6 P.M. (718) 268-1255.

Angina comes in many forms...

So does SORBITRATE®

(ISOSORBIDE DINITRATE)

Unsurpassed flexibility in nitrate therapy.

© 1985 ICI AMERICAS INC.

See following page for brief summary of prescribing information.

SORBITRATE
(ISOSORBIDE DINITRATE)

Please consult full prescribing information before use. A summary follows:

INDICATIONS AND USAGE: SORBITRATE (isosorbide dinitrate) is indicated for the treatment and prevention of angina pectoris All dosage forms of isosorbide dinitrate may be used prophylactically to decrease frequency and severity of anginal attacks and can be expected to decrease the need for sublingual nitroglycerin.

The sublingual and chewable forms of the drug are indicated for acute prophylaxis of angina pectoris when taken a few minutes before situations likely to provoke anginal attacks Because of a slower onset of effect, the oral forms of isosorbide dinitrate are not indicated for acute prophylaxis

CONTRAINDICATIONS: SORBITRATE is contraindicated in patients who have shown purported hypersensitivity or idiosyncrasy to it or other nitrates or nitrites Epinephrine and related compounds are ineffective in reversing the severe hypotensive events associated with overdose and are contraindicated in this situation.

WARNINGS: The benefits of SORBITRATE during the early days of an acute myocardial infarction have not been established. If one elects to use organic nitrates in early infarction, hemodynamic monitoring and frequent clinical assessment should be used because of the potential deleterious effects of hypotension.

PRECAUTIONS: General: Severe hypotensive response, particularly with upright posture, may occur with even small doses of SORBITRATE The drug should therefore be used with caution in subjects who may have blood volume depletion from diuretic therapy or in subjects who have low systolic blood pressure (eg, below 90 mmHg) Paradoxical bradycardia and increased angina pectoris may accompany nitrate-induced hypotension. Nitrate therapy may aggravate the angina caused by hypertrophic cardiomyopathy.

Marked symptomatic, orthostatic hypotension has been reported when calcium channel blockers and organic nitrates were used in combination. Dose adjustment of either class of agents may be necessary

Tolerance to this drug and cross-tolerance to other nitrates and nitrites may occur. Tolerance to the vascular and antianginal effects of isosorbide dinitrate or nitroglycerin has been demonstrated in clinical trials, experience through occupational exposure, and in isolated tissue experiments in the laboratory The importance of tolerance to the appropriate use of isosorbide dinitrate in the management of patients with angina pectoris has not been determined However, one clinical trial using treadmill exercise tolerance (as an end point) found an 8-hour duration of action of oral isosorbide dinitrate following the first dose (after a 2-week placebo washout) and only a 2-hour duration of effect of the same dose after 1 week of repetitive dosing at conventional dosing intervals On the other hand, several trials have been able to differentiate isosorbide dinitrate from placebo after 4 weeks of therapy and, in open trials, an effect seems detectable for as long as several months

Tolerance clearly occurs in industrial workers continuously exposed to nitroglycerin. Moreover, physical dependence also occurs since chest pain, acute myocardial infarction, and even sudden death have occurred during temporary withdrawal of nitroglycerin from the workers In clinical trials in angina patients, there are reports of anginal attacks being more easily provoked and of rebound in the hemodynamic effects soon after nitrate withdrawal The relative importance of these observations to the routine, clinical use of isosorbide dinitrate is not known. However, it seems prudent to gradually withdraw patients from isosorbide dinitrate when the therapy is being terminated, rather than stopping the drug abruptly

Information for Patients: Headache may occur during initial therapy with SORBITRATE Headache is usually relieved by the use of standard headache remedies or by lowering the dose and tends to disappear after the first week or two of use

Drug Interactions: Alcohol may enhance any marked sensitivity to the hypotensive effect of nitrates.

Isosorbide dinitrate acts directly on vascular smooth muscle; therefore, any other agent that depends on vascular smooth muscle as the final common path can be expected to have decreased or increased effect depending on the agent.

Carcinogenesis, Mutagenesis, Impairment of Fertility: No long-term studies in animals have been performed to evaluate the carcinogenic potential of this drug A modified two-litter reproduction study in rats fed isosorbide dinitrate at 25 or 100 mg/kg/day did not reveal any effects on fertility or gestation or any remarkable gross pathology in any parent or offspring fed isosorbide dinitrate as compared with rats fed a basal-controlled diet.

Pregnancy Category C: Isosorbide dinitrate has been shown to cause a dose-related increase in embryotoxicity (increase in mummified pups) in rabbits at oral doses 35 and 150 times the maximum recommended human daily dose There are no adequate and well-controlled studies in pregnant women SORBITRATE should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: It is not known whether this drug is excreted in human milk Because many drugs are excreted in human milk, caution should be exercised when SORBITRATE is administered to a nursing woman

Pediatric Use: The safety and effectiveness of SORBITRATE in children has not been established

ADVERSE REACTIONS: Adverse reactions, particularly headache and hypotension, are dose-related In clinical trials at various doses, the following have been observed

Headache is the most common (reported incidence varies widely, apparently being dose-related, with an average occurrence of about 25%) adverse reaction and may be severe and persistent Cutaneous vasodilation with flushing may occur Transient episodes of dizziness and weakness, as well as other signs of cerebral ischemia associated with postural hypotension, may occasionally develop (the incidence of reported symptomatic hypotension ranges from 2% to 36%). An occasional individual will exhibit marked sensitivity to the hypotensive effects of nitrates and severe responses (nausea, vomiting, weakness, restlessness, pallor, perspiration, and collapse) may occur even with the usual therapeutic dose. Drug rash and/or exfoliative dermatitis may occasionally occur Nausea and vomiting appear to be uncommon. Case reports of clinically significant methemoglobinemia are rare at conventional doses of organic nitrates. The formation of methemoglobin is dose-related and, in the case of genetic abnormalities of hemoglobin that favor methemoglobin formation, even conventional doses of organic nitrate could produce harmful concentrations of methemoglobin.

DOSAGE AND ADMINISTRATION: For the treatment of angina pectoris, the usual starting dose for sublingual SORBITRATE is 2.5 to 5 mg, for chewable tablets, 5 mg; for oral (swallowed) tablets, 5 to 20 mg, and for controlled-release forms, 40 mg

SORBITRATE should be titrated upward until angina is relieved or side effects limit the dose In ambulatory patients, the magnitude of the incremental dose increase should be guided by measurements of standing blood pressure

The initial dosage of sublingual or chewable SORBITRATE for prophylactic therapy in angina pectoris patients is generally 5 or 10 mg every 2 to 3 hours Adequate controlled clinical studies demonstrating the effectiveness of chronic maintenance therapy with these dosage forms have not been reported.

SORBITRATE in oral doses of 10 to 40 mg given every 6 hours or in oral controlled-release doses of 40 to 80 mg given every 8 to 12 hours is generally recommended. The extent to which development of tolerance should modify the dosage program has not been defined *The oral controlled-release forms of isosorbide dinitrate should not be chewed*

DOSAGE FORMS AVAILABLE: Sublingual Tablets (2 5, 5, 10 mg), Chewable Tablets (5, 10 mg), Oral Tablets (5, 10, 20, 30, 40 mg); Sustained Action Tablets (40 mg)

STUART PHARMACEUTICALS
Division of ICI Americas Inc.
Wilmington, DE 19897

STR-2282

continued from p 8A

AROUND THE NATION

ARIZONA

Aug 26–28. **Tutorials in the Tetons: Clinical Cardiology, Diagnostic and Therapeutic Advances.** Phoenix. Info: ACC, Extramural Programs Dept, 9111 Old Georgetown Rd, Bethesda, MD 20814. Tel: (301) 897-5400 ext 230.

CALIFORNIA

July 31–Aug 2. **Interventional Radiology: An Update for Radiologic Technologists and Nurses.** San Francisco. Info: Dept of Radiology, University of California, San Francisco, CA 94143.

Aug 12–16. **Wilderness Medicine.** 23 Cat 1 credits. Lake Tahoe. Info: CME M-017 University of California San Diego School of Medicine, La Jolla, CA 92093. Tel: (619) 452-3940.

Aug 17–23. **5th Annual Symposium on Fine Needle Aspiration.** Santa Barbara. Info: Extended Programs in Medical Education, Room 569-U, University of California, San Francisco, CA 94143. Tel: (415) 666-4251.

COLORADO

Aug 8–13. **Primary Care Orthopedics and Care of the Back.** Aspen. Info: University of Colorado School of Medicine, Postgraduate Medical Education, 4200 East 9th Ave, Box C-295, Denver, CO 80262. Tel: (303) 394-5241.

DISTRICT OF COLUMBIA

Aug. 18–24. **Health Care Expo ’85.** Info: Steven K. Herlitz, Inc, 404 Park Ave S, New York, NY 10016. Tel: (212) 532-9400/(800) 221-3987.

FLORIDA

Aug 8–10. **Controversies in Carcinoma of the Breast.** 16 Cat 1 credits. Orlando. Info: Dept of Radiology, University of South Florida, College of Medicine, 12901 N 30th St, Box 17, Tampa, FL 33612. Tel: (813) 974-2538.

continued on p 17A

continued from p 16A

MARYLAND

Aug 23–24. **Training for Health: Prevention and Treatment of Common Athletic Injuries.** Baltimore. Info: Trieber Associates, Inc, 9407 Jodale Rd, Randallstown, MD 21133. Tel: (301) 655-5062.

MASSACHUSETTS

Aug. 2–4. **The Cape Cod Conference on Pediatrics.** 20 Cat 1 credits. Hyannis. Info: Rayna Saville, Pediatric CME, Children's Hospital of Buffalo, 219 Bryant St, Buffalo, NY 14222. Tel: (716) 878-7630.

VIRGINIA

Aug 15–17. **Third Annual Review of Practical Dermatology for the Primary Care Physician—Controversies in Clinical Care.** Virginia Beach. 14 Cat 1 credits. Info: Eastern Virginia Medical School CME, POB 1980, Norfolk, VA 23501. Tel: (804) 446-5243.

WEST VIRGINIA

Aug 22–24. **Greenbrier Conference on Pulmonary Care.** White Sulphur Springs. Info: Kathi Elkins, Greenbrier Conference, POB 3980, Charleston, WV 25339. Tel: (304) 342-6600.

AROUND THE WORLD

AUSTRIA

Aug 31–Sept 5. **XVI Congress International Academy of Pathology and 7th World Congress of Academic and Environmental Pathology.** Vienna. Info: Congress Office, Institute for Pathologische Anatomie 4, Spitalgasse A-1090, Vienna, Austria.

BELGIUM

Aug 25–30. **Infant Nutrition and Gastrointestinal Disease.** Brussels. Info: The International Institute for Infant Nutrition and Gastrointestinal Disease, Children's Hospital of Buffalo, 219 Bryant St, Buffalo, NY 14222. Tel: (716) 878-7630.

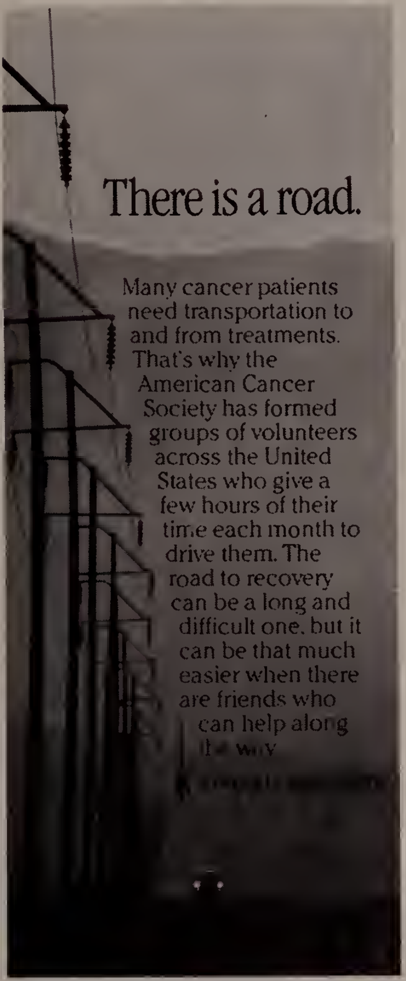
CANADA

Aug 3–10. **Effective Diagnosis and Treatment of Infectious Diseases.** 24 Cat 1 credits. Manitoba. Info: Kathy Rairigh, CME, Epic Medical Expeditions, POB 209, Sun Valley, ID, 83353. Tel: (208) 788-4995.

Aug 10–16. **10th International Congress of Hypnosis and Psychosomatic Disease.** Toronto. 28 Cat 1 credits. Info: International Society of Hypnosis, Pennsylvania Hospital, The Institute, 111 North 49th St, Philadelphia, PA 19139-2798.

HUNGARY

Aug 21–27. **14th International Cancer Congress.** Budapest. Info: Congress Bureau MOTESZ Budapest, H-1361, POB 32, Hungary. Tel: 36, (1) 125-012.


There is a road.

Many cancer patients need transportation to and from treatments. That's why the American Cancer Society has formed groups of volunteers across the United States who give a few hours of their time each month to drive them. The road to recovery can be a long and difficult one, but it can be that much easier when there are friends who can help along the way.

[illegible]

This space contributed as a public service.

DON'T COUNT ON THIS ALONE TO SAVE YOUR LIFE.

First-aid kits are fine for some emergencies. But what if you were choking? Having a heart attack? Or a stroke? This kit would be useless. Call the Red Cross. We'll teach you and your employees how to save a life. From work-safety to CPR, you can count on the Red Cross.

We'll help. Will you?

A Public Service of This Publication. Ad Council

Right from the start
in hypertension…

Once-daily INDERAL LA (propranolol HCl) for smooth blood pressure control without the potassium problems of diuretics

Once-daily INDERAL LA (propranolol HCl) avoids the risk of diuretic-induced ECG abnormalities due to hypokalemia.[1,2] In addition, INDERAL LA preserves potassium balance without additive agents or supplements while providing simple, well-tolerated therapy with broad cardiovascular benefits.

Once-daily INDERAL LA for the cardiovascular benefits of the world's leading beta blocker

Simply start with 80 mg once daily. Dosage may be increased to 120 mg to 160 mg once daily as needed to achieve additional control.

Like conventional INDERAL tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.

For beta-1/beta-2 blockade

Once-daily

INDERAL LA

(PROPRANOLOL HCl)

80 mg 120 mg 160 mg

The appearance of these capsules is a registered trademark of Ayerst Laboratories

Please see brief summary of prescribing information on the next page for further details.

Once-daily

For beta-1/beta-2 blockade INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES

The appearance of these capsules is a registered trademark of Ayerst Laboratories

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*
INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**
DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules
CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use Effects on plasma volume appear to be minor and somewhat variable INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential The significance of the membrane action in the treatment of arrhythmias is uncertain

The mechanism of the antimigraine effect of propranolol has not been established Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital. For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm

Propranolol is not significantly dialyzable
INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary
CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL
WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes In these patients, it may be more difficult to adjust the dosage of insulin

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case this resulted after an initial dose of 5 mg propranolol
PRECAUTIONS. *General* Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure

Clinical Laboratory Tests Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy Pregnancy Category C INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman

Pediatric Use. Safety and effectiveness in children have not been established
ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular bradycardia, congestive heart failure, intensification of AV block, hypotension; paresthesia of hands, thrombocytopenic purpura arterial insufficiency, usually of the Raynaud type.

Central Nervous System lightheadedness, mental depression manifested by insomnia, lassitude, weakness, fatigue, reversible mental depression progressing to catatonia visual disturbances, hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics

Gastrointestinal nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory bronchospasm

Hematologic agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous. alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol
DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION—*Dosage must be individualized* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic The dosage may be increased to 120 mg once daily or higher until adequate blood pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily
PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

REFERENCES

1. Holland OB, Nixon JV, Kuhnert L: Diuretic-induced ventricular ectopic activity *Am J Med* 1981;70:762-768. **2.** Holme I, Helgeland A, Hjermann I, et al: Treatment of mild hypertension with diuretics. The importance of ECG abnormalities in the Oslo study and in MRFIT *JAMA* 1984,251 1298-1299

9411/1184

Ayerst®
AYERST LABORATORIES
New York, N Y 10017

Copyright © 1984 AYERST LABORATORIES
Division of AMERICAN HOME PRODUCTS CORPORATION

CHIEF EXECUTIVE OFFICER

The Medical Society of the State of New York invites applications for the position of Executive Vice-President. The EVP is the Chief Executive Officer and directs the activities of the 27,000 member Society, located at Lake Success, N.Y.

Non-Physicians may apply. Salary and benefits negotiable. All replies will be considered in confidence.

Send Curriculum Vitae to:
Chairman, EVP Search Committee

Medical Society of the
State of New York
420 Lakeville Road
Lake Success, NY 11042

Index to Advertisers

Ayerst Laboratories 18A,19A,20A
Chemical Bank .... 2nd Cover
Classified Advertising 5A,6A,14A,21A
Knoll Pharmaceuticals 2A,3A,4A
Eli Lilly and Co. .... 3rd Cover
Marion Laboratories 9A,10A
Medical Quadrangle 6A
Stuart Pharmaceuticals 15A,16A
Upjohn Company .... 4th cover
U.S. Army Reserve 22A
Winthrop Breon 7A,8A

Need A Temporary Physician?
During: vacations, CME's, illness, recruiting.
Want Free Time While You Practice Medicine?
Join CompHealth's Locum Tenens Physician Group.
For further information, call 412-741-3310.

CompHealth A Physician Group
WILSON ROSS, Regional Administrator
114 Centennial Ave., Sewickley, PA 15143

NATIONAL EMERGENCY SERVICES, INC. (N.E.S.)

Directorships, full- and part-time emergency department opportunities are available throughout New York State and the country for primary care physicians with ER experience. Enjoy a variety of practice settings, competitive remuneration, paid malpractice insurance, and flexible scheduling. For information and application materials contact the recruitment department at (516) 365-4010, (Outside N.Y. State 1-800-645-4848) or send C.V. to N.E.S., One Hollow Lane, Suite 304, Lake Success, N.Y. 11042.

EMR

Emergency Medicine Review

A Comprehensive Continuing Education Course in Self-Study Format

The Emergency Medicine Review Self-Study Course was developed with the active emergency department professional in mind. Patterned after a six-day course presented at the University of Nebraska Medical Center, the series provides an indepth review of Emergency Medicine.

The Emergency Medicine Review Self-Study Course includes:

- 40 lectures by University of Nebraska Medical Center faculty members. Lectures were recorded live and edited for use on audiocassettes.
- A detailed study guide to review and augment material presented during lectures.
- A comprehensive final examination for CME credit.
- Bibliographies for additional study.

For further information about the self-study course or the six-day course to be held in fall 1985, please contact Susan Streitwieser, Center for Continuing Education, University of Nebraska Medical Center, 42nd and Dewey Ave., Omaha, NE 68105. Telephone: (402) 559-4152.

Cost: self-study course — $395
six-day course — $475

SURGEONS

Have you considered the U.S. Army Reserve?

1775

☐ SERVE IN THE ARMY RESERVE MEDICAL DEPARTMENT AS A COMMISSIONED OFFICER
☐ CME PROGRAMS FUNDED BY THE U.S. ARMY
☐ FLEXIBLE HOURS AND LOCATIONS AS NOT TO INTERFERE WITH YOUR PRACTICE
☐ NON-CONTRIBUTIVE RETIREMENT ANNUITY
☐ A CHANCE TO BROADEN YOUR MEDICAL EXPERTISE

CALL COLLECT
(718) 352-5850

USAR AMEDD Procurement
P.O. Box 4649
Bay Terrace, NY 11360-4649

NYMS–6

Name: ____________________

Specialty: ____________________

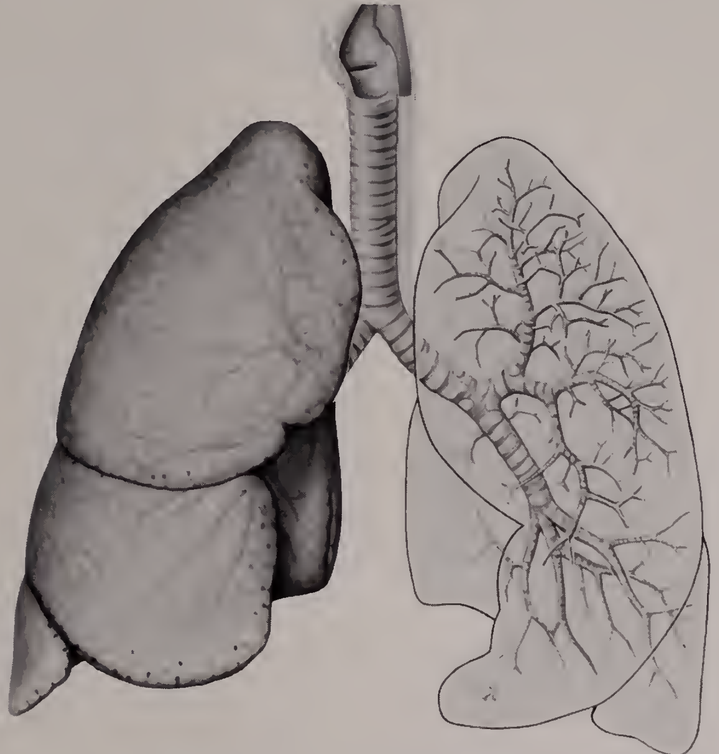
Address: ____________________

Business Phone: ____________________ Home Phone: ____________________

Best Time to Call: ____________________

COLLEGE OF PHYSICIANS OF PHILADELPHIA
3 2446 00021 8989

Consider the causative organisms...



Ceclor®
cefaclor
250-mg Pulvules® t.i.d.

offers effectiveness against the major causes of bacterial bronchitis

H. influenzae, *H. influenzae*, *S. pneumoniae*, *S. pyogenes*
(ampicillin-susceptible) (ampicillin-resistant)

Brief Summary Consult the package literature for prescribing information

Indications and Usage Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae* (*Diplococcus pneumoniae*), *Haemophilus influenzae* and *S pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor

Contraindication Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

Warnings IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins), therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics Such colitis may range in severity from mild to life-threatening

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C difficile* Other causes of colitis should be ruled out

Precautions: *General Precautions* — If an allergic reaction to Ceclor® (cefaclor, Lilly) occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e g , pressor amines, antihistamines, or corticosteroids

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms Careful observation of the patient is essential If superinfection occurs during therapy, appropriate measures should be taken

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

Ceclor should be administered with caution in the presence of markedly impaired renal function Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur This has been observed with Benedict's and Fehling's solutions and also with Clinitest* tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly)

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis

Usage in Pregnancy — Pregnancy Category B — Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor® (cefaclor, Lilly). There are, however, no adequate and well-controlled studies in pregnant women Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers — Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses Average levels were 0 18, 0 20, 0.21, and 0 16 mcg/ml at two, three, four, and five hours respectively Trace amounts were detected at one hour The effect on nursing infants is not known Caution should be exercised when Ceclor is administered to a nursing woman.

Usage in Children — Safety and effectiveness of this product for use in infants less than one month of age have not been established

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70)

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely

Hypersensitivity reactions have been reported in about 1 5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor Such reactions have been reported more frequently in children than in adults Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy No serious sequelae have been reported Antihistamines and corticosteroids appear to enhance resolution of the syndrome

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients)

Causal Relationship Uncertain — Transitory abnormalities in clinical laboratory test results have been reported Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic — Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic — Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal — Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200)

[061782R]

Note Ceclor® (cefaclor, Lilly) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information

© 1984, ELI LILLY AND COMPANY

Lilly

400441

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285

Eli Lilly Industries, Inc
Carolina, Puerto Rico 00630

Motrin®

ibuprofen, Upjohn

600 mg Tablets

More convenient for your patients.

Upjohn

© 1984 The Upjohn Company

The Upjohn Company • Kalamazoo, Michigan 49001 USA

J-4044 January 1984

STACKS

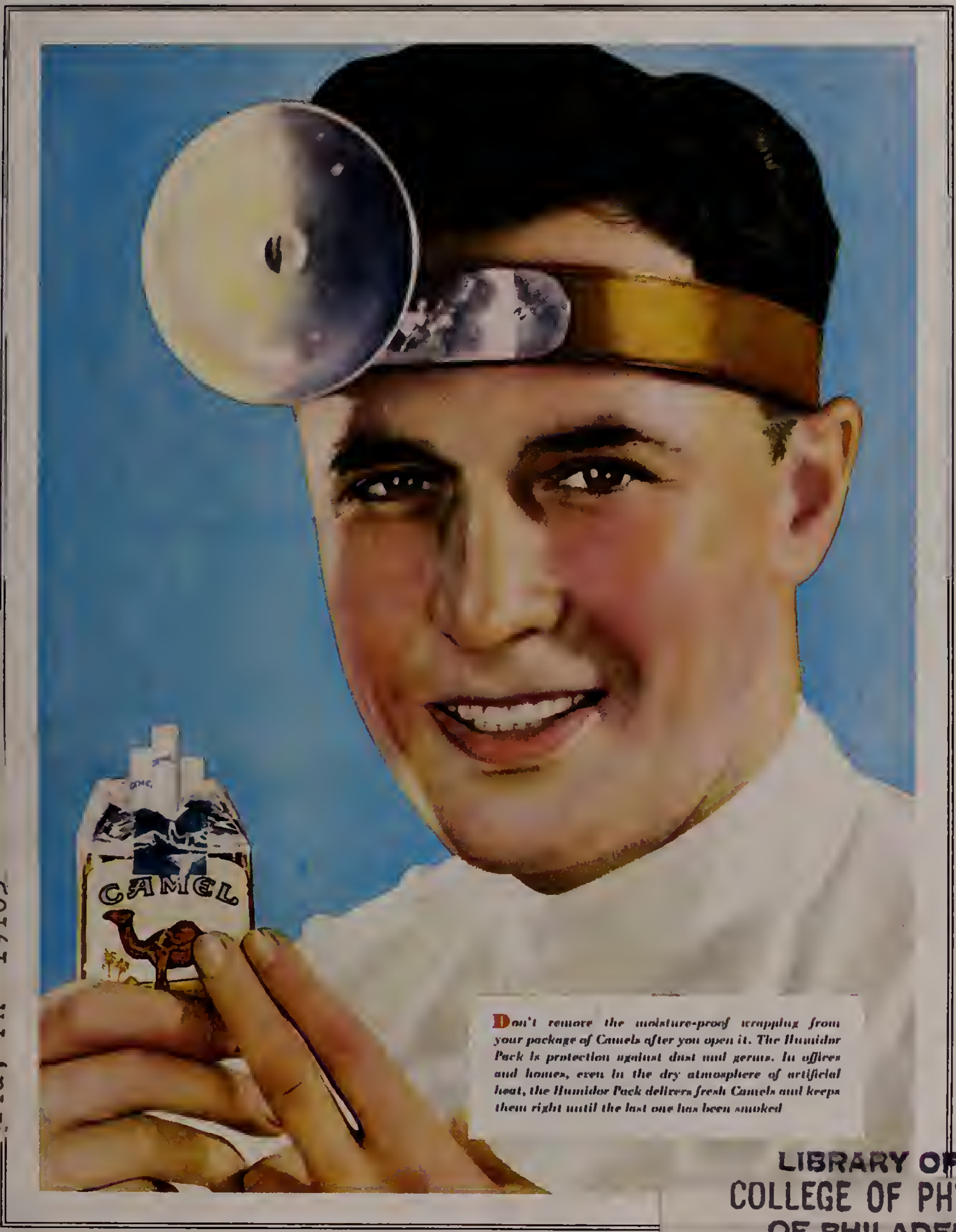
NEW YORK STATE JOURNAL OF MEDICINE

JULY 1985 • Volume 85, Number 7

Table of Contents, page 279

THE WORLD CIGARETTE PANDEMIC

Part II



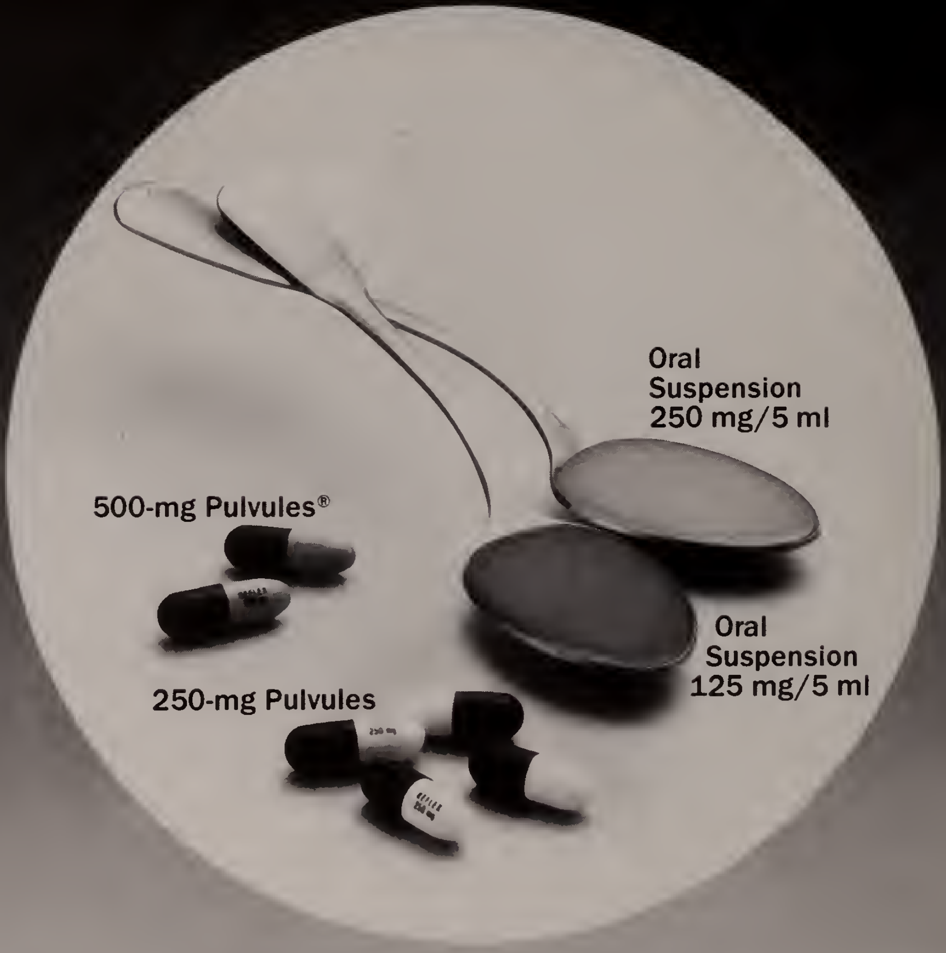
Don't remove the moisture-proof wrapping from your package of Camels after you open it. The Humidor Pack is protection against dust and germs. In offices and homes, even in the dry atmosphere of artificial heat, the Humidor Pack delivers fresh Camels and keeps them right until the last one has been smoked

Library of the College Physicia
of Philadelphia Dec. 85
19 S. 22nd St.
ila, PA 19103

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
AUG 9 - 1985

PRIMUM NON NOCERE?

Easy To Take



Keflex®
cephalexin

Additional information
available to the profession
on request.

DISTA
420113

Dista Products Company
Division of Eli Lilly and Company
Indianapolis, Indiana 46285
Mfd. by Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

NEW YORK STATE
JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
FRANK C. MAXON, JR, MD
ELIZA H. CALDWELL, MD
RICHARD B. NOLAN, MD
URSULA A. MATULONIS (Medical Student)
JOHN T. PRIOR, MD

Editor	ALAN BLUM, MD
Deputy Editor and Book Review Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Managing Editor	VICKI GLASER
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Administrative Assistant	IMELDA MORRISSEY
Editorial Assistant	JEANNE CONNERY
Librarian	ELLA ABNEY
Library Assistant	ELEANOR BURNS
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1985

RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MARK C. HOMONOFF, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. Synergistic Media Sales, 50 East 42nd St., Suite 504, New York, N.Y. 10017. Telephone: (212) 682-2200.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

DANIEL F. O'KEEFFE, MD,	*President*
ALLISON B. LANDOLT, MD,	*Past-President*
JOHN A. FINKBEINER, MD,	*President-Elect*
SAMUEL M. GELFAND, MD,	*Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
CHARLES D. SHERMAN, JR, , MD,	*Treasurer*
MORTON KURTZ, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1986	ROBERT A. MAYERS, MD, Westchester THOMAS D. PEMRICK, MD, Rensselaer RALPH E. SCHLOSSMAN, MD, Queens STANLEY A. STECKLER, MD, Suffolk ROBERT J. NEWBORN, Albany *(Councilor representing the medical student membership)*
Term Expires 1987	DAVID M. BENFORD, MD, New York DUANE M. CADY, MD, Onondaga JAMES M. FLANAGAN, MD, Wayne M. THEODORE TANENHAUS, MD, Brooklyn
Term Expires 1988	JAMES H. COSGRIFF, JR, MD, Erie RICHARD A. HUGHES, MD, Warren ANTONIO F. LASORTE, MD, Broome SIDNEY MISHKIN, MD, Nassau

Trustees

G. REHMI DENTON, MD, Albany, *Chairman*
RALPH M. SCHWARTZ, MD, New York
EDGAR P. BERRY, MD, New York
RICHARD D. EBERLE, MD, Onondaga
JOSEPH F. SHANAPHY, MD, Richmond
VICTOR J. TOFANY, MD, Monroe
BERNARD J. PISANI, MD, New York

Executive Vice-President	GEORGE J. LAWRENCE, JR, MD
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Associate	IRMA A. ERICKSON
Director, Division of Scientific Publications	ALAN BLUM, MD
Director, Division of Management Services	GEORGE FORREST, JR
Assistant Director, Division of Governmental Affairs	JERRY S. HOFFMAN, ESQ
Director, Division of Communications	EDWARD HYNES
Director, Division of Insurance and Membership Benefits	BERNARD M. JACKSON
Director, Division of Membership	EUNICE SKELLY
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Scientific Activities	PETER B. FARNSWORTH, MD
Director, Division of Advertising-Sales and Meeting Services	ROBERT MILLER
General Counsel	DONALD R. MOY, JD
Director, Division of Computer Services	IVAN H. NAJMAN
Acting Director, Division of Medical Services	MARIO MENGHINI
Director, Division of Long-Range Planning	DEBORAH R. SIEGEL
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. Editorial material published in the *Journal* is covered by copyright. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

Right from the start in hypertension...

Once-daily INDERAL LA (propranolol HCl) for smooth blood pressure control without the potassium problems of diuretics

Once-daily INDERAL LA (propranolol HCl) avoids the risk of diuretic-induced ECG abnormalities due to hypokalemia.[1,2] In addition, INDERAL LA preserves potassium balance without additive agents or supplements while providing simple, well-tolerated therapy with broad cardiovascular benefits.

Once-daily INDERAL LA for the cardiovascular benefits of the world's leading beta blocker

Simply start with 80 mg once daily. Dosage may be increased to 120 mg to 160 mg once daily as needed to achieve additional control.

Like conventional INDERAL tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.

For beta-1/beta-2
blockade

Once-daily
INDERAL LA
(PROPRANOLOL HCl) LONG ACTING CAPSULES

80 mg 120 mg 160 mg

The appearance of these capsules is a registered trademark of Ayerst Laboratories.

Please see brief summary of prescribing information on the next page for further details.

Once-daily

For beta-1/beta-2 blockade INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION SEE PACKAGE CIRCULAR)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use Effects on plasma volume appear to be minor and somewhat variable INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential. The significance of the membrane action in the treatment of arrhythmias is uncertain

The mechanism of the antimigraine effect of propranolol has not been established Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm

Propranolol is not significantly dialyzable

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

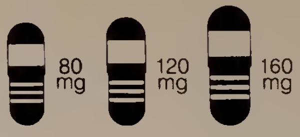
WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure if necessary they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE continued use of beta blockers can, in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure the patient should be digitalized and/or treated with diuretics, and the response observed closely or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)— PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures



The appearance of these capsules is a registered trademark of Ayerst Laboratories

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents e g dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes In these patients it may be more difficult to adjust the dosage of insulin

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General.* Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure

Clinical Laboratory Tests Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy Pregnancy Category C INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose

There are no adequate and well-controlled studies in pregnant women INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular. bradycardia, congestive heart failure, intensification of AV block, hypotension; paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type

Central Nervous System lightheadedness, mental depression manifested by insomnia lassitude, weakness, fatigue, reversible mental depression progressing to catatonia, visual disturbances, hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics

Gastrointestinal. nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory: bronchospasm

Hematologic: agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous: alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION—*Dosage must be individualized* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic The dosage may be increased to 120 mg once daily or higher until adequate blood pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

REFERENCES

1. Holland OB, Nixon JV, Kuhnert L. Diuretic-induced ventricular ectopic activity *Am J Med* 1981,70:762-768. **2.** Holme I, Helgeland A, Hjermann I, et al: Treatment of mild hypertension with diuretics The importance of ECG abnormalities in the Oslo study and in MRFIT. *JAMA* 1984,251:1298-1299

9411/1184

Ayerst®
AYERST LABORATORIES
New York, N Y 10017

Copyright © 1984 AYERST LABORATORIES
Division of AMERICAN HOME PRODUCTS CORPORATION

EXCLUSIVE

NEWS FOR NEW YORK

TALWIN® Nx

Ⓒ IV Each tablet contains pentazocine HCl, USP, equivalent to 50 mg base and naloxone HCl, USP, equivalent to 0.5 mg base.

NOW APPROVED FOR MEDICAID

T-540 NDC 0024-1951-04
100 tablets
Ⓒ IV
Talwin Nx
Each tablet contains pentazocine hydrochloride, USP, equivalent to 50 mg base and naloxone hydrochloride, USP, 0.5 mg
Caution: Federal law prohibits dispensing without prescription.
Winthrop

Talwin® Nx

Ⓒ IV Each tablet contains pentazocine HCl, USP, equivalent to 50 mg base and naloxone HCl, USP, equivalent to 0.5 mg base.

Major Analgesic Gains Reimbursement Status

Albany—TALWIN Nx has recently been included on the New York State Medicaid list of reimbursable drugs.

Physicians can now make available to their Medicaid patients the many advantages of TALWIN Nx for moderate to severe pain conditions such as fractures, acute lumbosacral pain, severe sprains, joint pain, and acute trauma.

The newly listed TALWIN Nx is characterized by dependable efficacy and a proven record of safety.[1] One tablet provides pain relief equivalent to one grain of codeine, yet allows patients to function.* TALWIN Nx offers a 15 to 30 minute onset of action, and is logical therapy for persistent pain because tolerance buildup is rare. The product is especially appropriate for many elderly patients since it has a low risk of constipation.†

---

*Since sedation, dizziness, and occasional euphoria have been noted, ambulatory patients should be warned not to operate machinery, drive cars, or unnecessarily expose themselves to hazards.

†See important product information concerning contraindications, warnings, adverse reactions, patient selection, and prescribing and precautionary recommendations on following page.

[1] Data from the Boston Collaborative Drug Surveillance Program, July 1984.

TALWIN Nx now approved for the Medicaid Drug List by New York State.

Winthrop-Breon

© 1985 Winthrop-Breon Laboratories

Talwin® Nx ⓒIV

Each tablet contains pentazocine HCl, USP equivalent to 50 mg base and naloxone HCl, USP, equivalent to 0.5 mg base.

Analgesic for Oral Use Only

Contraindications: Hypersensitivity to either pentazocine or naloxone.

TALWIN® Nx is intended for oral use only. Severe, potentially lethal, reactions may result from misuse of TALWIN® Nx by injection either alone or in combination with other substances (See **Drug Abuse and Dependence** section.)

Warnings: *Drug Dependence*. Can cause physical and psychological dependence. (See **Drug Abuse and Dependence**) *Head Injury and Increased Intracranial Pressure*. As with other potent analgesics, respiratory depressant effects of the drug may elevate cerebrospinal fluid pressure due to CO_2 retention; these effects may be markedly exaggerated in the presence of head injury, other intracranial lesions, or a preexisting increase in intracranial pressure. Can obscure the clinical course of patients with head injuries; in such patients, use with extreme caution and only if deemed essential. *Usage with Alcohol*. Due to potential for increased CNS depressant effects, alcohol should be used with caution. *Patients Receiving Narcotics*. Pentazocine is a mild narcotic antagonist. Withdrawal symptoms have occurred in patients previously given narcotics, including methadone. *Certain Respiratory Conditions*. Should be administered with caution in respiratory depression from any cause, severely limited respiratory reserve, severe bronchial asthma and other obstructive respiratory conditions, or cyanosis. *Acute CNS Manifestations*. Therapeutic doses have resulted in hallucinations (usually visual), disorientation, and confusion which have cleared spontaneously within hours. Such patients should be closely observed and vital signs checked. Reinstitution of the drug should be done cautiously since acute CNS manifestations may recur.
Precautions: *CNS Effect*. Use cautiously in patients prone to seizures; seizures have occurred though no cause and effect relationship has been established. *Impaired Renal or Hepatic Function*. Decreased metabolism of pentazocine in extensive liver disease may predispose to accentuation of side effects; it should be administered with caution in renal or hepatic impairment. In long-term use, precautions should be taken to avoid increases in dose by the patient. *Biliary Surgery*. Some evidence suggests that unlike other narcotics pentazocine causes little or no elevation in biliary tract pressures; the clinical significance of these findings is not yet known. *Information for Patients*. Since sedation, dizziness, and occasional euphoria have been noted, ambulatory patients should be warned not to operate machinery, drive cars, or unnecessarily expose themselves to hazards. May cause physical and psychological dependence taken alone and may have additive CNS depressant properties in combination with alcohol or other CNS depressants. *Myocardial Infarction*. Use with caution in patients with myocardial infarction who have nausea or vomiting. *Drug Interactions*. *Usage with Alcohol:* See **Warnings.** *Carcinogenesis, Mutagenesis, Impairment of Fertility*. No long-term studies in animals to test for carcinogenesis have been performed. *Pregnancy Category C*. Should be given to pregnant women only if clearly needed. *Labor and Delivery*. Use with caution in women delivering premature infants. Effect on mother and fetus, duration of labor or delivery, need for forceps delivery or other intervention or resuscitation of newborn, or later growth, development, and functional maturation of the child is unknown. *Nursing Mothers*. Caution should be exercised when administered to a nursing woman. *Pediatric Use*. Safety and effectiveness in children below the age of 12 years have not been established.
Adverse Reactions: *Cardiovascular:* Hypotension, tachycardia, syncope. *Respiratory:* Rarely, respiratory depression. *Acute CNS Manifestations:* In rare instances, hallucinations (usually visual), disorientation, and confusion which have cleared spontaneously within a period of hours. Such patients should be closely observed and vital signs checked. Acute CNS manifestations may recur if the drug is reinstituted. *Other CNS Effects:* Dizziness, lightheadedness, hallucinations, sedation, euphoria, headache, confusion, disorientation; infrequently weakness, disturbed dreams, insomnia, syncope, visual blurring and focusing difficulty, depression; rarely tremor, irritability, excitement, tinnitus. *Autonomic:* Sweating; infrequently flushing; and rarely chills. *Gastrointestinal:* Nausea, vomiting, constipation, diarrhea, anorexia, rarely abdominal distress. *Allergic:* Edema of the face; dermatitis, including pruritus; flushed skin, including plethora, infrequently rash, rarely urticaria. *Ophthalmic:* Visual blurring and focusing difficulty. *Hematologic:* Depression of white blood cells (especially granulocytes), which is usually reversible, moderate transient eosinophilia. *Other:* Headache, chills, insomnia, weakness, urinary retention, paresthesia.
Drug Abuse and Dependence: *Controlled Substance*. TALWIN Nx is a Schedule IV controlled substance.
Dependence and withdrawal symptoms have been reported with orally administered pentazocine. Patients with a history of drug dependence should be under close supervision. Possible abstinence syndromes in newborns after prolonged use of pentazocine during pregnancy have been reported. In prescribing for chronic use, the physician should take precautions to avoid increases in dose by the patient. Tolerance to the analgesic effect is rarely reported; there is no long-term experience with oral use of TALWIN Nx.
The amount of naloxone present (0.5 mg per tablet) has no action when taken orally and will not interfere with the pharmacologic action of pentazocine; however, this amount of naloxone given by injection has profound antagonistic action to narcotic analgesics. TALWIN Nx has a lower potential for parenteral misuse than the previous oral pentazocine formulation, but is still subject to patient misuse and abuse by the oral route.
Severe, even lethal, consequences may result from misuse of tablets by injection either alone or in combination with other substances, such as pulmonary emboli, vascular occlusion, ulceration and abscesses, and withdrawal symptoms in narcotic dependent individuals.
Overdosage: *Treatment:* Oxygen, intravenous fluids, vasopressors, and other supportive measures should be employed as indicated. Assisted or controlled ventilation should also be considered. For respiratory depression, parenteral naloxone is a specific and effective antagonist.
Please consult full product information before prescribing

WIN 5-51296

Winthrop-Breon

Winthrop-Breon Laboratories
Division of Sterling Drug Inc.
New York, NY 10016

Classified Advertising

PHYSICIANS WANTED

NEUROSURGERY FELLOWSHIP July 1, 1985 position available in extensive 1-2 year clinical program at LIJMC, a clinical campus of the School of Medicine of the State University of New York at Stony Brook. Program includes transphenoidal surgery, microneurosurgery, and pediatric neurosurgery. 1 year of neurology of neurosurgical training preferred. Salary commensurate with experience. Liberal fringe benefits. Submit inquiries and cv to Robert Decker, M.D., Division of Neurosurgery, Long Island Jewish Medical Center, New Hyde Park, NY 11042. An equal opportunity employer.

EXCELLENT OPPORTUNITY FOR READY MADE PRACTICE for General Practice Specialist or Internist willing to do a little general practice, available immediately, in an attractive Medical Building with in-house lab and x-ray facilities. Permanent relationship very desirable. Beautiful countryside-Cortland, NY. Phone collect (607) 756-9941 or submit c.v. with references to: Donald R. Gibbs, M.D., 110 North Main Street, Cortland, NY 13045.

PRIMARY CARE PHYSICIANS—A newly established multi-specialty medical facilty is seeking Board certified primary care family practice/internal medicine physicians to staff its new Primary Care Unit, complete with full service ancillaries, and served by four (4) excellent acute care hospitals. Opportunity is unique, and the Upstate New York offers outstanding life style advantages. The position offers a competitive salary and incentives with possible options for full partnership. Direct C.V., salary requirements and availability to Mary Street Medical, P.O. Box 603, New Hartford, New york 13413.

STAFF POSITIONS for Primary Care Physicians available at HMO Clinic Settings in Miami-Ft. Lauderdale Area. Attractive Salary, benefits and settings. Call or write Health-Med Systems, Inc., 19337 N.W. 2nd Ave., Miami, Fla. 33169. Phone (305) 651-8830.

NEW YORK, NEW PALTZ—Seeking full-time experience or residency trained physicians; ACLS certified; for state of the art FEC in beautifull Mid-Hudson Valley, 80 miles from NYC. Start date is July 15, 1985. Package includes Health, Life, Disability and Malpractice Insurance. Excellent salary, attractive scheduling and potential profit sharing. Send CV/Inquiries to Gary W. Greer, M.D., RD 3 Box 44A, Cogan Station, PA., 17728: phone 1-717-494-0420.

CARDIOLOGIST—BC/BE—to organize and direct a model rural heart station-222 bed hospital. 10 internists on staff. Guaranteed income for first year. Contact Sister Mary Pierre, Administrator, Mercy Hospital of Watertown, Watertown, New York 13601. Reply with resume.

Angina comes in many forms...

So does SORBITRATE®

(ISOSORBIDE DINITRATE)

Unsurpassed flexibility in nitrate therapy.

2.5 mg 5 mg 10 mg
Sublingual Tablets

5 mg 10 mg
Chewable Tablets

5 mg 10 mg 20 mg 30 mg 40 mg
Oral "Swallow" Tablets

40 mg
Sustained Action
"Swallow" Tablets

© 1985 ICI AMERICAS INC

See following page for brief summary of prescribing information.

SORBITRATE®
(ISOSORBIDE DINITRATE)

Please consult full prescribing information before use. A summary follows:

INDICATIONS AND USAGE: SORBITRATE (isosorbide dinitrate) is indicated for the treatment and prevention of angina pectoris. All dosage forms of isosorbide dinitrate may be used prophylactically to decrease frequency and severity of anginal attacks and can be expected to decrease the need for sublingual nitroglycerin

The sublingual and chewable forms of the drug are indicated for acute prophylaxis of angina pectoris when taken a few minutes before situations likely to provoke anginal attacks. Because of a slower onset of effect, the oral forms of isosorbide dinitrate are not indicated for acute prophylaxis

CONTRAINDICATIONS: SORBITRATE is contraindicated in patients who have shown purported hypersensitivity or idiosyncrasy to it or other nitrates or nitrites. Epinephrine and related compounds are ineffective in reversing the severe hypotensive events associated with overdose and are contraindicated in this situation.

WARNINGS: The benefits of SORBITRATE during the early days of an acute myocardial infarction have not been established If one elects to use organic nitrates in early infarction, hemodynamic monitoring and frequent clinical assessment should be used because of the potential deleterious effects of hypotension.

PRECAUTIONS: General: Severe hypotensive response, particularly with upright posture, may occur with even small doses of SORBITRATE The drug should therefore be used with caution in subjects who may have blood volume depletion from diuretic therapy or in subjects who have low systolic blood pressure (eg, below 90 mmHg). Paradoxical bradycardia and increased angina pectoris may accompany nitrate-induced hypotension. Nitrate therapy may aggravate the angina caused by hypertrophic cardiomyopathy

Marked symptomatic, orthostatic hypotension has been reported when calcium channel blockers and organic nitrates were used in combination Dose adjustment of either class of agents may be necessary.

Tolerance to this drug and cross-tolerance to other nitrates and nitrites may occur. Tolerance to the vascular and antianginal effects of isosorbide dinitrate or nitroglycerin has been demonstrated in clinical trials, experience through occupational exposure, and in isolated tissue experiments in the laboratory. The importance of tolerance to the appropriate use of isosorbide dinitrate in the management of patients with angina pectoris has not been determined However, one clinical trial using treadmill exercise tolerance (as an end point) found an 8-hour duration of action of oral isosorbide dinitrate following the first dose (after a 2-week placebo washout) and only a 2-hour duration of effect of the same dose after 1 week of repetitive dosing at conventional dosing intervals. On the other hand, several trials have been able to differentiate isosorbide dinitrate from placebo after 4 weeks of therapy and, in open trials, an effect seems detectable for as long as several months

Tolerance clearly occurs in industrial workers continuously exposed to nitroglycerin. Moreover, physical dependence also occurs since chest pain, acute myocardial infarction, and even sudden death have occurred during temporary withdrawal of nitroglycerin from the workers In clinical trials in angina patients, there are reports of anginal attacks being more easily provoked and of rebound in the hemodynamic effects soon after nitrate withdrawal The relative importance of these observations to the routine, clinical use of isosorbide dinitrate is not known. However, it seems prudent to gradually withdraw patients from isosorbide dinitrate when the therapy is being terminated, rather than stopping the drug abruptly.

Information for Patients: Headache may occur during initial therapy with SORBITRATE Headache is usually relieved by the use of standard headache remedies or by lowering the dose and tends to disappear after the first week or two of use.

Drug Interactions: Alcohol may enhance any marked sensitivity to the hypotensive effect of nitrates.

Isosorbide dinitrate acts directly on vascular smooth muscle; therefore, any other agent that depends on vascular smooth muscle as the final common path can be expected to have decreased or increased effect depending on the agent.

Carcinogenesis, Mutagenesis, Impairment of Fertility: No long-term studies in animals have been performed to evaluate the carcinogenic potential of this drug. A modified two-litter reproduction study in rats fed isosorbide dinitrate at 25 or 100 mg/kg/day did not reveal any effects on fertility or gestation or any remarkable gross pathology in any parent or offspring fed isosorbide dinitrate as compared with rats fed a basal-controlled diet.

Pregnancy Category C: Isosorbide dinitrate has been shown to cause a dose-related increase in embryotoxicity (increase in mummified pups) in rabbits at oral doses 35 and 150 times the maximum recommended human daily dose. There are no adequate and well-controlled studies in pregnant women. SORBITRATE should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when SORBITRATE is administered to a nursing woman.

Pediatric Use: The safety and effectiveness of SORBITRATE in children has not been established

ADVERSE REACTIONS: Adverse reactions, particularly headache and hypotension, are dose-related In clinical trials at various doses, the following have been observed

Headache is the most common (reported incidence varies widely, apparently being dose-related, with an average occurrence of about 25%) adverse reaction and may be severe and persistent Cutaneous vasodilation with flushing may occur Transient episodes of dizziness and weakness, as well as other signs of cerebral ischemia associated with postural hypotension, may occasionally develop (the incidence of reported symptomatic hypotension ranges from 2% to 36%). An occasional individual will exhibit marked sensitivity to the hypotensive effects of nitrates and severe responses (nausea, vomiting, weakness, restlessness, pallor, perspiration, and collapse) may occur even with the usual therapeutic dose. Drug rash and/or exfoliative dermatitis may occasionally occur Nausea and vomiting appear to be uncommon. Case reports of clinically significant methemoglobinemia are rare at conventional doses of organic nitrates The formation of methemoglobin is dose-related and, in the case of genetic abnormalities of hemoglobin that favor methemoglobin formation, even conventional doses of organic nitrate could produce harmful concentrations of methemoglobin.

DOSAGE AND ADMINISTRATION: For the treatment of angina pectoris, the usual starting dose for sublingual SORBITRATE is 2.5 to 5 mg, for chewable tablets, 5 mg; for oral (swallowed) tablets, 5 to 20 mg, and for controlled-release forms, 40 mg

SORBITRATE should be titrated upward until angina is relieved or side effects limit the dose. In ambulatory patients, the magnitude of the incremental dose increase should be guided by measurements of standing blood pressure

The initial dosage of sublingual or chewable SORBITRATE for prophylactic therapy in angina pectoris patients is generally 5 or 10 mg every 2 to 3 hours. Adequate controlled clinical studies demonstrating the effectiveness of chronic maintenance therapy with these dosage forms have not been reported

SORBITRATE in oral doses of 10 to 40 mg given every 6 hours or in oral controlled-release doses of 40 to 80 mg given every 8 to 12 hours is generally recommended. The extent to which development of tolerance should modify the dosage program has not been defined. *The oral controlled-release forms of isosorbide dinitrate should not be chewed.*

DOSAGE FORMS AVAILABLE: Sublingual Tablets (2.5, 5, 10 mg); Chewable Tablets (5, 10 mg); Oral Tablets (5, 10, 20, 30, 40 mg), Sustained Action Tablets (40 mg)

STUART PHARMACEUTICALS
Division of ICI Americas Inc.
Wilmington, DE 19897

STR-2282

PHYSICIANS WANTED—CONT'D

PHYSICIAN—Part-time (20 hours/week) position available in Health Center of Erie County Health Department. Candidates must be licensed to practice Medicine in NYS and must be board certified or board eligible in Family Practice or Internal Medicine. Liberal Fringe Benefits. Send resume to Erie County Personnel, Room 900, 95 Franklin Street, Buffalo, New York 14202. An Equal Opportunity Employer.

ARIZONA BASED PHYSICIAN RECRUITMENT firm has opportunites Coast to Coast. "Professionals working with Professionals". Over 13 years experience. Call (602) 795-7474; or send CV to: Mitchell & Associates, Inc., 2761 N. Country Club Rd., Suite 202, Tucson, AZ 85716.

ITHACA, NY—PSYCHIATRIC PRACTICE OPPORTUNITY in small and culturally sophisticated college community. Local assets include scenic beauty and outstanding educational systems. Patient population highly receptive to psychotherapeutic intervention. New two hundred bed hospital opened, includes acute psychiatric unit. J. Hersh, M.D., 402 N. Cayuga Street, Ithaca, NY 14850, (607) 273-8505.

PRACTICES AVAILABLE

MID HUDSON VALLEY AREA: Internist after 35 years of practice wishes to retire. Office and 3 bedroom home recently aluminum sided with two car garage on corner lot close to St. Francis Hospital and Vassar Brothers Hospital. Completely equipped air conditioned office attached to house with separate entrance. Office easily adaptable to a two man practice. Reply Dept. 391 c/o NYSJM.

SELLING PRACTICES IS OUR ONLY BUSINESS—Want to maximize the return on your investment? Before you buy or sell, call for a consultation to assure the best financial and transfer terms. We guide you through the entire sales process from initial meeting to closing. Serving 100 mile radius of New York City. Countrywide Business Brokerage, Inc., 319 East 24th Street, Suite 23-G, New York, NY 10010 (212) 686-7902.

FAMILY PRACTICE AVAILABLE WITH OFFICE BUILDING: Fully equipped. Located Ballston Spa, N.Y. near Albany, N.Y. Doctor retiring after 35 years. Information from Norman Fox, 404 Broadway, Saratoga Springs, N.Y. 12866. (518) 587-7778.

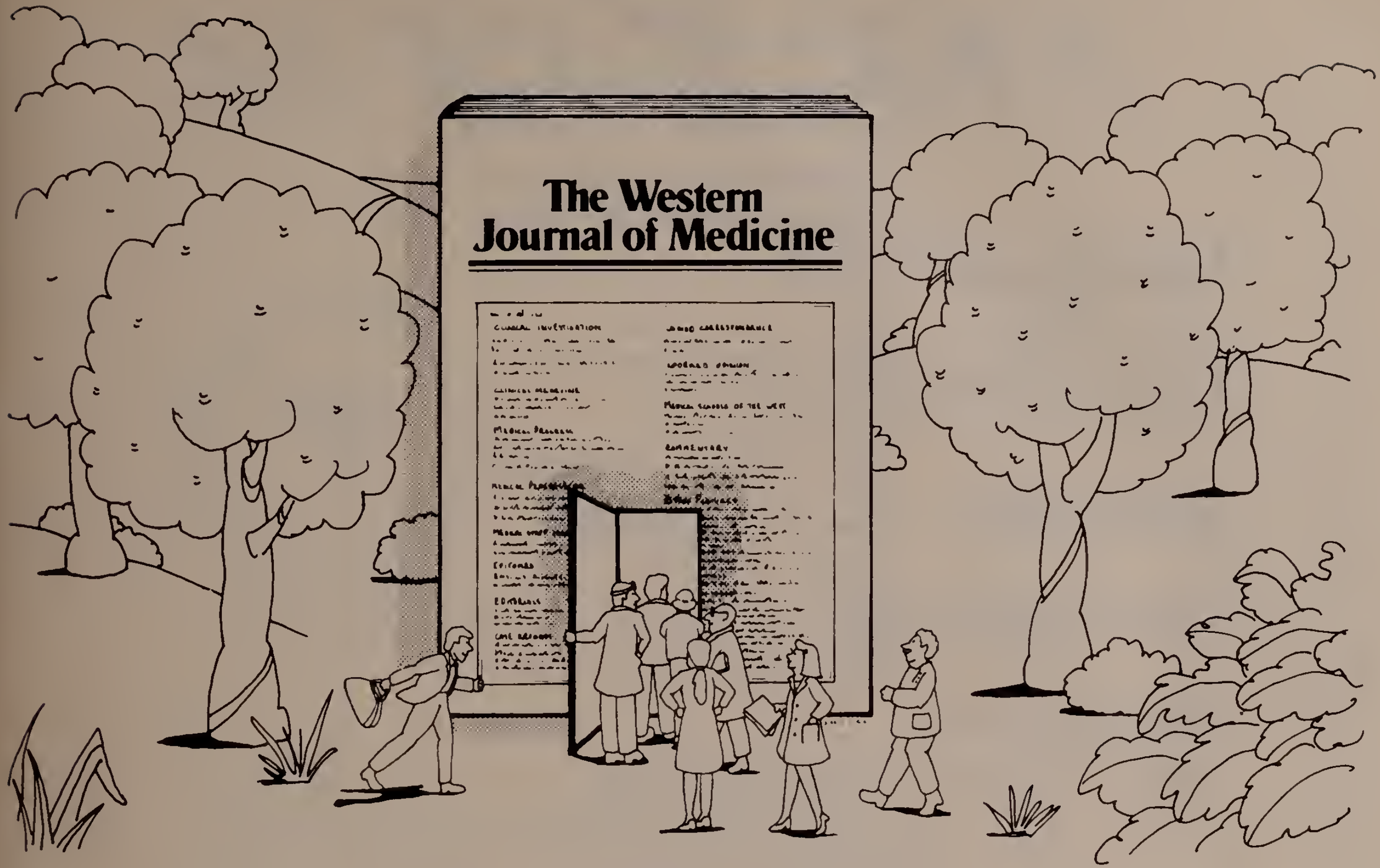
COLO-RECTAL to take practice. Retiring, 26,000 records. Hospital appointment guaranteed. Reply Dept. 392 c/o NYSJM.

MEDICAL PRACTICE SALES and Appraisals. We specialize in the valuation and selling of medical practices. If interested in buying or selling a medical practice, contact our Brokerage Division at: The Health Care Group, 400 GSB Building, Bala Cynwyd, PA 19004. (215) 667-8630.

PRACTICES WANTED

YOUNG INTERNIST, wishes to purchase Nassau County office based or nursing home based private practice. Available immediately. Strictly confidential. Reply Dept. 365, c/o NYSJM

Inside Information


The Western Journal of Medicine

Consider a few features of **The Western Journal of Medicine:**

- **Epitomes of Progress**—Each month this section highlights the major advances in a different specialty with 15 or 20 brief epitomes by experts in the field.
- **Clinical Investigation**—Five major clinical research societies coordinate important new research findings, emphasizing clinical value.
- **Socioeconomics**—WJM is often the first to predict, examine and evaluate socioeconomic changes, trends.
- **Yearly Special Issue**—Each December's issue is devoted to a topic vital to physicians: clinical nutrition (1979), cost of care (1980), geriatrics (1981), occupational/environmental medicine (1982) cross-cultural medicine (1983), personal health maintenance (1984), high-tech medicine (1985).

The Western Journal of Medicine

Please mail this coupon with
enclosed payment to

Circulation Department
44 Gough Street, San Francisco, California 94103

Subscriptions, 12 Issues Per Year
U.S. and Canada $20
All other countries $30

NYJ

Enclosed is $________ to cover ________ subscriptions.
Subscription renewal ☐ Regular subscription ☐ Foreign subscription ☐

TO: ____________________

____
Street

____
City State Zip

____

Specialty ____________________

Gift Card to read "From ____________________"

1986 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
New York Hilton, New York City

Maintaining the Quality of Health Care into the 21st Century

FRIDAY, MARCH 7

10:00 a.m. - 1:00 p.m.	Cardiovascular Diseases Gastroenterology/Alcoholism Obstetrics/Gynecology Neurosurgery Physical Medicine & Rehabilitation
2:00 p.m. - 5:00 p.m.	Neurology Pediatrics/Alcoholism Chest Diseases Medical Legal & Workers Comp./Occupational Medicine Medical Liability Mutual Insurance Co.

SATURDAY, MARCH 8

10:00 a.m. - 1:00 p.m.	Ophthalmology (All Day) Internal Medicine (All Day) Hand Surgery/Anesthesiology Urology/Dermatology Psychiatry
2:00 p.m. - 5:00 p.m.	Ophthalmology Internal Medicine Otolaryngology Hand Surgery/Anesthesiology Psychiatry

SUNDAY, MARCH 9

10:00 a.m. - 1:00 p.m.	Allergy & Immunology General Surgery Women's Medical Society of N.Y./Family Practice Pathology

□

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis.

□

Technical Exhibits

SURGEONS

Have you considered the U.S. Army Reserve?

- ☐ SERVE IN THE ARMY RESERVE MEDICAL DEPARTMENT AS A COMMISSIONED OFFICER
- ☐ CME PROGRAMS FUNDED BY THE U.S. ARMY
- ☐ FLEXIBLE HOURS AND LOCATIONS AS NOT TO INTERFERE WITH YOUR PRACTICE
- ☐ NON-CONTRIBUTIVE RETIREMENT ANNUITY
- ☐ A CHANCE TO BROADEN YOUR MEDICAL EXPERTISE

CALL COLLECT
(718) 352-5850

USAR AMEDD Procurement
P.O. Box 4649
Bay Terrace, NY 11360-4649

NYMS–7

Name: ____________________

Specialty: ____________________

Address: ____________________

Business Phone: __________ Home Phone: __________

Best Time to Call: ____________________

FOR PHYSICIANS AND SURGEONS WITH A PART-TIME EXPOSURE ONLY

Now offering Medical Malpractice Insurance on an ***occurrence basis*** at drastically reduced rates thru Frontier Insurance Co., Monticello, NY.

With the tail on claims made policies, you might lose your shirt.

Call or write for further information and application.

MEDICAL MQ QUADRANGLE

Medical Quadrangle, Inc.
P. O. Box 456
318 Bedford Road
Bedford Hills, New York 10507
914 666-0170
212 829-3654
"Specializing in Medical Liability Insurance"

MISCELLANEOUS

PROFESSIONAL MISCONDUCT ATTORNEYS. WILLIAM L. WOOD, JR., formerly Executive Director of the New York State Office of Professional Discipline and ANTHONY Z. SCHER, formerly Director of Prosecutions, announce the opening of their law office, WOOD & SCHER. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct proceedings, malpractice, licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. WOOD & SCHER, Attorneys at Law, One Chase Road, Scarsdale, New York 10583, Telephone 914-723-3500.

DOCTOR, YOU CAN'T BEAT the quality or the price! Holter Monitor Scanning Service. Physician owned, trained and supervised. No contracts to sign. We can arrange for lease/purchase of Holter equipment. New dual channel recorders with test cables $1,550. Now using UP Service for faster turn around time. DCG Interpretation (313) 879-8860.

ARCHITECTURE & PLANNING, Comprehensive services from analysis of alternative office locations through design and construction. Experienced in both new construction and renovation. Lewis & Gould (212) 807-6588.

MISCELLANEOUS—CONT'D

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuariat certification and employee statements of participation—partnership agreements and professional incorporation—No insurance required—references upon request—Jack Wachstock, PC, 3000 Marcus Ave., Lake Success, N.Y. 11530 (516) 437-4848.

50% ROI!!! AdvoCom, an advisory service, tracks more than 100 of the best futures traders—and we think we have singled out the best of all. This man is a genius—in a class by himself. 300K mimimun. Get the full story. Call 24 hours—(212) 732-0871 or write AdvoCom, POB 1809, Madison Square Station, NY, NY 10159.

MISCELLANEOUS—CONT'D

ACCOUNTING AND TAX SERVICES—Professional accounting services including preparation of individual income tax and business tax returns, consultation on all tax and accounting matters. Call anytime. Marvin J. Siegel, CPA, (718) 275-0858. If no answer leave message. References available upon request.

MEDICAL EDITOR/WRITER: Seeks freelance assignments. Experienced in clinical/socioeconomic subjects, MD manuscripts preparation, article research, presentations, graphics, copyediting, proofreading. Call (212) 861-7280.

ARCHITECTURAL DESIGN AND CONSTRUCTION: Designers, builders of custom professional offices. Consulting services available. Whether you're opening a new office, relocating, remodeling or expanding an existing office in New York City area and adjacent New Jersey counties. Donald Young, Inc. Tel: (718) 948-5191.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate, with no points, fees or charges of any kind. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

Only Ativan®
(lorazepam) Ⓒ IV

offers all these
benefits in
addition to rapid
relief of anxiety:

relief of anxiety
associated with
depressive symptoms

clearance not
significantly delayed
y age, liver or
idney dysfunction

umulative sedative
ffects seldom
problem

tle likelihood of
rug interaction
benzodiazepines produce additive
fects when taken with alcohol or
her CNS depressants)

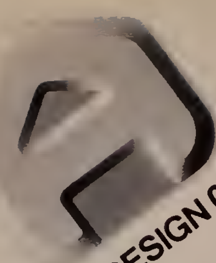
significant changes
vital signs in
rdiovascular patients*

ort duration of action,
mple metabolism

zodiazepines have not been shown to
f benefit in treating the cardiovascular
ponent.

[illegible]

Laboratories



The appearance of these tablets is a registered trademark of Wyeth Laboratories

®

DISTINCTIVE DESIGN 0.5, 1.0 and 2.0 mg

Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease Safety and effectiveness in children under 12 years have not been established

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study No studies regarding mutagenesis have been performed.

PREGNANCY Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%) Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety

Transient amnesia or memory impairment has been reported in association with the use of benzodiazepines

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P Usefulness of dialysis has not been determined

Ativan® Ⓒ IV for (lorazepam) Anxiety

DOSAGE: Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.

HOW SUPPLIED: 0.5, 1.0 and 2.0mg tablets.

Wyeth Laboratories
Philadelphia, PA 19101
TM

Medical Society of the State of New York
ANNUAL CME ASSEMBLY
MARCH 7-9, 1986
The New York Hilton, New York City
Combined With The Annual Meeting of
The House of Delegates
MARCH 6-9

MISCELLANEOUS—CONT'D

DISCIPLINARY LAW; SCIENCE LAW. BIRNBAUM & MANAKER, P.C., law firm actively engaged in science law and scientific litigation with emphasis on: defense and prosecution of medical malpractice actions; product liability suits involving medical and hospital devices and equipment; representation of physicians and other scientists in patent royalty negotiations and marketing of inventions; representation of physicians and other health care providers before state agencies, professional societies, review boards; litigation in anti-competition disputes concerning privileges, appointments and use of diagnostic facilities. Partners have extensive scientific, medical, and engineering law backgrounds. Senior partner, Irwin Birnbaum, has held law school adjunct teaching appointments, has lectured at medical schools and engineering colleges, and served as Chair of the Roscoe Pound Commission on Professional Responsibility. Partners' CV's available on request. BIRNBAUM & MANAKER, P.C., MONY Tower II, Syracuse, New York 13202, (315) 422-0246, (800) 358-8568.

REAL ESTATE FOR SALE OR RENT

BAY SHORE, Leasing new luxurious medical building. Located near Southside Hospital. Minimum suite 740 sq. ft. Large parking area, handicapped designed. Individual gas heat, a/c-electric. (516) 665-4064, 10 A.M. to 5 P.M., Monday to Friday.

WANTED: MEDICAL OFFICE BUILDINGS. As principals we are seeking "existing buildings"—fully tennanted by physicians, dentists and health care services. Contact: Paul Gellert, Gelco Associates, 155 West 68th St., N.Y., N.Y. 10023; (212) 724-7900.

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

SPACE FOR RENT in new Medical Building. Orangeburg, Rockland County. (914) 359-1877.

MEDICAL OFFICE AVAILABLE in village of Syosset on the north shore of Long Island. 1800 sq. ft., prime location on first floor, for rent with option to buy in co-op ownership. Call (516) 921-6020.

FOREST HILLS-QUEENS BLVD. Office to share, lobby floor, 24 hour doorman, central air conditioning, near all transportation. (718) 896-2344.

New Motrin® 800 mg TABLETS
ibuprofen

Extra strength
Convenience
Economy

Upjohn

The Upjohn Company
Kalamazoo, Michigan 49001 USA

© 1985 The Upjohn Company

J-5491 June 1985

Acknowledgments

My interest in cigarette smoking and its promotion was inspired by my late father, Leon Blum, MD, a general practitioner in Rockaway Beach, NY, and a member of the Medical Society of the State of New York for 37 years until his death in 1969. Dr Blum was one of the first physicians to volunteer for the front lines in the health promotion campaigns of the late Drs Alton Ochsner and Luther Terry. In the 1950s while listening to Dodger games or watching programs like *Sergeant Bilko* or *The Jack Benny Show*, he would urge me to tape record the cigarette commercials. Without evidence of this insidious propaganda, he predicted, some day no one will believe that our society could have permitted a handful of people to seduce children and others to use a costly, unattractive, and lethal product—and all at a time of great medical advances.

I deeply appreciate the patience, support, ideas, and criticism of my wife Doris Fields Blum (and our sons Leon and David) who permitted me the time needed to edit this issue. Leon, age 5, may be the youngest originator of an idea published in a medical journal: at age 3, just before his brother was born, he suggested that all parents leaving the hospital with their newborn infant be presented with a special patch that reads, "NO SMOKING OVER BABIES."

Three executive vice-presidents of the Medical Society of the State of New York under whom I have worked have fostered an environment conducive to the publication of an independent scientific journal: George J. Lawrence, Jr, MD, the late Milton Rosenberg, MD, and Edward Siegel, MD. Also owed a debt of thanks are Milton Gordon, MD, Chairman of the Committee on Publications, Library, and Archives, Pascal James Imperato, MD, Deputy Editor, and Michael Wroblewski, research assistant for this issue.

The staff of the *Journal* has been outstanding, and special thanks must go to Vicki Glaser, managing editor, Imelda Morrissey, administrative assistant, Ella Abney, librarian, Ellie Burns, associate librarian, and Jeanne Connery, editorial assistant. Invaluable assistance was given by Robert Miller of MSSNY and by Alane Fogel and the staff of Mack Printing.

This issue is dedicated to the numerous unsung physicians, other health professionals, teachers, students, and others who have devoted time and effort to counteract smoking and its promotion. These include Paul Richburg, MD, Joseph Davis, MD, Brigitte Nahmias, MD, George Gitlitz, MD, Jack Manzari, MD, Rick Richards, MD, Tom Houston, MD, Charles Tate, MD, Bob Higgins, MD, Anne Morris, MD, Lee Fairbanks, MD, David Fletcher, MD, Stuart Hanson, MD, Elizabeth Wilson, RN, Jay Lavine, MD, Gene Cotton, MD, Ron Davis, MD, Elizabeth Whelan, PHD, Joanne Luoto, MD, the late Harold Levine, MD, the late Norman Simon, MD, the late Elliott Podoll, MD, and many others, some of whom have contributed to this issue and to the December 1983 issue of the *Journal*. Journalists who have repeatedly written on this subject, including George Seldes (the father of investigative journalism), Joan Beck, Jack Mabley, Bill von Maurer, Ann Landers, Abigail van Buren, Carl Rowan, Morton Mintz, Howard Wolinsky, Charles Whited, Ellen Goodman, Joanna Wragg, Scott Ostler, Dick Young, Roy Larson, Charles Kenney, Mark Metherell, Bard Lindeman, Jon Van, Art Carlson, Cristine Russell, David Branch, Art Buchwald, and Bob Greene, are also acknowledged.

In addition to the authors of this issue and the December 1983 issue, the following persons and organizations also must be thanked for their encouragement, inspiration, or assistance: Charlie Reid, Ed Sweda, Eric Landis, Janet Terry, Regina Carlson, Kathy Weltzen, Peter Vogel, Brian Robson, William M. Gaines, Steve Woodruffe, Ira Kurzban, Prof J. Fred MacDonald, Bruce Wolf, MD, Wayne Firestone, Janette Goldberg, Phyllis Fields, Mike Sharlin, Sandy Denham, Michael Blakeney, Jill McKeough, George Melcher, MD, Charlotte Wallace, Charles Howard, Chris Hood, Don Shopland, Alan Cohen, PHD, Gary Seidler, Donna Rapp, Walt Dartland, E R W Fox, MD, Kurt Baumgartner, Tom Adams, Hank Resnick, Charles Aswad, MD, Charles Sherman, MD, Kevin Smith, Bob Moser, MD, Luke Burchard, MD, Chris Shank, MD, Brian Freedman, MD, Chris Fletcher, MD, Paul Fischer, MD, Dale Carroll, MD, Charles Derby, MD, Mark Glassner, MD, Bob Jaffe, MD, Nancy Schmitz, MD, George McClure, MD, Alan Sechtin, MD, Ferd Proano, MD, Bill Townsley, Wally Phillips, Marilyn Miller, Adam Goldstein, Sam Nixon, MD, Jason Chao, MD, Tom Paluch, MD, Scott Frank, MD, Craig White, MD, Zeke Rabkin, MD, Jeet Sandhu, Beth Goodrich, Dick Aronson, MD, Bob Jones, Brian Strickman-Levitas, Bill Beery, Julie Gerson, Courtenay Stark, James Benerofe, Susan Raskin, Cathy Popescu, Selwyn Waingrow, John Davis, MD, Stephen Nordlicht, MD, Robert Brandstetter, MD, Betty Widman, MD, Carl Aschoff, MD, Robert McAfee, MD, Joseph Tollison, MD, Brian Leaderer, PHD, Craig Worthing, Simon Nathan, Ed Donatelle, MD, Ripley Forbes, Randi Bregman, Natalie Swit, Jonas Ragnarrson, G. Snorri Ingimarsson, MD, Peter Frishauf, Sigurdur Arnason, MD, Sigurdur Bjornsson, MD, Thorvardur Ornolfsson, Dale Moquist, MD, Athena Mueller, Jo Cameron, Paula Jolly, Mickey Schaefer, Dan Ostergaard, MD, Pat Malone, Panagiotis Symbas, MD, Terry Smith, MD, Mac Macdonald, MD, Sonja Lessne, MD, Doug Everett, MD, Beth Mortensen, Rich Wilson, MD, Robert B. Howard, MD, Ed Huth, MD, Kathleen Case, Marvin Kristein, PHD, W. F. Forbes, PHD, David Simpson, Blake Cady, MD, Donna Shimp, Karen Martinsen, George Caldwell, Richard Bard, Kate O'Daniels, RN, the late Charles Kappes, Francine Sevel, Ozanna Waltz, Laurence Huber, DDS, Richard McNeely, Ruby Smith, Steven Furrow, Cpl Ed Moses, Ron Gould, Ross Winans, Ed Haskell, MD, Lynn Carmichael, MD, Matt Martin, Marianne Ronan, Lisa Langer, Mark Worden, Martin Busaldua, MD, Brent Blue, MD, Chuck Colodny, MD, Douglas Henley, MD, Joseph Scherger, MD, Phil Sloane, MD, Steven Larson, Burk Gossom, MD, Alfio Raussa, MD, Patsy Silver, H. Kenneth Walker, MD, Dewey Richards, MD, Andrew Skolnick, Gary McMahan, Bery Engebretsen, MD, Gordon DeFriese, John Stephenson, MD, Van Beth Schuler, MD, Harmon Patrick, MD, Seymour Gluck, MD, the late Gene Dayton, MD, William Barclay, MD, David West, MD, John Prior, MD, Dennis Saver, MD, Mary Lundy, Gene Wilcox, Lou Goldstein, Rick Kellerman, MD, Amy Wilson, Ross Black, MD, Stephen Brunton, MD, Kenneth Warner, PHD, Bob Devermann, MD, Herb Auer, Craig White, MD, Jim Kirby, Ted Adderton, Stephen Barrett, MD, Dick Whitten, MD, George Snell, MD, Jeanne Weigum, Ken Harvey, MB,BS, DeWayne Birkhofer, MD, Nigel Gray, MB, BS, David Hill, Ernest Lewis, MD, John Stone, MD, Edwin Dale, PHD, Mike Hartsell, MD, Chris Merz, MD, Clive Caplan, MD, Kenneth Levites, MD, Marie Shear, Robert Kohn, MD, Robert Neibling, MD, John Dowling, MD, Michael D'Andrade, Russel Thomsen, MD, Carolyn Peter, Francis Soper, William Plymat, Sr, Kirk Torney, Blake Torney, Prof Howell D. Chickering, Bob Rosner, Alexander Knisely, MD, Hal Post, Nancy Babb, Leighton Smith, MD, Gloria Cooper, Prof Carl Jensen, Harvey Shanies, MD, Sarah Ollman, George Lewis, Celia Schrader, Susanna Jaeger, Anthony DiBenedetto, George Piper, MD, Gary Kodel, MD, Dr Harry Crawley, Dr Bill Castleden, Mat Smith, MD, Norwegian Council on Smoking and Health, *World Medicine*, the Icelandic Cancer Society, the Alaska Council on Prevention of Alcohol and Drug Abuse, numerous staff members of chapters of the American Lung Association, and the Councilors, Trustees, Delegates and staff of MSSNY. —AB

ORDERING ADDITIONAL COPIES

Additional copies of this issue are available for $7.50 postpaid (foreign, add postage) from *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042, Tel: (516) 488-6100. (Reduced prices on bulk orders (foreign, add postage): 2–9 copies—$7.00 each; 10–24 copies—$6.50; 25–49 copies—$6.00; 50–99 copies—$5.50; 100–149 copies—$5.00; 150–500 copies—$4.00.)

A free copy of this issue will be sent with every new one-year subscription ($25 US + Canada, $30 foreign). Copies of *The Cigarette Underworld* (December, 1983 issue) are available for $9.95, plus $1.50 postage, from Lyle Stuart Inc, 120 Enterprise Ave, Secaucus, NJ 07094.

NEW YORK STATE JOURNAL OF MEDICINE

JULY 1985 • Volume 85, Number 7

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

THE WORLD CIGARETTE PANDEMIC-Part II

Contents

EDITORIALS

If smoking killed baby seals . . . 282

Follow-up of a cover-up . . . 285

THE STRATEGISTS

Competing agenda in smoking control agencies: *"Those who pay the piper . . ."* 287
SIMON CHAPMAN, PhD

A smoke-free society by the year 2000 290
C. EVERETT KOOP, MD

AN APPRECIATION

Luther Terry, MD, 1911–1985 293
JOSEPH A. CALIFANO, Jr

TEACHING

A disease model of cigarette use 294
JOHN D. SLADE, MD

The underemphasis on smoking in medical education 299
K. H. GINZEL, MD

ECONOMICS

Perspectives on the economic magnitude of cigarette smoking 302
JAMES M. SHULTZ, MS

An insurance company perspective on smoking 307
MICHAEL J. COWELL

TOXICOLOGY

Changes in overwrap and butt length of American filter cigarettes: An influence on reported tar yields 310
NEIL E. GRUNBERG, PhD, DAVID E. MORSE, PhD, VIRGINIA A. MAYCOCK, LYNN T. KOZLOWSKI, PhD

The "less hazardous" cigarette: a deadly delusion 313
G. H. MILLER, PhD

PRODUCT LIABILITY

Cipollone v Liggett Group, Inc: The application of theories of liability in current cigarette litigation 318
MARC Z. EDELL, ESQ,
STEWART M. GISSER, ESQ

Product liability in cigarette-caused fires 322
DONALD W. GARNER, ESQ

Cigarette marketing: ethical conservatism or corporate violence? 324
JOE B. TYE

TABLES

Diversification in the tobacco industry 328
ALAN BLUM, MD
MICHAEL WROBLEWSKI

ROLE MODELS

Mixed messages for women: A social history of cigarette smoking and advertising 335
VIRGINIA L. ERNSTER, PhD

Getting women hooked: Defending the indefensible 341
BOBBIE JACOBSON, MB, BS

Superman and the Marlboro woman: The lungs of Lois Lane 342
PAUL MAGNUS, MB, BS

Cigarette smoking and ill health among black Americans 344
RICHARD COOPER, MD,
BRIAN E. SIMMONS, MD

Pharmacists who dispense cigarettes: With reference to drug store chains and pharmaceutical companies 350
JOHN W. RICHARDS, Jr, MD,
ALAN BLUM, MD

Smoking and the military 354
GREGORY H. BLAKE, MD

Helping Youth Decide: "When the fox preaches, beware the geese" 357
W. R. COULSON, PhD, EdD

IN MEMORY

The last cigarette advertisement 359
JOYCE H. PORTER

A DIALOGUE WITH JOSEPH A. CALIFANO, JR

Obstacles to the prevention of teenage smoking 360

LEGISLATION

The Comprehensive Smoking Education Act 363
HON. HENRY A. WAXMAN

New Jersey's hard-fought battle for clean indoor air legislation: An insider's view 366
HON. MARTIN A. HERMAN

Proposition P: anatomy of a non-smokers' rights ordinance 369
PETER HANAUER, ESQ

Local values versus tobacco industry power: The experience of GASP of Massachusetts 375
EDWARD L. SWEDA, Jr, ESQ

THERAPEUTICS

An assessment of the effectiveness of nicotine chewing gum: unfulfilled expectations 378
JOSEPH FELDMAN, DrPH

INDOOR AIR QUALITY

An indoor air quality standard for ambient tobacco smoke based on carcinogenic risk 381
JAMES L. REPACE, MS, ALFRED H. LOWREY, PhD

A pilot's guide to cabin air quality and fire safety 384
H. B. FULTON, Jr

CASE REPORT

Endobronchial foreign body (cigarette filter tip) with inflammatory pseudotumor of the lung: A "True" xanthogranuloma 389
ANIL K. BATRA, MD, LLOYD REICH, MD, MARTIN BERNSTEIN, MD

INTERNATIONAL REVIEW

The clinician and smoking in Great Britain 391
DONALD J. LANE, DM

Physicians, smoking, and health in the Netherlands 394
HANS ADRIAANSE, DRS, RUUD HALFENS, DRS, MARIA J. DROP, PhD, JAN VAN REEK, DRS

Economic dependence on tobacco manufacturing in Northern Ireland: An obstacle to prevention 396
GABRIEL J. SCALLY, MB

Slow but steady progress against smoking in Ireland 400
EMER B. SHELLEY, MB

An attempt to circumvent the ban on cigarette advertising in Norway 403
ASBJØRN KJØNSTAD

Innovation in Iceland: graphic health warnings on tobacco products 405
THORSTEIN BLONDAL, MD, GUDJON MAGNUSSON, MD

Smoking in France: a dismal picture 407
RENÉE BITTOUN

Smoking and its promotion in Greece: a 20th century Trojan horse 409
ELIZABETH KEENAN

Smoking and health in the Soviet Union and Eastern Europe 413
RICHARD COOPER, MD

Israel: gaining momentum against smoking and cigarette advertising 416
CHERI M. PAPIER, MPH, MS

Cigarette smoking in Papua New Guinea: A model for keeping a people down 420
EUAN M. SCRIMGEOUR, MB, BS

Ending the social acceptability of smoking and tobacco advertising in New Zealand 422
DEIDRE KENT, BSc

Japan: land of the rise in lung cancer 425
ALAN BLUM, MD

NYJ NEWS FEATURES

Maverick doctor-politician puts pillory on tobacco and alcohol companies 428

Western Australia gives health promotion the nod 429

Medical students take action outside children's hospital 431

Medical journal editor stuns and angers health campaigners 432

BUGA-UPdate 434

MOP-UP battles tobacco sponsorship—legally 436
CALVIN MILLER, PHD

Canada's oldest daily newspaper is country's first to drop cigarette advertising 438

The Canadian Non-Smokers' Rights Association: shaming the establishment 439
ANGELA MANGIACASALE

How smoking promotes hunger 442
JOHN MADELEY

American Nurses' Association passes resolutions for action on smoking issues 444
ELIZABETH WILSON, RN, EDD

How tobacco companies have found religion 445
ALAN BLUM, MD, KATHLEEN FITZGERALD

When researchers accept funding from the tobacco industry, do ethics go up in smoke? 451
HOWARD WOLINSKY

Tobacco dilemma intensifying in North Carolina 455
LAWRENCE SPOHN

Auto racing: slaughter on tobacco road 458
ALAN BLUM, MD

LETTERS TO THE EDITOR

Tobacco related diseases among native Americans 464
LELAND L. FAIRBANKS, MD

Hospital smoking policy 464
CHARLES NORTH, MD

Registry of health facilities that fail to provide clean indoor air 465
REGINA CARLSON

Use of *Journal* in schools 465
ROBERT BASES, MD

Inappropriate magazines for the waiting room 465
PAUL FISCHER, MD

Progress against smoking in Kuwait 466
ABDUL RAHMAN AL-AWADI, MD,
HUSSAIN A. AL-MUMEN, MD

The Sudan: physicians act against cigarette advertising 467
KEITH BALL, MD

Cigarette advertising in the Third World 467
DAVID C. SOKAL, MD

The tobacco industry and legislative redress 468
AMOS HAUSNER

Chronic tobacco use confounds the diagnosis of thyroid illness 469
D. W. SEPKOVIC, PHD, N. J. HALEY, PHD,
E. L. WYNDER, MD

Ad agency conflict: selling cigarettes and dentistry 469
DOUGLAS LEE CHESTER, DDS

Smoking prevalence rates for Western Australian adolescents 470
RUTH E. SHEAN

***Death in the West* curriculum project** 470
STANTON A. GLANTZ, PHD

Attorney General Abrams speaks out against smokeless tobacco 471
HON. ROBERT ABRAMS

Toward smoke-free comic strips 472
HENRY S. KAHN, MD

BOOK REVIEWS

Preventing complications of tobacco use 474
JOHN SLADE, MD

PERIODICALS THAT REPORT ON SMOKING 477

ACKNOWLEDGMENTS/ADDITIONAL COPIES 278

NEW YORK STATE
JOURNAL OF MEDICINE

July 1985 • Volume 85, Number 7

EDITORIAL

If smoking killed baby seals . . .

This issue is a sequel to the December 1983 issue of the *New York State Journal of Medicine,* which was devoted to a consideration of aspects of the world cigarette pandemic. That issue, which has been updated and reissued by Lyle Stuart Inc (see p 278) has begun to play a part in continuing education courses for physicians and other health professionals, as well as in medical schools, universities, and high schools. A teaching guide to the issue (and this one) is being prepared, and suggestions would be appreciated.

What was intended in early 1984 to be a small supplement to the issue bloomed into an opus even larger than the original, as numerous ideas emerged for articles not addressed in print elsewhere. Further stimuli for this issue have been the visibility and success of the nonsmokers' rights movement, greater interest by employers in providing a smokefree workplace, the signing by President Reagan of the Comprehensive Cigarette Education Act, the appearance of an advertising campaign by RJ Reynolds that has attempted to cast doubt on scientific evidence in regard to smoking and disease, a call by the National Advisory Council on Drug Abuse for a ban on cigarette advertising, the dropping of cigarette advertising by two Canadian and one American daily newspaper, and the filing of lawsuits against tobacco companies by persons with cigarette-related diseases.

In assembling the articles in this issue the objective has been to move beyond a discussion of the well-known medical consequences of smoking to a consideration of ethical, social, political, economic, agricultural, religious, and legal aspects of this growing world problem. The articles in the section on Role Models provide a sobering account of the challenge posed by the cigarette industry's stepped-up efforts to recruit new smokers among women, blacks, the military, and young people in general. The paucity of leaders willing to stand up to cigarette advertisers does not bode well for the next generation.

Several myths perpetuated by the tobacco industry are further deflated by authors in this issue, including the notion that there can be a "less hazardous" cigarette or that smoking aboard aircraft or in other indoor areas does not pose a threat to health. It will come as a surprise to many that some of the sharpest criticism of the continued existence of the tobacco industry is emanating from the South. A coalition of health professionals and religious leaders, led by the North Carolina Council of Churches, is pushing hard for a greater awareness of the state's role in the promotion of a product that causes death and disease. At the same time reporter Lawrence Spohn points out the complexities of the tobacco subsidy. Those who are pushing for an end to this program as a high priority health measure should understand that this will adversely affect small farmers but not the manufacturers—and not the distribution or availability of cigarettes to young people. Similarly, although the passage of new cigarette warning labels is symbolic of legislative progress, it is hardly a potent way to fight the billions of tax-deductible dollars now spent promoting cigarettes to new recruits. By printing these warnings, the manufacturers may even have been given some protection against future product liability lawsuits.

Although it is encouraging to learn of the increased showings of the film *Death in the West* in universities, such news is tempered by the report of Dr Heinz Ginzel on the absence of a sizable component on smoking in the medical curriculum. In this issue Dr John Slade presents an ingenious framework for the study of smoking, in which the activities of tobacco companies are likened to the immunologic strategies of certain parasites and viruses to counter a host's natural defenses. The most important aspect of the study of the cigarette pandemic is an examination of the way in which the tobacco industry controls the image and idiom of smoking in society. Both this issue and the earlier one on smoking have employed numerous cigarette advertisements from past decades to illustrate the exploitation of various popular themes. But their outrageousness should not lead one to lose sight of the fact that today's advertisements are far more insidious by having blended current images and idioms so that one is less likely to be offended by the idea of promoting cigarettes. Just as one must study the life cycle of a parasite before attacking it, so one would prepare to tackle smoking by reading annual corporate reports, marketing surveys, and other resources used by the tobacco industry itself. Through an understanding of the tactics of the industry, and not just the health effects of smoking, one realizes that the leading cause of death in the US is not smoking per se, but rather Marlboro, followed by Winston, followed by Kool, etc. Countering the images of these specific brands may prove to be a key in eradicating smoking. In this way, efforts to end the pandemic will move along the lines described by Simon Chapman, with an emphasis on entire populations through the mass media, rather than on individuals through traditional medical models.

The two issues have included articles on progress and problems in regard to smoking and cigarette advertising in over 30 countries. Global marketing by cigarette manufacturers—designed by many of the same advertising agencies retained by pharmaceutical companies to promote drugs and other health-related products*—has enabled smoking to take hold in even the poorest of nations. The efforts of the Union International Contre le Cancer, the World Health Organization, and a quadrennial world conference on smoking are no match for the sophisticated network of cigarette companies, which is often in league with local governments, and publishers. Private foundations that have supported efforts to eradicate infectious diseases in impoverished nations could make a more complete contribution to international health by aiding innovative projects designed to prevent and eliminate smoking.

In this issue, some encouragement can be taken from the descriptions of efforts underway in several countries notably Iceland, Australia, New Zealand, Norway, Canada, and Kuwait. Ambitious programs are starting in Ireland and Israel. But the news is more often than not disturbing, such as in Japan, the Netherlands, France, Greece, Papua New Guinea, Burkina Faso, Northern Ireland, and the Soviet Union.

In the United Kingdom the British Medical Association (BMA) has launched an attack on the tobacco industry. As evidence it will not be engaging in just another antismoking campaign, the BMA has distributed cards to doctors designed to be sent to members of parliament notifying them when a patient (and constituent) dies of a cigarette-related disease. The BMA's publication of the *Report on Investment in the UK Tobacco Industry,* written by Charles Medawar of Social Audit, has proven embarrassing to many organizations. Among the 350 educational institutions, hospitals, religious bodies, and health charities found to have large holdings of tobacco stocks were the Imperial Cancer Research Fund, the Institute of Cancer Research, the Royal College of Pathologists, the Royal College of Psychiatrists, the Royal College of Surgeons of England, the British Heart Foundation, and Charing Cross Hospital Medical School. The BMA is persuading such shareholders to invest in more useful enterprises.

It is hoped that the new efforts of British doctors, as well as those of doctors in the Sudan who have succeeded in banning cigarette advertising, will inspire other medical associations around the world to devote time and money to tackle this leading international health problem. In the preparation of this issue, it has been difficult to identify health professionals in Central and South America, Asia, Africa, and Eastern Europe who are involved (or would like to become involved) in efforts to counteract smoking and its promotion. It is imperative that a network be developed beyond the official smoking control agencies, and the *Journal* welcomes correspondence from those who may wish to aid in identifying and working with individuals around the world to end this scourge.

In contrast to the United States, where consumption of cigarettes is at last no longer increasing (but still remains among the highest in the world), the problem in most countries is getting worse. In Papua New Guinea, as Dr Euan Scrimgeour writes, a single company (British American Tobacco) has used advertising to build the image of smoking as a sign of success and sporting prowess. Similarly, according to Dr David Sokol, in Burkina Faso—and in most other countries in Africa, Asia, and South America—street vendors from British American Tobacco, many of whom are children and teenagers, sell cigarettes by the piece, even in front of hospitals. During the current African famine cigarette shipments and promotional campaigns have increased in drought-stricken countries. As Dr Keith Ball notes in this issue, doling out cash payments for cigarettes drains the capacity of a country to pay for food and other essential needs.

A more hopeful picture is emerging in Western Australia, where health minister Barry Hodge and health promotion director Mike Daube have strong governmental and popular backing. The $2 million a year mass media effort described by Dr Calvin Miller in this issue includes a new electronic billboard that ticks off the 45 Australian deaths a day attributed to cigarette smoking. BUGA UP (Billboard Utilizing Graffitists Against Unhealthy Promotions), whose most recent activities are also reviewed, continues to set the pace. Its new book AD EXPO, an expose of techniques used by advertisers, has been distributed by BUGA UP to Australian school children to help them develop their skills in seeing through advertising images and claims. This group is deserving of a Nobel prize.

Governments in Scandinavia have generally been more supportive than elsewhere of efforts to curtail smoking and its promotion. If present trends continue, one country above all has a chance to eliminate smoking early in the next century: Iceland. Its isolated location is one advantage. Another is pride in capital city Reykjavík's reputation as "the smokeless city" (because it is heated by thermal energy from underground springs and lacks industrial air pollution). A talented and well-coordinated group consisting of the Health Minister, the Icelandic Cancer Society, volunteer health professionals, local and national government officials, and advertising personnel have taken up the challenge to eradicate smoking. Their recent efforts to close loopholes in laws against tobacco advertising and discourage the sale of tobacco to minors are described in this issue. In an attempt to negate the effect of the newly mandated full-color graphic warnings on all cigarette packs, tobacco companies have introduced new pack designs and have slashed prices. Although these brands have sold well, the companies' plan may have backfired as health officials witness the way in which the intent of the law is being undermined.

The pattern of end-running of laws restricting the sale and promotion of cigarettes helps to explain why consumption is not declining in most areas of the world. In addition, the absence of frequent and engaging counteradvertising messages to redress years of cigarette company propaganda, and the lack of enforcement of measures to restrict smoking (and littering of smoking materials) in public places permits cigarette companies to control virtually all of the imagery and vocabulary of the smoking ritual. In her book, *Legislative Action to Combat the World Smoking Epidemic,* (Geneva, World Health Organization, 1982) Professor Ruth Roemer emphasizes that the enactment of legislation is a necessary but not a sufficient prerequisite for the effectiveness of any campaign to combat smoking:

---

* The following advertising agencies create promotional campaigns in various countries to increase the sale of cigarettes as well as pharmaceutical products, raising the question of a conflict-of-interest in matters of public health. In parentheses, the tobacco company client is listed first, followed by the pharmaceutical clients: Ted Bates, NY (BAT, Schering-Plough, Warner-Lambert); BBDO International, NY (Rothmans, Bayer/Miles); Leo Burnett Co, Chicago (Philip Morris, Beecham, Bayer/Miles); Foote, Cone & Belding, Chicago (Philip Morris, Rothmans, Warner-Lambert); Grey, NY (BAT, Bristol Myers, CIBA-Geigy); J. Walter Thompson (RJ Reynolds, Eli Lilly, Beecham, Abbott, Glaxo, Hoechst, Johnson & Johnson, Warner-Lambert, American Home Products); McCann-Erickson (RJ Reynolds, Johnson & Johnson, Bayer, Warner-Lambert, Dow Chemical).

> Legislation is essential as a means of establishing and promulgating public policy, enlisting the resources of all government departments (not merely of the health department), strengthening the activities of voluntary organizations, and contributing to the development of a non-smoking environment; but it is only one component in a comprehensive attack on the smoking epidemic, which includes preventive action, public education, assistance with smoking cessation, special activities for high-risk groups, and research on the biological and behavioral aspects of smoking.

Several successful American battles for the enactment of clean indoor air legislation, warning labels on tobacco products, and bans on the free distribution of cigarettes are described in this issue. However gratifying such victories may be, the passage of laws must not be allowed to lead to complacency and a loss of momentum. Rather, support should then be given to non-legislative activities such as the development and dissemination of counter advertising to discourage cigarette consumption.

The constant refrain of the tobacco industry that countries that have imposed bans on cigarette advertising have not seen a decline in smoking is a dangerous distortion. Such a claim, in addition to being untrue, presumes that advertising comprises solely billboards, magazines, newspapers, and the broadcast media. In fact advertising constitutes a far broader range of activity seldom covered in legislated bans, such as political lobbying and corporate underwriting of sporting organizations, cultural institutions, and educational programs. Promotions can involve an array of symbols, colors, and sounds. Even in countries such as Iceland that have banned all sports and cultural sponsorships, the favorable depiction of smoking has continued in imported movies, rock videos, and such television programs as *Miami Vice.* In addition the absence of enforcement of measures to restrict smoking in public reinforces in children and adolescents the image of smoking as the social norm. Advertising bans do not work, then, unless a more complete social system evolves that reduces access of young people to cigarettes, as well as incentives to profit from the sale of the product and to smoke in public. The implementation of counter advertising to ridicule smoking also lessens the likelihood that cigarettes will be regarded as forbidden fruit. The high level of smoking in the Soviet Union, where there is no commercial advertising, may exist in part as the result of a lack of engaging educational efforts to counteract smoking. There is some question, as raised by Dr Richard Cooper in this issue, as to whether the state really wants to decrease consumption and risk the loss of a lucrative source of short-term income. "They don't have much in the Soviet Union," adds Elton Braude, who writes on advertising, "Anything available is *ipso facto* encouraged." The tobacco industry also does not cite countries such as Kenya or Austria where there is a private or state-owned monopoly and a high level of advertising. As Chapman points out in *Cigarette Advertising and Smoking: A Review of the Evidence* (British Medical Association, 1985), the purpose of cigarette advertising is to increase overall consumption.

Recognizing the adverse effects of cigarette advertising on young people, the House of Delegates of the Medical Society of the State of New York at its annual meeting in June 1985 passed a resolution supporting "an allocation in the State budget used specifically to purchase space in the mass media as a means of countering the current youth-oriented advertising campaigns of cigarettes and tobacco products." An increasing number of resolutions calling for legislative measures to curtail smoking are being passed by medical societies throughout the country. Also in the past year the American Medical Association and its publications have begun to devote greater attention to disseminating information on the hazards of smoking. Other medical societies, however, notably those that deal with heart disease, cancer, and chest diseases, are doing little. Moreover it is unlikely that physicians can pin their hopes on legislation unless they are willing to visit legislative offices at the local, state, and national levels. Taking a cue from the rally by several thousand physicians in Albany to protest the increase in premiums for medical liability insurance, physicians could well establish an annual legislative rally or vigil in support of key bills such as those aimed at curtailing smoking. All physicians and medical students should take off at least one day during the course of the year to participate in the legislative process. Another strategy, modeled after the success of the New York Medical Political Action Committee (NYMPAC), has been the establishment of PAC-PAC (People Against Cigarettes Political Action Committee, P.O. Box 1018 Times Square Station, N.Y., N.Y. 10036) which plans an ambitious program to educate legislators on the smoking issue.

Another important step would be for medical students and faculty to follow the lead of the University of Sydney by urging the end to all affiliations with the tobacco industry, including stocks and research grants. Other questions must be directed at health-oriented organizations such as hospital corporations and pharmaceutical companies that ought to be lending greater support to efforts to end smoking. To demonstrate that they mean business, health professionals could launch boycotts not only of tobacco company products but also of products of companies that aid the tobacco industry.

To be successful in reducing and eliminating smoking will entail anticipating the tactics of the tobacco industry rather than merely reacting to them. Thus while the expression of outrage by health organizations over the advertising campaign by RJ Reynolds (aimed at sowing seeds of doubt about the medical evidence on smoking) is understandable, it is disturbing that these organizations do not also speak out often and forcefully enough against the far more prevalent image-based advertising that appeals to children. The futility of petitioning the Federal Trade Commission (FTC) to crack down on Reynolds is exemplified by the year-and-a-half-long such effort by a group of physicians led by Thomas Houston, MD, of

Rome Georgia. (Dr Houston has also urged enforcement of the law against cigarette advertising on television in regard to advertisements on scoreboards frequently shown in telecasts. Other organizations have joined his petition for a ban on smokeless tobacco advertising.) By the time the FTC rules on the Reynolds matter, it is likely the advertising campaign will have run its course and the company will have moved on to other dirty tricks.

The onus of responsibility for the health of the user of cigarettes must be shifted onto the manufacturer, as it is for almost any product. In view of years of misleading advertising, tobacco companies should be forced to foot the bill for prime-time corrective advertising, designed by advertising agencies under the direction of health promotion experts. One might also anticipate that the recent court decision in Chicago that a corporation can be held criminally liable in a case of homicide may well soon apply to malefactors in the cigarette industry.

One effort in which the medical profession could play a key role would be in dispelling the notion that the cigarette manufacturer is in any way a friend of the consumer. No other product has inspired the perverse misplaced loyalty of the consumer in the manufacturer as has tobacco. This is largely the result of the industry's exploitation of the psychology and physiology of smoking. Yet it is nonetheless amazing that the more reports implicating tobacco in serious illness, the more stalwart becomes the defense of the industry. If the undercarriage of the family automobile were found to be made of cardboard instead of steel, one would sue the manufacturer. Not so with the toxic cigarette. One would never spend $50 for a pound of hotdogs; yet the consumer is paying a similar mark-up for cigarettes. No other product contains as many chemical additives as cigarettes without having so much as a single ingredient listed on the package; yet consumers don't realize that there is anything besides tobacco and paper in a cigarette.

But in the face of the enormous image-based campaign of the cigarette industry, it may not be possible to succeed in getting to people who take up smoking even by educating them about chemical additives, diseases, money down the drain, and premature aging. However, there remains one promising way to attract massive support for efforts to end smoking: disseminate a photograph around the world of a baby seal dying from a cigarette-related disease. A precedent for this occurred in 1975 when a newspaper photograph of beagle puppies strapped into a smoking machine sparked a flood of protest. The success of animal rights and wildlife organizations in mobilizing public sympathy for the plight of various animals has been far greater than 30 years worth of publicity about the adverse effects of smoking in human beings. Perhaps the entire antismoking campaign should be turned over to Greenpeace. Who knows what other species might be saved?

ALAN BLUM, MD

Follow-up of a cover-up

Only the *Columbia Journalism Review*, *The Washington Monthly* and a handful of other publications reported on the censorship issues raised by the *New York State Journal of Medicine* in Dr George Gitlitz' article, "Cigarette advertising and *The New York Times:* an ethical issue that's unfit to print?" And only one daily newspaper, the conservative *New York Tribune*, with a circulation of just 20,000, has raised the subject in depth, in an article by Karen Shelton entitled, "Lucrative cigarette ads pose moral paradox for *NY Times*" (May 16, 1984). Apart from a single documentary on ABC-TV, no network television station or newsweekly has covered the subject of the print media's acceptance and solicitation of cigarette advertising.

By ignoring repeated challenges to inform readers about the amount of revenue it derives from cigarette advertising, the New York Times Company is effectively fostering complacency about the problem while profiting from it. In contrast to the numerous ethical judgments *The Times* makes in its editorial columns—such as the newspaper's opposition to the appointment as surgeon general of C. Everett Koop, MD, (whom it described as "Dr Unqualified"), because of his views against abortion—*The Times* has refrained from editorial scrutiny of the ethics of the cigarette advertisers. The reasons for this silence become clearer upon consideration of the company's overall holdings: many of its 20 daily newspapers are located in tobacco-growing regions, including four in North Carolina and three in Kentucky. In New York City, the headquarters or subsidiaries of five of the six American cigarette companies are within walking distance of The New York Times Company. By dint of their non-tobacco product advertising (eg, Gimbels and Saks Fifth Avenue of British American Tobacco), The New York Times Company is one of the country's largest beneficiaries of tobacco industry advertising expenditures (Fig 1). The company, the 12th largest media conglomerate in the US with total media revenues of $1.2 billion in 1984, owns such magazines as *Family Circle*, *Golf Digest*, and *Tennis*, in all of which cigarettes are heavily advertised. A letter written to a member of *The Times'* board of directors, Judith Levinson, MD, concerning the acceptance of cigarette advertising and the absence of articles on cigarette smoking during the past 15 years in *Family Circle* was referred for reply to the Times Company's vice chairman Sydney Gruson "since business matters were involved." Gruson responded that "there have been a number of short *Family Circle* references, though not articles, on the wisdom of stopping smoking."

There have been several developments since the December 1983 issue related to the ethics of the acceptance of cigarette advertising by *The Times*. In November 1984 the House of Delegates of the Medical Society of the State of New York passed a resolution urging newspaper and magazine publishers within the state to decline all cigarette advertising. In response to a letter from the late Milton Rosenberg, MD, executive vice president of MSSNY, Gruson wrote that "the company has debated internally the issue of tobacco advertising for many years" (thus acknowledging an ethical dilemma) "but feels that closing its columns to such advertising would set a dangerous precedent."

In December 1984, Dr Gitlitz, in association with DOC (Doctors Ought to Care), sought to purchase a ⅙-page $3,000 advertisement (Fig 2) in a special section of *The Times* entitled "Healthcheck," along with the following small advertisement on the front page of the newspaper: "The two faces of *The New York Times:* Cigarette advertising and 'Healthcheck.' See advertisement . . . ." Initially, the manager of advertising acceptability would not accept the advertisements because, he said, they might leave "an adverse implication against *The Times*." After negotiations, in part with a First Amendment lawyer, *The Times* offered to publish the larger ad and a watered down version of the smaller one: "Cigarette advertising and

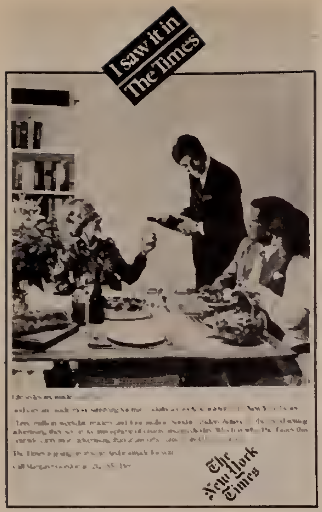

I saw it in The Times
The New York Times

FIGURE 1. One of a series of advertisements by *The New York Times* in the *US Tobacco and Candy Journal* in which *The Times* solicits cigarette advertising with the slogan, "Lifestyles are made, not born."

The New York Times. See DOC advertisement page 34 . . . ." (The advertising acceptability manager did not recall any instance in which *The Times* had questioned an advertisement from a tobacco company.)

In March, 1985 Elizabeth Whelan, PhD, of the American Council on Science and Health, wrote to *The Times* regarding its acceptance of an advertisement by RJ Reynolds purporting to give scientific facts on smoking's relationship to heart disease. Gruson replied, "We published the advertisement in accordance with our acceptance policy. The alternative is censorship and we're not going to engage in that."

In April Dr Gitlitz' article and follow-up material in the *Journal* were selected from more than 250 nominations by journalists, editors and others as one of the 25 most important under-reported national news stories of 1984 in an annual media research effort at Sonoma State University, California, known as "Project Censored." The primary objective of "Project Censored" is to explore the extent of censorship in our society by locating stories about significant issues of which the public should be aware but is not.

In late April, after several months of correspondence, *The Times* agreed to publish a commentary by Dr Gitlitz, originally entitled, "*The New York Times* should stop advertising cigarettes," if he would agree to make it a general criticism of cigarette advertising and not of *The Times* in particular. The published version, entitled, "Drop cigarette advertising," eliminated all but one of six original mentions of *The Times.* The only letter *The Times* published concerning the article was a rebuttal by the same Philip Morris vice president who had written a commentary for *The Times* a few months earlier, one of several such articles and letters from Philip Morris executives *The Times* has published in recent years. Notwithstanding the fact that Dr Gitlitz was addressing the newspaper's role in the acceptance of cigarette advertising, *The Times* deferred to its advertiser to respond.

The New York Times Company is not alone in helping to serve the interests of the tobacco industry. The same criticism applies to most media conglomerates including Gannett, CBS Inc, the Chicago Tribune Co, the Times-Mirror Co, Newhouse Newspapers, TIME Inc, the Washington Post Co, Knight-Ridder, and Triangle Communications (this last company, publishers of *TV Guide,* the largest single source of cigarette advertising, notes in its promotions to advertisers that each issue reaches 8.8 million adolescents aged 12–17).

Cigarette Advertising and *The New York Times*

An Ethical Issue that's Unfit to Print?

From January 4, 1975 to May 25, 1983 *The New York Times* was repeatedly requested, the requests eventually becoming challenges, to discuss publicly its policy of advertising cigarettes.

The response to these communications was occasionally a personal reply, often no reply at all, but never a response in print.

This unpublished correspondence was printed, under the heading used above, in the December, 1983 issue of *The New York State Journal of Medicine*, an issue devoted entirely to an analysis of the manner in which the world of business unrelentingly perpetuates the "world cigarette pandemic." In the year since its publication, this issue has been acclaimed as a landmark in public health journalism.

Despite that acclaim, and despite the fact that copies of *The Journal* were hand-carried or mailed directly to, and in some cases discussed personally with, members of *The New York Times* from the publisher, Mr. Sulzberger, to the former Senior Editor, Mr. Oakes, to the medical writer, Dr. Altman, to the "social conscience" columnists Mr. Safire and Mr. Schanberg, and many others — *The Times* has not acknowledged the existence of this issue of *The Journal* by so much as a one-sentence news item, much less editorially.

By this time, whatever *The Times* might have to say about its policy of advertising cigarettes would be almost anticlimactic. What is intriguing is its dogged and unwavering determination not to say anything at all!

The Times speaks out on every other subject under the sun. Why is it so uncomfortable with this one?

We again challenge *The Times* to tell its readers, on the Editorial Page, why, in 1985, with all that is known about cigarettes, it persists in advertising them. The explanation should be fiscal as well as philosophical. We urge readers of this "HEALTHCHECK" section — people with a serious interest in health — to join us in making this challenge.

DOC Broome County
4513 Old Vestal Rd., Binghamton, NY 13902

(DOC — Doctors Ought to Care — is a chapter of DOC, Inc., Floyd Medical Center, Rome, GA 30161, a national organization of physicians and others devoted to educating the public about the major preventable causes of poor health and high medical costs)

FIGURE 2. Advertisement questioning the ethical standards of *The New York Times* was published in spite of the newspaper's attempt to change the wording.

But because of *The Times'* uniquely influential and uniquely respected position, its ignoring of the ethical issues surrounding cigarette advertising enables all other media corporations to perpetuate the myth that there is no issue. And because *The Times* is fully aware of the harmfulness of smoking—something its tobacco company advertisers will not admit—and chooses to profit from the promotion of cigarettes anyway, it may be even more at fault than the tobacco companies themselves. In effect, vice chairman Gruson and other executives at *The Times* seem to be saying to readers, including children and teenagers, "Let them eat smoke."

Competing agenda in smoking control agencies

"Those who pay the piper . . ."

SIMON CHAPMAN, PHD

People who feel strongly enough about the avoidable toll of death and disease caused by smoking that they choose to work in smoking control have a small number of prospective employers. Jobs are usually limited to units within government health departments, to national heart and cancer charities, and to academic research and teaching institutions. Each of these groups can be expected to embrace the usual litany of smoking control goals and have in many instances contributed greatly to community awareness about the health risks of smoking. They are each constrained, however, in what they ultimately can be expected to contribute toward the attainment of these goals by agenda that lie at the heart of the groups' existence. This paper reviews the limitations on establishment agencies' contributions to further progress in reducing the prevalence of smoking and underlines the necessity for independent splinter groups.

POLITICS AND SMOKING CONTROL

There are two sets of questions associated with smoking control: questions about how its objectives are best achieved, and questions about whether they ought to be achieved. The former are essentially empirical, technical, and creative questions: how great a tax increase will cause how much of a decrease in smoking prevalence? What educational strategies "work" with which groups? How much of a media budget is needed to take a message past a threshold of community awareness? These are the sort of questions that preoccupy smoking control agencies, and especially academic institutions. However, without the questions of "how" having reference to the "ought" questions, the issues can take on a rather naive complexion, in the end consigned to the basket of "so what" research.

Smoking control—the totality of effort that has as its objective the decline and eventual elimination of tobacco smoking and therefore of one of the world's largest and most powerful industries—is a deeply political issue. Those persons concerned with smoking control represent a group whose interests are antithetical to those of the tobacco industry. The tobacco industry's concern is to maximize consumption of its commodity, whereas antismoking groups represent a voice that seems inconsistent with the goals of capitalism: they seek to eliminate the consumption of a commodity by appealing to values of health, social acceptability, and aestheticism. In the words of two chief apostles of free enterprise, Milton and Rose Director Friedman:

> Few trends could so thoroughly undermine the very foundations of our free society as the acceptance by corporate officials of a social responsibility other than to make as much money for their shareholders as possible.[1]

Against this view, smoking control advocates are firm political enemies of not only the tobacco industry, but of political interests that reject the intrusion of the state into the free marketing relationship between producers and consumers. Such governments are in power in many nations including the United States and Great Britain, where their respective health establishments have long and strongly urged smoking control. In recent times, the attempted appropriation and championing by the tobacco industry of ideologies concerning freedom of choice, employment, and an anti-"nanny state" theme have amplified the politics of smoking control. The issues go beyond the self-contained arguments about community health that have defined smoking control for the apolitical health worker. Policy makers in smoking control institutions who cannot or will not address the political dimensions of their task cannot hope to contribute any substantial gains to smoking control objectives.

UNPOPULAR PREVENTION

The history of government commitment to smoking control is mostly one of "the control you're having when you're not having control." With the Nordic countries leading the way in comprehensive programs—advertising bans, vigorous and changing pack warnings, mandatory school health education, tough price policies—others bear witness to the yawning gap between the unanimous recommendations of expert committees and what governments are prepared to do. Politicians and their advisers who advocate smoking control are just one voice in an arena of competing interests documented so thoroughly by Taylor.[2] Against the seemingly self-evident arguments for smoking control, centering on preventing premature death, are the economic advantages of a large tobacco industry. Dominant political thinking has seldom been put more plainly than by the British Conservative Member of Parliament Anthony

---

Dr. Chapman is the author of *The Lung Goodbye—a manual of tactics for counteracting the tobacco industry in the 1980s.*

Address correspondence to Mr Chapman, Research Fellow, Health Education Council, 78 New Oxford St, London WC1A 1AH England.

Marlow:

> If people are sick from smoking, go into a hospital and die after six months, they die. If they do not die from smoking they live perhaps for 10 to 15 years longer, and may go into a geriatric home. During that time, they will have been drawing old age pensions, they will have been a further burden to the Health Service and so on. I accept the fact that people who smoke get diseases, are ill, and cost the Health Service money. But they are cheap compared with the alternative.[3]

In the United States, where many of the health care costs must be borne by the individual rather than by the state, such an argument would be even more persuasive to politicians who embrace a victim-blaming, caveat emptor ideology of the individual's responsibility to choose not to smoke.

People committed to smoking control who work for the government inevitably and quickly learn what measures and methods are out-of-bounds for their political employers, who may often already be persuaded of the electoral advantages of the rhetorical flourish and token funding of an anti-smoking campaign. In most cases, their work consists of running underfunded media campaigns, distributing low-key pamphlets, preparing reports—the recommendations of which will seldom be adopted—to assuage those who urge that government ought to be doing something substantial. Ongoing expert committees are the salvation of politicians who, to criticism about government inactivity, can always reply that the complex issues are being looked at by a committee. By the time committees report, governments and responsible ministers have often changed guard, and the process can be expected to start again.

In 1985, government needs only to be told the fine print of how to act against smoking. The chapter headings, such as those clearly set out by the International Union Against Cancer[4] and the World Health Organization[5] have been written long, loud, and clear. The fact that so few governments have been prepared to take decisive and comprehensive action against smoking should become the problematic focus of the most important tasks facing smoking control agencies.

Worden's distinction between popular and unpopular prevention could hardly be better exemplified than in the case of most governments' records in smoking control. Popular prevention "spawns programs that are piecemeal, cosmetic, ineffective, fun and futile,"[6] sometimes reducing smoking prevalence among the programs' small, select audiences but rarely sufficiently funded to have an impact on whole communities or national populations.

In contrast, unpopular prevention is policy-oriented and is aimed at the social, economic, and political structures that contribute to widespread smoking and to the economic health of the tobacco industry. The day-to-day tactics of an "unpopular" smoking control program would often seem sacrilegious to the orthodox health care worker. Unpopular prevention is not easily described in terms of programs. What is done in unpopular prevention does not lend itself to precise statement as an independent variable capable of direct replication by others. There are seldom definable steps amenable to the requirements of the "methods-results-discussion" format demanded by journals, which seem to define the boundaries of the acceptable in popular prevention. In working toward changing public and political opinion in favor of, for example, an advertising ban, the unpopular prevention worker will need to adopt the same arsenal of opportunistic, responsive, imaginative, flexible, newsworthy, dramatic, and mythologizing tactics that are the stuff of all successful public opinion, political, and commercial compaigning. Clearly, such tactics are rarely the modus operandi of bureaucratic governmental departments.

HEALTH CHARITIES

Charities that raise funds to counteract heart disease and cancer have played a vanguard role in contributing to public awareness of the health consequences of smoking. Here are people, reasons the public, who are opposed to smoking because they are confronted daily with its consequences. However, the charities are mindful of their public image and its relationship to the preparedness of the public to donate money to their funds. It seems that an entrenched axiom of such charities is that people will give more money if it is to fuel the drama of saving lives via elaborate and expensive buildings, machinery, and dramatic procedures than if a major profile of prevention is taken. Despite their lofty commitment to the rhetoric of prevention, the expenditure of charities' funds is almost always in favor of research into treatment—reflecting the hegemony of professional interest of the medical executives who make the main decisions within the charities. Over the years, they have developed dignified and established reputations as impeccably apolitical bodies, dedicated to the allegedly apolitical tasks of curing and preventing disease.

These characteristics can be enormously important in lending a special sort of credibility to the charities in conflicts or outspokenness against government or industry. But generally, the tactics and conflicts involved in confronting the tobacco industry or conservative governments on their manifest or latent pro-tobacco industry policies, sit uneasily with health agencies' chosen roles as behind-the-scenes funders of research and treatment. When appearing as a commentator or in a debating role on smoking control, health charity spokespersons typically fall back into the supposed political neutrality of letting the facts about smoking and disease speak for themselves. The decision to soft-pedal on confrontation with government and the industry is a highly political decision in itself.

ACADEMIC INSTITUTIONS

There is now, 23 years after the publication of the Royal College of Physicians' first report on smoking and health, several voluminous reports from the office of the Surgeon General of the United States Public Health Service, and four world conferences on smoking and health, a massive amount of research—biochemical, clinical, epidemiologic, attitudinal, and behavioral—that provides any government willing to act with both justification and direction. Most of this research has been conducted in academic institutions; and there will always be an ongoing need to refine, reassess, and replicate persuasive and educational strategies in readiness for such times when they might be put into practice. Behavioral scientists and educators with interests in smoking will have work for a long time.

However, the current status of behavioral research into smoking is deeply inconsequential, serving mostly the career interests of those who conduct it. The overriding problem with the outpourings of behavioral research into smoking is that its implications are rarely taken up by governments or given mass currency in the media. On one extreme there are those academic researchers who are funded directly by the tobacco industry in alleged "no strings" arrangements. In Australia, for example, the industry's Tobacco Research Foundation, administered by the Department of Medicine at the University of Sydney, has enabled the industry to boast publicly about its research commitment to the pursuit of truth in what it describes as the smoking and health "debate." While those in receipt of grants have continued to turn out damning evidence about smoking's effects on health, none have publicized their findings in anything but the turgid and obscure academic press. It might be argued that, not wanting to bite the hand that feeds them, research scientists in receipt of tobacco industry money renege on whatever social responsibility their privileged knowledge gives them to publicize the socially relevant aspects of their findings.

For the most part, behavioral research, despite being well-intentioned, concludes its life in dusty reports and in applauded meetings of like-minded colleagues. Pilot and experimental programs rarely become incorporated into school curricula, and recommendations about the accessibility of cigarettes to children and their recall and liking for advertising fade through want of a sustained campaign to influence those with power to act. Why does the tobacco industry never attack or lobby against academic research in the way that it does against public pressure groups and highly placed maverick politicians? The answer is surely that it realizes that smoking control energy can be safely diverted into research, where it will be self-contained and cloistered.

The terms of reference of the recently established Harvard Institute for the Study of Smoking Behavior and Policy to the Carnegie Corporation[7] encompass all the main research areas about smoking as a behavior. The prestige of the institute's setting will doubtless attract researchers of high caliber, whose findings will be given the added attention a Harvard report can generate. The research plans, however, give little indication that the institute's work will not simply put more stacks on an already towering mill, rather than act as a force for making waves in the big ponds of public opinion and political action. It would be rewarding to learn from the institute's directors their plans for using the research findings generated, and how their approach differs from that of others who have tried to influence less than enthusiastic governments.

MICE THAT ROAR:
INDEPENDENT SPLINTER GROUPS

The three types of smoking control agencies described each work under larger, constraining agenda that limit the final contribution to the ultimate goals of prevention. While being quite divergent in their approaches, independent groups such as Action on Smoking and Health (ASH) [United States, United Kingdom, Australia, New Zealand, Malaysia], Non-Smokers' Rights Association [Canada], Movement Opposed to the Promotion of Unhealthy Products (MOP UP) [Australia, New Zealand], Billboard Utilizing Graffitists Against Unhealthy Promotions (BUGA UP) [Australia], Action Group to Halt Advertising and Sponsorship by Tobacco (AGHAST) [United Kingdom], Doctors Ought to Care (DOC) [United States], Californians for Nonsmokers' Rights, and various American chapters of GASP (Group Against Smoking Pollution) have, on comparatively miserable resources, managed to make sometimes unprecedented contributions to smoking control. Most of these groups are composed of persons who, frustrated by the constraints on their professional smoking control roles, have sought a vehicle for independent action. Such groups can usually be relied on by journalists to shoot from the hip against the industry and complicit governments, in contrast to the guarded equivocations of establishment agency representatives. Independent groups have more flexibility than their established counterparts and can decide for themselves what should be said and done about smoking control issues, as well as the form their actions should take. This allows a freer and more responsive range of tactics to be used—so crucial in improving the probability of access to a mass media that is hungry for newsworthy conflict. Their acronymic names are memorable, with populist appeal. Their main constraint is their lack of funding to pay for direct access to mass media.

Where they have impeccable support—as in the case of the British ASH with its royal patronage and long list of credentialed supporters—they may have direct access to influential members of the government. Where such independent groups have set up autonomously and are mindless of treading on the sensibilities of the orthodoxy (BUGA UP, for example, with its civil disobedience graffiti campaign), they have tended to radicalize the activities of other groups. So uncompromising is their position, that health charities, academics, and government representatives unavoidably have their tentative positions seem more conservative on matters such as advertising bans and subsidies for the industry. People who tend to be sensitive to the questioning of journalists usually inch further along the road of outspokenness, thanks to the benchmarks established by the independent groups.

The task ahead in smoking control is manifold. Outstanding among all the claims being made for priority action is the need to politicize the issue to the point at which governments will be embarrassed not to act against an industry whose fundamental and obvious role in promoting and maintaining smoking prevalence is still politely avoided in the statements of agencies like the Harvard Institute.

REFERENCES

1. Friedman M, Friedman RD: *Capitalism and Freedom.* Chicago, University of Chicago Press, 1962.

2. Taylor P: *Smoke Ring. The Politics of Tobacco.* London, Bodley Head, 1984.

3. Marlow A: House of Commons, May 11, 1982. Quoted in Foot P. Paul Foot reporting. "Warning! This MP can damage your health." *Daily Mirror* October 20, 1982.

4. Gray N, Daube M: *Guidelines for Smoking Control.* Technical Report Series, vol 52, 2nd ed. Geneva, Union International Contre Le Cancer (UICC), 1982.

5. World Health Organization: Report of the WHO Expert Committee on Smoking Control. *Controlling the Smoking Epidemic.* Technical Report Series 636. Geneva, World Health Organization, 1979.

6. Worden M: Popular and unpopular prevention. *J Drug Issues* 1979; 9: 425–433.

7. Allison GT, Schelling TC, Richmond JB: "A proposal for the establishment of an Institute for the Study of Smoking Behaviour and Policy." Submitted to the Carnegie Corporation of New York by the John F. Kennedy School of Government, Harvard University.

A smoke-free society by the year 2000

C. EVERETT KOOP, MD

Dr Julia M. Jones was a great physician, a true humanitarian, and an excellent scientist. She devoted her professional life—and most of her personal life—to the fight against tuberculosis. In the spirit of Dr Jones, I would like to review the history of the fight against smoking and to suggest a course of action for the future.

It is appropriate to take stock at this time.Twenty years ago, one of my predecessors, Dr Luther Terry, released "Smoking and Health: the Report of the Advisory Committee to the Surgeon General." It was an important event, since the report described a major health risk that could be controlled. My first Surgeon General's report in 1982 documented the causal link between cigarette smoking and cancer, particularly lung cancer. The data established the link, implicating smoking in as many as 129,000 smoking related cancer deaths per year. The 1983 report focused on the close relationship between smoking and cardiovascular disease, and the numbers were even larger: it was estimated that cigarette smoking was directly related to 170,000 deaths from coronary heart disease each year.

LESSER CULPRITS, GREATER ATTENTION

Society's response to other cancer-causing agents has been documented. The Agent Orange case is apparently being settled, with seven chemical companies agreeing to pay $180 million to 16,000 veterans. The veterans claimed the herbicide has brought on a variety of disease conditions, including cancer. The evidence against Agent Orange as the cause of the alleged diseases is less substantial than the evidence against cigarettes. But the American public, the major corporations involved, the Congress, agencies of the executive branch, and most of the country's lawyers seem to agree that there is enough evidence to implicate Agent Orange.

But, taking into account all the possible victims of Agent Orange during the several years it was used in the Vietnam War and adding up the potential costs associated with the alleged illnesses yields a sum that falls far short of the devastation that is produced by cigarette smoking in this country in just one year.

The nation has recently reacted the same way toward ethylene dibromide (EDB), a pesticide that had been used for the past 35 years to protect stored grain, grain milling machinery, and citrus and tropical fruits from insect infestation. But eight years ago the National Cancer Institute released research findings that implicated EDB as a cause of cancer in laboratory animals. Since the first announcement, more tests were conducted, with similar results. Then EDB residue was found in ground water, and, together with a number of other events, this led to restrictions against EDB. It is not a safe product after all. However, there is no clear case of an individual who developed cancer as a direct result of ingesting EDB, but such explicit proof is not necessary under the law. Therefore, on March 2, 1984, the Environmental Protection Agency (EPA) ordered that EDB must be eliminated from the American food marketplace by September 1, 1984. The ban took place just nine years after the first, somewhat tentative report out of the National Cancer Institute. All things considered, that is rather fast.

Part of the veterans' claim of injury from Agent Orange is that they had no choice about their exposure. They were under orders to be in a place where the material was being sprayed, and, thus, unwillingly and unwittingly, they became victims. Part of the EPA's argument against further use of EDB is that the public has no other way to protect itself. The pesticide is now pervasive in the environment; hence, people can unwillingly become victims of its harmful effects.

There is a vital difference between the tolerance of those cancer-causing agents and cigarettes. The act of smoking is not mandated by law. It is a voluntary act: one does not have to smoke if one does not want to. One hundred and thirty million Americans over the age of 14 do not smoke, but 53 million Americans do. For many reasons, some that are understood and some that are not, these 53 million people are willing victims of one of the most destructive substances in the environment.

BEGINNING OF THE END?

Hence the reason for the Surgeon General's report on smoking and health. The 1984 report documents the relationship between cigarette smoking and chronic obstructive lung disease. Once again, there is a grim picture of high morbidity—chronic, long-term suffering—among cigarette smokers. This report reflects the many years of research and service spent not only by the government but also by the American Lung Association and the American Thoracic Society that have led to better methods of identification, prevention, and treatment of lung disease.

But government reports alone will not get the job done. Other things have to happen. The public, for example, has to believe the reports. It is a tortuously slow process, but I believe the message is getting through and the strength of those 130 million adult non-smokers is finally being felt.

The year 1984 marked the 100th anniversary of the invention of the cigarette machine by James Bonsack and was an appropriate time to initiate a major health campaign. In 1983, there was a 7% drop in the annual per capita cigarette consumption, bringing the figure down to less than 3,500. It has not been that low since 1949. According to provisional figures drawn from the health interview survey carried out by the National Center for Health Statistics, male cigarette smokers as a percentage of all men declined from 37.9% to 33% between 1980 and 1983.

Based on the Julia M. Jones Lecture presented to the annual meeting of the American Lung Association, Miami Beach, FL, May 20, 1984.

Address correspondence to Dr Koop, Surgeon General, US Public Health Service, Rockville, MD 20857.

Among women, the decline was from 29.8% to 29%. In addition, in 1983 21% of all high school seniors smoked cigarettes; six years ago nearly 30% said they did. Maybe the 20 years of research are finally having an effect.

In addition to the weight of the research data, there are other reasons for the slow but steady decline in smoking in America. First, the prevention message of public service advertisements is getting through. For example, women are finally getting the message about the link between smoking and lung cancer and cancers at other sites of the body, and none too soon, with lung cancer beginning to eclipse breast cancer as the number one cause of cancer-related deaths among women. Also, women are demonstrating a strong preference for an improved diet and more exercise, two mainstays of any program of prevention and health promotion. Once in that frame of mind, a woman can accept the anti-smoking message and act on it, and apparently many thousands of women have.

A second reason for the decline in smoking is economic. The increasing cost of a pack of cigarettes has had an effect on cigarette consumption, but also of importance is the realization by consumers, insurers, and employers that cigarette smoking adds to the health care costs of the individual and the nation. The effects of cigarette smoking are all too often manifested in long hospital stays and extended outpatient care for a variety of chronic health problems that could have been avoided.

POSITIVE INCENTIVES

The third reason for the decline in cigarette smoking is the new militancy of the non-smoking consumer, voter, and taxpayer. In 1973, Arizona, a state hardly known for its "radical chic," took the plunge and became the first to prohibit smoking in public places, on commercial airliners in Arizona airspace, and on buses. In 1975, Maryland enacted Article 43, Section 5411, specifically designed to protect non-smoking patients from the harmful effects of passive smoking in all health facilities. Since then, more than 30 states and hundreds of communities have followed suit, enacting ordinances to prohibit or restrict smoking in restaurants, government offices, theaters, indoor sports arenas, bus station waiting rooms, clinics, and retail stores. Minnesota has one of the toughest laws—the Clean Indoor Air Act—that bans smoking of tobacco in any public place unless a sign is posted that permits smoking.

One of the most interesting of these laws was enacted in Virginia in 1975. According to that law, if a state-employed firefighter, police officer, or deputy sheriff is disabled or dies because of heart disease, respiratory illness, or high blood pressure, the state assumes that the disease was caused by the condition of employment and, therefore, the state agrees to be fully liable for all compensation and pension claims. However, cardiovascular and respiratory diseases are also linked to smoking, as has been demonstrated during the past 20 years. Hence, some counties in Virginia have decided to screen out employees who bring those conditions upon themselves by smoking. The reasoning is the same as that of many life insurance companies that now offer low-cost policies to non-smokers. In 1985 Fairfax County in Virginia joined Alexandria and Arlington counties in demanding this written and signed pledge from new employees in public safety:

> I agree not to smoke at any time, so long as I am employed by the above-mentioned agencies (that is, police, fire, and sheriff's office), whether on or off duty, and that my failure to comply with the above-mentioned conditions of employment shall constitute good cause for my employer to terminate my employment.

This is a fine example of just how completely the public has accepted, and believes, the facts published in the annual report by the Surgeon General on smoking and health. Another example is the action taken in San Francisco, where voters approved a major public health ordinance, "Proposition P."

Proposition P is a tough law. It says, for example, that every workplace and public gathering place must segregate smokers from non-smokers. A complaint by a single non-smoker is enough to require segregation in order to protect the health and well-being of the non-smoker. In addition, if the persons responsible for a public space do not segregate the smokers after a complaint has been lodged, they are subject to heavy daily fines.

Countless school programs have initiated no-smoking rules—and this is all the more encouraging when one considers that it has been largely brought about by students with sympathetic faculty participation.

In 1984 the Food and Drug Administration approved a nicotine chewing gum, so that the physician now has a crutch to add to advice to patients that they stop smoking.

ULTIMATE GOAL

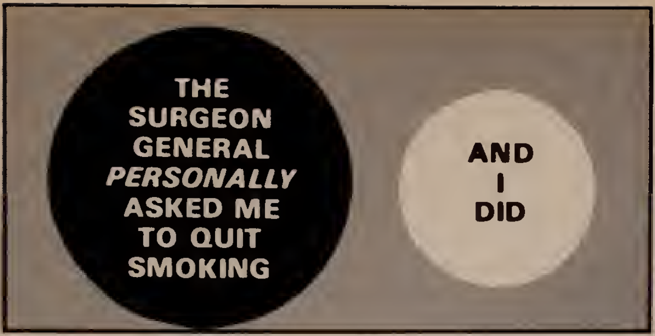
But toward what goal are we moving? Is the publication of each annual report a celebration all to itself or is it only to be another in a long shelf of annual reports? And what of the anti-smoking laws? Are they to be enacted only to make the air a little safer and sweeter—place by place and environment by environment? There has to be more purpose to our efforts than that. What objective is down the road? What kind of achievement do we want to celebrate in the future?

I believe the ultimate goal should be a smoke-free society by the year 2000.

I recently returned from the 37th World Health Assembly in Geneva, where I was the chief delegate of the United States. In 1978 the World Health Organization set as its goal health for all by the year 2000. That is a tall order. This country's goals for health for all are embodied in the 1979 publication *Healthy People: The Surgeon General's report on health promotion and disease prevention.* These goals are to be completed not in 2000 but 1990. Many countries doubt that their resources are up to the task. Others, with outside help, will reach certain attainable goals, only to see them slip after 2000.

I have sat through three such world assemblies with a certain feeling of hyprocisy, a feeling that comes from the realization that the nation not only has to do something to address the number one public health problem, but also to stop doing something—and that something is smoking.

Most of the nation's schools, churches, libraries, government agencies, and other major public institutions have already taken steps to limit, if not altogether eliminate, smoking from their premises. Most transportation companies have taken a tougher line on smoking, banning it altogether from their vehicles or aircraft or severely restricting the numbers of seats for smokers. Every hospital


THE SURGEON GENERAL *PERSONALLY* ASKED ME TO QUIT SMOKING
AND I DID

Two "campaign" buttons distributed by Surgeon General Koop.

and clinic and most doctors' offices now prohibit smoking altogether or allow only cigarette smoking in a small reserved space.

This trend has the earmarks of preparation for the achievement of a national goal. This ought to be the accepted goal for all Americans, not a goal set by the Surgeon General or the US Public Health Service. In fact, the achievement of such a goal ought to be the triumph primarily of private citizens and of the private sector and not of the government.

Call to Alms

I believe it is my duty, as Surgeon General, when I see the need and the opportunity for a far-reaching public health initiative, to come before the public and suggest it. And that is what I am doing now. I am calling on not only the American Lung Association and the American Thoracic Society but I am calling on the individual agencies that comprise the National Interagency Council on Smoking and Health to join this effort toward a smoke-free society by the year 2000.

I call upon the physicians and other health providers to participate. Fewer than 10% of physicians smoke, but all physicians must provide information clarifying risks associated with smoking, clarify risk reduction if a patient stops smoking, encourage abstinence by advice and direct service, refer patients to smoking cessation programs, and provide specific cessation and maintenance strategies.

Inasmuch as 70% of North Americans see their physicians at least once a year, 33 million of the 54 million adults who smoke could be reached annually. Even if only 10% quit, the impact on health and the economy would be enormous.

But physician contact alone is not sufficient; rather, a mix of physician, educational, and training efforts are called for. Already 90% of physicians do this with pregnant patients—let more of them do it with all patients.

I call on the American Association of Respiratory Therapists to organize children to help their parents to stop smoking. I call on the American Academy of Pediatrics to continue the efforts they have made with A Non-Smoking Generation-United States, Inc and to bring about the change in adolescent behavior that will support the effort of the Coalition on Smoking or Health as they push for the first smoke-free generation. I call on the American Medical Association to give the major preventable cause of death the emphasis it deserves from the largest group of physicians in organized medicine. I call on the American Public Health Association as the largest American public health organization to take up this cause, about which they have written and talked. I call on the March of Dimes Birth Defects Foundation, the national boards of the YMCA and the YWCA, the Boys' Clubs of America, the Boy Scouts of America, and the 4H Clubs to barrage their constituencies with a steady flow of educational material aimed at the goal of a smoke-free society by the year 2000. I call on state, county, and city health officials to work with their colleagues to support a smoke-free society.

Recently, the Chief of Staff of the Army, General John F. Wickham, eliminated low-priced liquor at army bars during the happy hour. The Navy has two excellent slogans, each with a pertinent double meaning: "Chart a course for life" and "Fitness for life." One course to chart for fitness would be the end of low-priced cigarettes. Veterans' organizations could follow suit by developing their own slogans.

I call on the mass media to take up the educational process. Every not-for-profit health agency is ready to help. I call on *Time* magazine or *Newsweek* to devote a cover story to the nation's main preventable cause of death as *Time* did recently in reference to cholesterol. There has not been such coverage since 1983, when *Newsweek* published an early report on the 1984 Report to the Surgeon General.

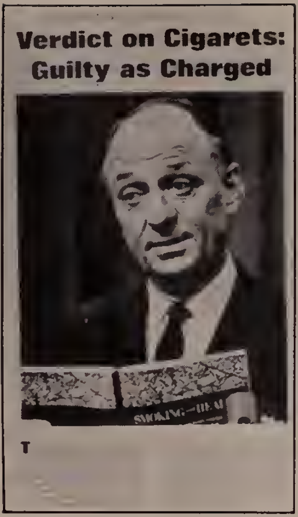
I call on manufacturers of bumper stickers, T-shirts, lapel pins, and other gadgetry to flood the market with health education materials. Experience in Australia proves what can be accomplished by community effort. The percentage of reduction was greatest in the town with the most effort in the printed media, television, and radio, supplemented by stickers, T-shirts, and balloons. The most extensively studied community project was that of North Karelia, Finland, where two five-year periods were studied. The program included service, education, and training. The news media was involved. Physicians and public health nurses provided special training and were encouraged to recommend cessation to all patients visiting clinical centers. Restrictions on smoking and point of purchase advertising were adopted. National legislation during the period raised cigarette tax revenue, limited smoking in public places, and banned tobacco advertising. The results were impressive.

I call on the executives of America who well know the health cost of smoking to heed President Reagan's words to the Health Insurance Association of America on March 13, 1984 when he spoke of disease prevention and health promotion. He said "The illness resulting from smoking is costly both to the smoker and his or her boss. A helping hand to assist employees to break the habit might be a wise investment."

What can the average citizen do? He or she can be more aggressive as a nonsmoker; demand smoke-free hotels, taxis, rental cars, waiting rooms, offices, schools, and restaurants; and demand that *Death in the West*, a classic film of 1976, which surfaced again in 1982, be shown on television as a public service.

Hippocrates, the father of medicine, said "Healing is a matter of time, but it is also sometimes a matter of opportunity." This is both the time and the opportunity to maintain the momentum of this initiative and to remind citizens that as far as smoking is concerned, their well being is largely in their own hands.

AN APPRECIATION

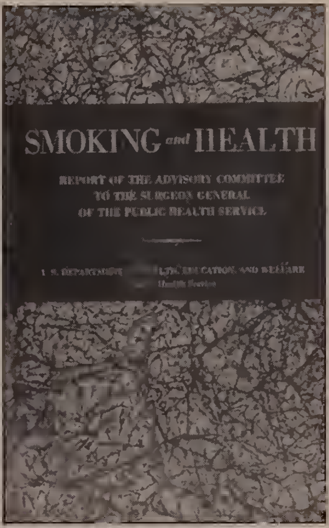


Verdict on Cigarets: Guilty as Charged

SMOKING—HEAL

T

An 11-page article appeared in *LIFE* magazine—coverage seldom equalled since then in the print media.



SMOKING and HEALTH

REPORT OF THE ADVISORY COMMITTEE
TO THE SURGEON GENERAL
OF THE PUBLIC HEALTH SERVICE

Cover of the famous report, 1964.

Luther Terry, MD, 1911–1985

A caring, courageous man

Luther Terry, MD, internist, researcher, teacher, and Surgeon General of the United States Public Health Service from 1961 to 1965, died of a heart attack on March 29, 1985. His behind-the-scenes account of the preparation of the Surgeon General's first report on smoking and health was published in the New York State Journal of Medicine *in 1983 (83:1254–1255). The following tribute, based on the eulogy delivered at Arlington National Cemetery on April 2, 1985, was written by Joseph A. Califano, Jr, who served as Secretary of Health, Education, and Welfare from 1977 to 1979.*

Great discoveries in the scientific laboratory have saved millions of lives: pasteurization of milk; vaccines to eliminate smallpox and check childhood diseases; antibiotics to stamp out bacterial infections. In the first part of the century, the infectious diseases these medicines conquered were the public health challenges of the day.

Luther Terry's public health challenge was far more difficult. It could not be met in the tranquility of a laboratory or medical campus. This quiet, private man had to face his public health challenge in public. He met that challenge. He pioneered the recognition that one's lifestyle was the killer and crippler for the last half of the 20th century. He taught us that we could do more for ourselves than any doctor or hospital could do for us.

Luther Terry did not practice public health with platitudes. He gave it to us straight and true with the first Surgeon General's report on smoking and health. He tried and convicted cigarettes of murder and mayhem by cancer, heart disease, emphysema, and chronic bronchitis. His findings and his soft but steady voice moved a reluctant Congress to act, first with warning labels, then with a ban on television advertising. He saved as many lives as Salk or Pasteur or Curie. He is as much a medical giant.

But Luther Terry was more. He was a genuine hero, a man of extraordinary courage. No powerful economic forces rose up to defend polio, or smallpox, or unsafe milk. When Luther Terry told us of the dangers of smoking, political and financial powers of magnum force rose to attack him. His warning was one they had tried to hide and did not want to talk about. But Luther Terry spoke out. Softly, but firmly he stood his ground. He persevered, and in the end he revolutionized our thinking about the causes of modern day diseases.

Luther Terry touched my life twice. For a decade I ignored his warning, but my son Joe did not. He learned about the Surgeon General's report in school, and he asked me to quit for his 11th birthday present. It was one of the hardest things I ever did, but I did it.

Four years later, while I was Secretary of Health, Education, and Welfare, the most comprehensive of all the Surgeon General's reports on smoking and health was published to celebrate the 15th anniversary of his work. The report vindicated him as a scientist and a humanitarian. It provided overwhelming evidence that his warnings were justified.

Luther Terry kept fighting to the last. His recent efforts to eliminate smoking from the workplace have marked the paths to new frontiers for his followers. Many of us have walked and will walk in his footsteps, but none of us can fill his shoes.

In searching for some words to describe this committed, caring, courageous man, I could find none better than those of Theodore Roosevelt:

> *It's not the critic who counts, not the man who points out how the strong men stumbled or where the doer of deeds could have done better. The credit belongs to the man who is actually in the arena, whose face is marred by dust and sweat and blood, who strives valiantly, who errs and comes up short again, and again, who knows the great enthusiasms, the great devotions, and spends himself in a worthy cause . . . .*

A disease model of cigarette use

John D. Slade, MD

There are many ways to interrelate the complex phenomena that result in cigarette-caused morbidity and mortality. The model developed by medical researchers to establish the nature and extent of health consequences uses smoking as the central event (Fig 1). However, more comprehensive models are helpful when one considers control and prevention strategies. In particular, for individuals familiar with conventional biomedical languages, a model framed in the language of disease, especially infectious disease, is appealing. This article develops a model of cigarette use as a disease, discusses the stages of this disease, and demonstrates parallels between cigarette marketing (the performance of business activities that direct the flow of goods and services from producer to consumer or user[1]) and tobacco lobbying techniques on the one hand and the actions and effects of agents of infectious disease on the other.

SHIFTING THE ONUS

The view that smoking is the central phenomenon in a disease process implies the formal structure depicted in Figure 1: cigarettes deliver an addicting substance plus toxins to the smoker. Whereas this model is appropriate for research of health consequences, it leaves the social context of smoking in a peripheral position, and places responsibility for the problem only upon cigarettes and smokers. Although this model can take social and cultural aspects into account, it does not accord them a prominent position.

If one shifts the critical interaction to the step that precedes smoking, the acquisition of cigarettes (usually by purchase) becomes the center of the model (Fig 2). Cigarette use begins with the decision to buy the product and continues through the act of smoking. The concept of cigarette use accounts for the fact that the cigarette purchaser buys a particular brand and may participate in that brand's image simply by possessing a given variety of cigarettes.[1-3] The brand's image is part of the product, and only owners of the product may share in the image. In this structure, the cigarette producer as well as the user explicitly participate in the central interaction.[4]

Widespread cigarette use and its consequent morbidity and mortality are modern phenomena. Prior to 1883, there was no mechanical device that rolled finely shredded tobacco into a cylinder and held it together with a thin paper. Mass production created the need for mass marketing: these have been twin phenomena in the cigarette industry since the 1880s.[5] Today, aided by modern production methods and marketing techniques, cigarettes are a multibillion dollar international industry.

Traditional scientific methods have been used to document the havoc that the use of cigarettes causes, but cigarette-caused illness is only the end result of a pathologic process that begins decades earlier. Cigarette use regularly leads to a specific array of illnesses in a manner similar to the way in which infection with the tubercle bacillus leads to a variety of illnesses, depending on which tissues become infected.

While tuberculosis is primarily a pulmonary disease, it may cause serious problems in many organs. Because of stereotyped clinical presentations, characteristic pathologic findings, and the fact that the organism can often be recovered from the affected tissue, accurate diagnosis is usually accomplished.

In a similar manner, prolonged cigarette use results in a predictable pattern of cigarette-caused pathologic changes and illness in cigarette users. Illness may be prevented if one never uses cigarettes; among users the development of illness is dose-dependent and at least partially reversible by cessation. Accurate diagnosis is impeded by the lack of

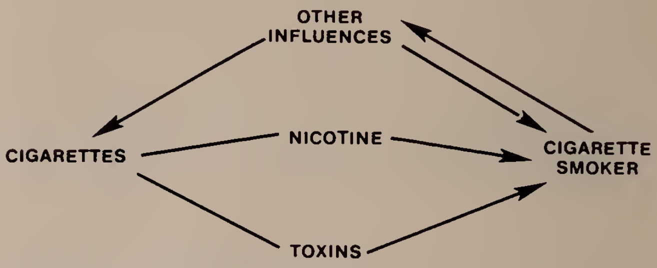


FIGURE 1. Cigarette smoking network.

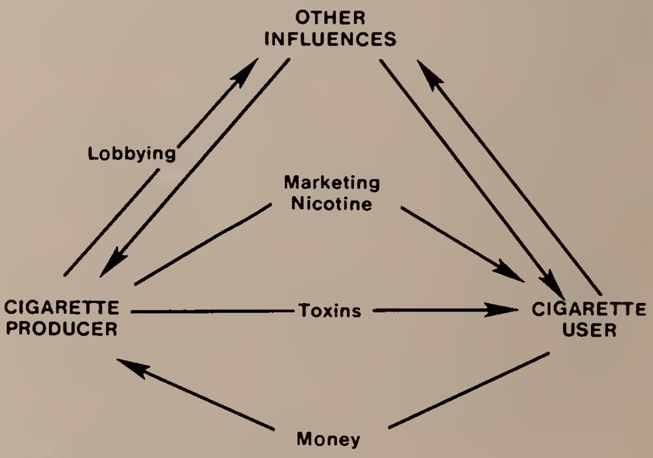


FIGURE 2. Cigarette use network.

Address correspondence to Dr Slade, Assistant Professor, Division of Rheumatology, Department of Medicine, University of Medicine and Dentistry of New Jersey-Rutgers Medical School, 254 Easton Ave, New Brunswick, NJ 08903.

pathognomonic histologic changes and the absence of a specific agent that can be recovered from a patient's tissue. As with many other diseases induced by toxins, the accurate diagnosis of a cigarette-caused illness depends on a thoughtful regard for the patient's complete history.[6]

STAGES OF CIGARETTE USE

The Table outlines four stages of cigarette use and the phenomena which characterize each stage. In stage I, the future user learns about cigarettes. He sees others use cigarettes, reads about them, learns attitudes towards them from others, learns about the different varieties and their promoted distinctions, plays with facsimiles (candy cigarettes, corn husk cigarettes), and has fantasies about smoking. Much, if not most, of this "pre-clinical" learning occurs during the first and second decades of life.[7]

The earliest clinical stage, stage II, is one of experimentation. The user actually smokes cigarettes and begins to learn other behaviors necessary to sustain use. Immediate, reversible health problems are apparent, such as nausea, coughing, and mouth and throat irritation. Thinking about cigarettes continues as the user actually learns to smoke. Stage II may, on average, continue for about two years,[8] and people who enter stage II are at enormous risk of becoming stage III users.

The Stages of Cigarette Use

	Pre-Clinical	Clinical		
Stage	I	II	III	IV
Thought	+	+	+/−	+/−
Behavior	−	+	+	+/−
Reversible organ damage	−	+	+	+/−
Latent period for permanent, symptomatic damage	−	−	+/−	+
Permanent, symptomatic damage	−	−	−	+

Stage III is characterized by the habitual use of cigarettes. By now the user has learned how to purchase cigarettes and ignition materials. He or she has experimented with a variety of brands and has settled on one or a few whose taste and image he likes. He has acquired devices in which to dispose of ashes and has usually considered or experimented with products to reduce the immediate, usually cosmetic, consequences of smoking such as bad breath. He has dealt with whatever opposition to smoking there might be among those around him in such a way that his usage can continue.

Stage III, in which the user may remain for decades, is the stage in which toxic exposures accumulate, so that the risk of major illness develops. The development of bronchitis or peptic ulcer disease may stimulate the user to cease smoking, but cancer, atherosclerosis, and emphysema may develop in a latent or symptomatically silent form at this stage. Cigarette use is sustained in this period by the addictive features of cigarette use and by environmental cues that encourage continued use. Typically, a user will purchase and smoke a particular brand of cigarettes, and will remain loyal to that brand. This pattern of use is sustained in part by the marketing program associated with the particular cigarette. Ambivalence about smoking and attempts at stopping are other common features of this stage.

A user enters stage IV when major, irreversible organ damage and illness occur. These phenomena are usually described as diseases in and of themselves, in part because of their often dramatic symptoms, and in part because there are often other causes of identical phenomena. However, the compelling evidence that much of the lung cancer, coronary artery disease, and chronic obstructive lung disease in users of cigarettes is related to cigarette use implies that the cancer or the myocardial infarction or the emphysema are only signs of an underlying disease: chronic cigarette use.

In stage IV the patient may not be a current user, and his thoughts and behaviors regarding cigarettes may not resemble those he had during earlier stages. On the other hand, usage may continue, either with the same cigarette variety or with another brand, often one imagined by the user to be safer.

In the case of tuberculosis, the infectious agent is inside the host. The factors that render the host susceptible and the interactions that lead to infection involve the agent, the host, and the environment in which both live. The situation is similar in the case of cigarette use, except that the agent remains external to the host. Cigarette use is characterized by an organized force that seeks to promote and sustain the behavior; the cigarette itself transmits the toxin nicotine, which also plays a sustaining role,[9] as well as other toxins (Fig 2). There are other important influences involved in the propagation and continuation of cigarette use, but the presence of cigarette producers, their allied industries, and their supporters in business, agriculture, government and other fields are necessary and, perhaps, given a susceptible population, sufficient. (Nearly every cultural group is at risk of cigarette use and the development of norms that support its continuation.)

OVERCOMING HOST DEFENSES

Most of the promoting and sustaining factors attributable to cigarette manufacturers can be classified as marketing. Other factors are also involved in the pathogenesis of cigarette use in a particular individual, such as the widespread acceptance of cigarette smoking, the nearly universal availability of cigarette packages, and the cultural expectation that smoking is the norm unless specifically prohibited. However, of all of these factors, marketing alone is highly organized, analytic, and deliberate. Because it is so well structured, it lends itself to further analysis.

Marketing is a major influence in shaping the thoughts of the cigarette user. This technologically advanced set of devices attacks the defenses of users in sophisticated ways. The common strategies can be compared to some of the ways in which certain infectious disease parasites outwit the body's specific immune system and take advantage of their hosts.

The specific arm of the immune system depends upon interactions among antigens, macrophages, T cells, and antibody-producing B cells. An antigen is presented to B-lymphocytes via macrophages, often with the help of T-lymphocytes. The B cells respond to this antigenic challenge with the production of type-specific antibody which, in turn, neutralizes the effect of the agent associated with the an-

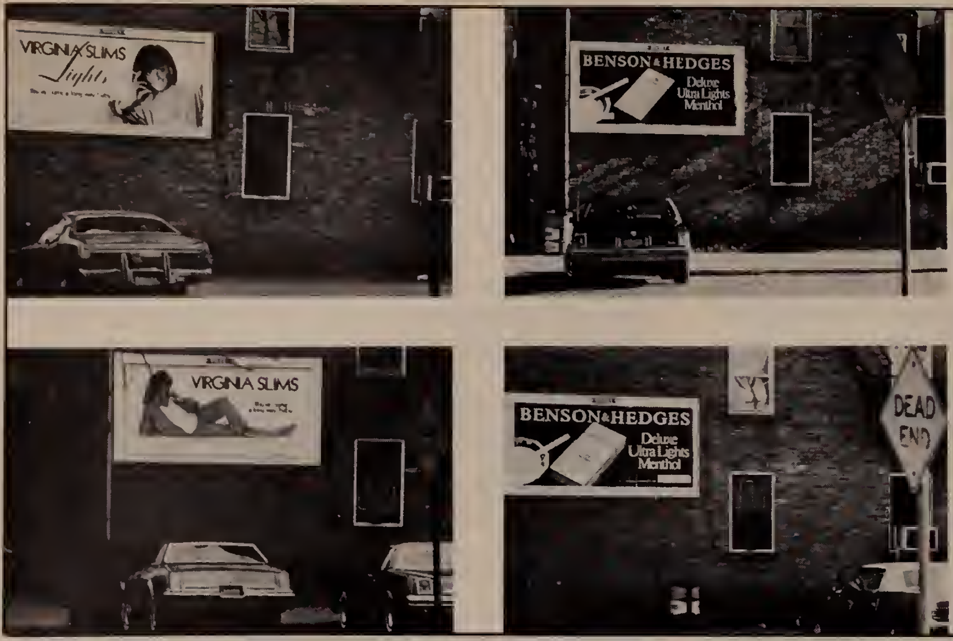


FIGURE 3. This New Brunswick, NJ billboard has featured a different poster of a Philip Morris cigarette brand every month for 15 consecutive months of observation. Top left, January 1984; top right, February 1984; bottom left, March 1984; bottom right, April 1984.

tigen. The system features a long-lived memory which permits rapid and specific response upon rechallenge by the same or similar antigens in the future. This defense is by no means certain.

Trypanosoma brucei is the hemoflagellate that causes African sleeping sickness. It has the interesting property of being able to change its major surface antigens periodically.[10,11] Thus, as the host mounts an antibody response to the latest antigen and clears the parasite from the blood, the parasite changes its surface so that the antibodies are unable to recognize the parasite.

Cigarette marketing depends upon variety. Advertisements are rotated at regular intervals so that the eye continues to be entertained and the mind pays attention. Just as one image becomes commonplace, it is changed, either for a hauntingly similar image or for a strongly contrasting one (Fig 3). The images are also presented in multiple locations; the host carries a heavy burden of advertising images which change so often that it is difficult for him to become immune or mount another appropriate defense.

Influenza A virus is notorious for its antigentic drifts and shifts. Year after year, various strains of the virus find new groups of susceptible people who lack specific antibody. The recent experience with H1N1 strains of influenza A has been that people born since H1N1 viruses were last common have been those most susceptible to these viruses. Influenza has the highest attack rates among groups with the least specific antibody. These are the people in a population who are at greatest risk for infection with particular strains of influenza.

Similarly, market segmentation, by the presentation of dozens of different cigarette brands and their extensions into brand families, is designed to divide the population into demographically and culturally defined groups at risk.[1] Virtually the entire population is at risk of using at least a few brands targeted to specific preferences, interests, and concerns. Virginia Slims (manufactured by Philip Morris) is marketed to women; Kool (Brown & Williamson) is the best selling brand among blacks; Sterling (RJ Reynolds) is sold as a status symbol, and Carlton (American Brands) is sold to people concerned about their health, who may have already developed serious cigarette-caused illnesses.

Plasmodium falciparum causes a particularly serious form of malaria. This infection is often characterized by the production of enormous amounts of immunoglobulin, very little of which is helpful in combating the disease.[10] The parasite fools the host into making a variety of antibodies that are of little benefit. There is widespread activation of the host's B cells without effective, specific antibody production.

This property of malaria is also seen in cigarette marketing practices. The RJ Reynolds Tobacco Company has provided the clearest recent example of this in its series of advertisements that seek "an open debate about smoking." [12] These advertisements confuse the casual reader, deflect criticism, and give the appearance of fairness. None has spoken directly to the established health consequences of cigarette usage; the series reassures the user that the industry is concerned, but suggests that nothing especially bad is proven about its product.

The three preceding examples, trypanosomiasis (rotational advertising), influenza (market segmentation), and malaria (promotion of confusion and denial) use antibody production as a metaphor for an individual's psychological defenses. Another strategy employed by the tobacco industry that facilitates thinking conducive to cigarette use is analogous to an attack upon the T cells in the immune system.

Helper T cells are often involved in decisions about which antigens should be presented to B cells for antibody production. The antigens that do not alarm them are not passed onto B cells for the production of specific antibody. Sometimes these T cells are, themselves, the object of attack. When they are impaired or destroyed, the organism may become immune-deficient. A virus named the human T cell leukemia-lymphoma virus-III, or HTLV-III, specifically attacks helper T cells and may create a state of immune deficiency in the host. This is associated with opportunistic infections and with the development of malignancies.[13]

The decision makers for the immune system are the T cells. In the culture in which cigarette use occurs, political leaders, mass media officials, administrators, and various other authorities make and shape decisions that influence society.

The cigarette industry not only markets its products to users and potential users but also lobbies for its interests with opinion leaders and policy makers (Fig 2). The industry is positioned so that virtually every part of the economy is affected in some way by cigarette sales. In addition to simple denial that there are any health problems and appeals to patriotism and to personal freedom, industry lobbyists have presented the political and business leadership of society with data that imply that it would be damaging to the economy to permit the cigarette market to collapse.[14,15] Illness and death from cigarette use is much more difficult to see under these circumstances, and deliberate, rational action which would lead to a substantial reduction in tobacco use becomes exceedingly difficult.

The quantitative importance of each of the four marketing and lobbying phenomena outlined above and the relative importance of these marketing and lobbying techniques compared to other influences that affect the thoughts of cigarette users remain to be determined. At the risk of over-extending the metaphors, it is tempting to point out that two drugs of proven utility in treating trypanosomiasis (suramin)[16] and influenza (ribavirin)[17] have recently been found to protect T cells from HTLV-III infection or to inhibit growth of the virus in infected tissue cultures. Thus, strategies that counter the effects of cigarette marketing may have salutary effects on lobbying phenomena, and vice versa.

CONCLUSION

Widespread cigarette use became possible just a century ago, but it rapidly became the major form of tobacco use. It is only in the past generation that compelling evidence of the harm caused by cigarette use has been developed. Largely because of the complex cultural and economic relationships involved, knowledge about the enormous losses caused by cigarettes has not yet led to large-scale, rational control measures. The model of cigarette use suggested here could serve as a framework in the development of such control measures. It may also provide a structure that can be used for further research and for the instruction of health professionals about cigarette use.

REFERENCES

1. Schultz DE, Martin D, Brown WP: *Strategic Advertising Campaigns*, 2nd ed. Chicago, Crain Books, 1984.

2. Blum A: Medicine vs Madison Avenue. Fighting smoke with smoke. *JAMA* 1980; 243:739–740.

3. Foote E: Advertising and tobacco. *JAMA* 1981; 245:1667–1668.

4. Blum A (ed): The business of smoking. *NY State J Med* 1983; 83:1324–1332.

5. Wagner S: *Cigarette Country—Tobacco in American History and Politics.* New York, Praeger, 1971.

6. Pollin W, Ravenholt RT: Tobacco addiction and tobacco mortality. *JAMA* 1984; 252:2849–2854.

7. Chapman S, Fitzgerald B: Brand preference and advertising recall in adolescent smokers: some implications for health promotion. *Am J Public Health* 1982; 72:491–494.

8. Baugh JG, Hunter S MacD, Webber LS, Berenson GS: Developmental trends of first cigarette smoking experience of children: the Bogalusa heart study. *Am J Public Health* 1982; 72:1161–1164.

9. Pollin W: The role of the addictive process as a key step in causation of all tobacco-related diseases. *JAMA* 1984; 252:2874.

10. Bloom BR: Games parasites play: how parasites evade immune surveillance. *Nature* 1979; 279:21–26.

11. Kolata G: Scrutinizing sleeping sickness. *Science* 1984; 226:956–959.

12. RJ Reynolds Tobacco Company: "Can we have an open debate about smoking?" (advertisement) *Newsweek*, February 6, 1984.

13. Gallo RC, Salahuddin SZ, Popovic M, et al: Frequent detection and isolation of cytopathic retroviruses (HTLV-III) from patients with AIDS and at risk for AIDS. *Science* 1984; 224:500–503.

14. *A study of the US tobacco industry's economic contribution to the nation, its fifty states and the District of Columbia, 1979.* Philadelphia, Wharton Applied Research Center and Wharton Econometric Forecasting Associates, Inc, 1980.

15. *A study of the US tobacco industry's economic contribution to the state and counties of Pennsylvania, 1979.* Philadelphia, Wharton Applied Research Center and Wharton Econometric Forecasting Associates, Inc, 1980.

16. Mitsuya H, Popovic M, Yarchoan R, Matsushita S, Gallo RC, Broders S: Suramin protection of T cells in vitro against infectivity and cytopathic effect of HTLV-III. *Science* 1984; 226:172–174.

17. McCormick JB, Gretchell JP, Mitchell SW, Hicks DR: Ribavirin suppresses replication of lymphadenopathy-associated virus in cultures of human adult T-lymphocytes. *Lancet* 1984; 2:1367–1369.

A glossary of tobacco terms

Several points of view are evident in the range of contemporary writings about tobacco. The major poles are the tobacco industry and its apologists and analysts on the one hand, and health professionals on the other. This glossary has been compiled as an aid to the health professional who begins to sift through the tobacco industry's literature. The list is derived from a variety of sources. It reflects the language in use among tobacco companies, investment analysts, the industry lobby, and industry-supported publications. References containing an example of each usage are provided.

An adult custom. The habit or addiction of smoking.[1]

Anti-smokers. In various contexts, this term is used to describe researchers, health care providers, parents, teachers, and others who express concerns about smoking.[2] However, people called anti-smokers by tobacco company spokespersons do not oppose people who smoke, as this term would imply. Rather, they oppose smoking and its promotion.

Basal cell hyperactivity, severe. Locally invasive squamous cell carcinoma.[3]

Big Brother. The federal government when its public health activities threaten tobacco interests.[4]

The cigarette controversy. See **The health question.**[5]

Common courtesy. Cultural practices of nonsmokers which enable tobacco use. Enabling is a concept used in the chemical dependency literature. It refers to behaviors of those around an addicted person which permit and actually encourage the continuation of an addiction despite clear, negative consequences for both the enabler and the enabled. In the chemical dependency literature, enabling is regarded as a sign of the social pathology induced by the addictive process.[2]

Corporate social responsibility. Support of fine arts and popular culture by a tobacco company for prestige, image, and influence. This term is applied to activities which are not directly related to the corporate enterprise. Thus, responsibility does not extend to the adverse consequences which might be experienced by a person who uses the company's products as intended.[6]

Excessive smoking. That amount of smoking which might cause illness. Excessive smoking is not quantified in industry writings, but it seems to represent a great amount. It may be that the industry would consider smoking excessive when the amount smoked is more than that consumed by the reader. Since a threshold is never specified in industry writings, health workers have the opportunity to add precision to this term. For instance, one might consider the threshold to be any use of tobacco.[7]

The extreme environmentalist position. The hypothesis that smoking, in and of itself, causes lung cancer and heart disease. In industry-sponsored writing, this hypothesis is contrasted with a genetic hypothesis that smoking merely happens to be more common among people who are susceptible to these diseases and who would acquire them just as often if they did not smoke.[8]

Full flavor brand. The opposite of a low tar and nicotine brand. Hence, a high tar and nicotine brand.[9]

Gas. Carbon monoxide.[10]

The health question. A tobacco industry term for the debate over whether tobacco use contributes to morbidity and mortality.[9] Actually, the discussion is about disease, not health. The question may remain unresolved in industry writings because the control measures implied by the data are repugnant to the industry. See **Scientific proof.**

Holy Grail. The cup used by Jesus at the Last Supper. An image in western literature for an unattainable goal with magical properties sought by noble heroes who encounter bizarre and incredible obstacles in the pursuit of their quest.

A legal product. Cigarettes and other tobacco products. This attribute is used to justify the continued promotion of tobacco products in paid advertising. In this usage, tobacco is contrasted with illegal products, such as marihuana and cocaine, which receive abundant publicity without charge through sensationalized news accounts.[11,12]

Market segmentation. Enlarging the population at risk of cigarette use by subdividing the population into many smaller groups at risk of using particular brands. These brands have varying images which are calculated to appeal to various specific segments or risk groups.[13]

Menthol fresh (or cool). The sensation created by the local anesthetic action of menthol.[14]

Physiological satisfaction. Relief of the nicotine withdrawal syndrome by the self-administration of the drug in the form of tobacco.[15]

Research. The acquisition of data to answer **The health question** and resolve **The cigarette controversy.** See **Scientific Proof.**[5]

Satisfaction. See **Physiological satisfaction.**

Scientific proof. Data acceptable to the tobacco industry which would answer **The health question.** See **Holy Grail.**[16]

Smokers' rights. Sanction to smoke in public. In American history, democratic rights usually have included a right to be fully and fairly informed about an issue. Physicians are better informed about the disease consequences of smoking than the general public, and the substantially lower smoking rates among American physicians compared to the general population reflect this clearer understanding of the data. The right to be informed has not been exercised to the same extent as has the right to use tobacco.[17,18]

Tar. A complex mixture of chemicals including many toxins.[19]

The tobacco industry. A group with a vested economic interest in the continued acceptance and expansion of tobacco use around the world.[9]

Vested interests. Not precisely identified, but probably nearly identical with those who hold **The extreme environmentalist position.**[20]

To suggest additional terms for this glossary, please submit the term, a definition, and a citation of its usage to John Slade, MD, Department of Medicine, St. Peter's Medical Center, 254 Easton Ave, New Brunswick, NJ 08903.

REFERENCES

1. RJ Reynolds Tobacco Company: Some surprising advice to young people from RJ Reynolds Tobacco. (advertisement) *New York Times Magazine*, May 27, 1984.
2. Scott SS: Smokers get a raw deal. *New York Times*, Dec 29, 1984.
3. Bernfield P, Homburger F, Russfield AB: Strain differences in the response of inbred Syrian hamsters to cigarette smoke. *J Natl Cancer Inst* 1974; 53:1141-1157.
4. Say what? (editorial) *The Tobacco Observer*, February 1985; 10:4.
5. *The Cigarette Controversy: Why More Research is Needed*. Washington, The Tobacco Institute, February, 1984.
6. Weissman G: Philip Morris and the arts. Denver, First Annual Symposium, Mayor's Commission on the Arts and Business Committee for the Arts, Inc, September 5, 1980.
7. Wagner S: *Cigarette Country—Tobacco in American History and Politics*. New York, Praeger, 1971:100.
8. Mangan GL, Golding JF: *The Psychopharmacology of Smoking*. Cambridge, Cambridge University Press, 1984, 208.
9. Holman RA (ed): Tobacco industry. *Wall Street Transcript* 1981; 72:1-26.
10. Advertisement for Fact cigarettes, 1978.
11. Gitlitz G: Cigarette advertising and *New York Times:* an ethical issue that's not fit to print? *NY State J Med* 1983; 83:1284-1291.
12. Blyth M: Cigarette ads in the *Ladies' Home Journal. N Engl J Med* 1984; 311:52.
13. Hollie PG: Segmented cigarette market. *New York Times*, March 23, 1985.
14. Blum A: What the Surgeon General didn't tell us, in Barrett S (ed): *The Health Robbers*, Philadelphia, Stickney, 1979:5.
15. Samfield M: *Research and Manufacturing in the U.S. Cigarette Industry*. New York, Lockwood, 1980:102, 151.
16. No scientific proof for 'public smoking' theory. *Special report: Smoking and the Public*. Washington, The Tobacco Institute, undated.
17. RJ Reynolds Tobacco Co: A message from those who do to those who don't. (advertisement) *Newsweek*, May 28, 1984.
18. Myers ML, Iscoe C, Jennings C, et al: *Staff Report on the Cigarette Advertising Investigation*. Washington, Federal Trade Commission, May, 1981.
19. Blum A: Butting in where it counts. *Hospital Physician* 1980; 16(4):22-35.
20. Sociologists examine cigarette controversy. (book review) *The Tobacco Observer*, 1984; 9(5):3.

The underemphasis on smoking in medical education

K. H. Ginzel, MD

Wynder[1] has expressed concern about "the general apathy of the medical and scientific professions toward tobacco related issues," a sentiment echoed by others.[2-4] Since cigarette smoking is the leading preventable cause of disease, disability, and death,[5-7] this apathy is a serious matter. What is its origin? Is the exposure of the student in the health professions to the tobacco issue inadequate? How does it compare to the coverage of alcohol-related problems and drug abuse? The unfortunate exemption of tobacco from the Food and Drug Act of 1906 and its subsequent revisions and extensions detracts from the fact that tobacco is not less dependence-producing than other addictive drugs. The same is true for alcohol. As a preliminary probe, the following analysis focuses on a regular constituent of every medical school curriculum—the pharmacology course—which deals exclusively with drugs and traditionally covers the topic of substance abuse.

PHARMACOLOGY TEXTS: UNEVEN EMPHASIS?

In 21 American, three British, and six German editions of 15 different pharmacology textbooks published after 1960, the number of pages covering tobacco, alcoholism, and drug abuse were counted. The pharmacologic description of nicotine, alcohol, and drugs with abuse liability, without reference to their recreational use and abuse, was not included. The assumption is made that the relative amount of space devoted to these topics reflects their rating by the experts in the field and guides the individual course coordinator in alloting lecture time and emphasis. In Figure 1, the American sample, consisting of 21 editions of 10 different textbooks, shows that tobacco occupies 0.05% of total book pages, while alcoholism and drug abuse command 5 and 40 times as much space. Comparisons are made between the means of individual percent values. The relative coverage of the three topics within their combined total is 2.8% for tobacco, 14.5% for alcoholism, and 82.7% for drug abuse (Fig 1). The order is reversed for the estimated annual mortality from diseases and accidents associated with tobacco use, alcoholism and drug abuse (Fig 1).

British and German textbooks devote 7 and 17 times as much space to tobacco, respectively, as do the American ones. During the 20 years following the Surgeon General's first report on smoking and health in 1964, there was little change in tobacco coverage by the American sample, except for a modest increase in the Goodman and Gilman text from 0.1% of total book pages (1.7 pages) in 1965 to 0.17% (3.2 pages) in 1980. In contrast, the leading British textbook (Bowman and Rand) expanded the coverage of tobacco from a mere mention (0.01% = 0.1 pages) in 1968 to a 16-page section (0.85%) in 1980, thus devoting the largest actual space to tobacco of all texts examined. Ever since its inception in 1975, the German pharmacology text by Forth et al has maintained seven pages of coverage of tobacco (1.2% decreasing to 0.9%). Significantly, tobacco is placed in the section on "Important Poisons."

Based on content, the message one receives from the majority of medical pharmacology texts in the United States as to the significance of the health threat of smoking is one of considerable uncertainty and controversy. Where the danger is stressed, its impact is diminished due to small print, lack of space, and omission of epidemiologic data in the form of graphs and tables, which abound in the British and German texts.

PREVENTION LOWEST ON TOTEM POLE

The results presented here are reminiscent of the findings of Jaffe,[9] who reports "that not one of the standard works in psychiatry treats the problem of tobacco use as seriously as the problem of glue-sniffing." Tobacco suffers similar neglect in a popular pharmacology text, where it is submerged under subsections of miscellaneous drugs on a par with nutmeg and cantheridine, yet exceeded in coverage by topics such as morning glory seeds.

It is not likely that pharmacology and psychiatry are the exceptions, while other courses accord tobacco the needed emphasis. The two leading texts in medicine rank drug abuse first, alcohol second, and tobacco last with 0.17% and 0.24% coverage, respectively. Pediatric texts also give tobacco the least exposure. Other courses that address behavioral, social, and public and family health issues, vary from school to school in name, content, and length, and do not lend themselves to evaluation. The majority of textbooks in behavioral sciences, preventive medicine, and family medicine allot from 0.2% to 0.4% of their total pages to tobacco, whereas considerably more space is devoted to alcohol and drug abuse. Besides self-contained chapters on tobacco, there may be several brief references to smoking dispersed among other pertinent sections. The only noteworthy exception to the rule is Maxcy-Rosenau's text *Public Health and Preventive Medicine* (11th edition), which, with a detailed 10-page section (0.5% of total pages) and numerous reinforcing remarks throughout the book, accords smoking substantially more space than alcohol and drug abuse.

During the past 40 years, the Association of American Medical Colleges has repeatedly cited the need to teach prevention as an integral part of clinical medicine, but this effort has met with limited success.[10] The two electives with the smallest enrollments among the clinical specialties taken by 10,279 students graduating from American medical schools in 1983 were preventive medicine (1.5%) and public health (2.1%).[11] Hence, it is not realistic to hope that, as far as tobacco is concerned, preventive medicine or an equivalent course is making up for the deficiencies in the rest of the curriculum. (The neglect of the

---

Address correspondence to Dr Ginzel, Professor, Department of Pharmacology and Interdisciplinary Toxicology, University of Arkansas for Medical Sciences, Little Rock, AR 72201.

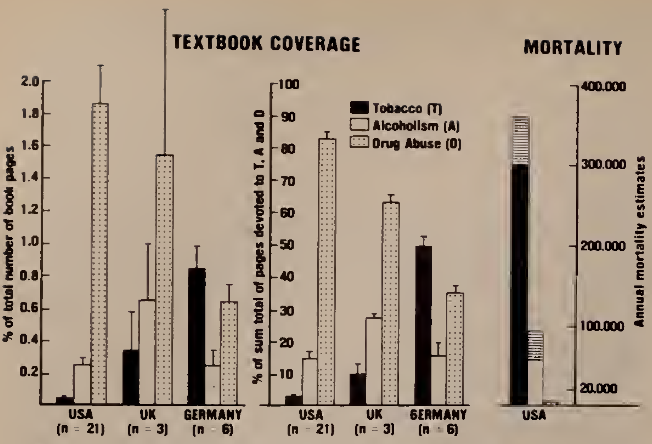


FIGURE 1. Pharmacology textbook coverage in comparison to mortality estimates for tobacco, alcoholism and drug abuse. Sets of three bars each denote the percent book space (mean + standard error) alloted to tobacco (left bar), alcohol dependence (alcoholism) (center bar), and drug abuse (right bar). Percent values refer to total number of book pages (left section) and to the sum total of pages of the three topics combined (middle section). On the right, mortality estimates for tobacco,[5,7] alcohol problems[8] and drug abuse (personal communication, National Institute of Drug Abuse) are presented. The shaded portions at the top of the columns for tobacco and alcohol problems indicate the range of these estimates. Textbooks used were: USA: Goodman, Gilman: *The Pharmacological Basis of Therapeutics*, 3rd through 6th ed.; Drill: *Pharmacology in Medicine*, 3rd and 4th ed.; DePalma: *Basic Pharmacology in Medicine*, 1st and 2nd ed.; Meyers, Jawetz, Goldfien: *Review of Medical Pharmacology*, 2nd ed.; Katzung: *Basic and Clinical Pharmacology*, 1st ed.; Goth: *Medical Pharmacology*, 6th, 7th, 10th and 11th ed.; Cutting: *Handbook of Pharmacology*, 4th and 7th ed.; Bevan, Thompson: *Essentials of Pharmacology*, 1st and 3rd ed.; Craig, Stitzel: *Modern Pharmacology*, 1st ed.; Melmon, Morelli: *Clinical Pharmacology:* 1st and 2nd ed. United Kingdom (UK): Modell, Schild, Wilson: *Applied Pharmacology*, 1st ed.; Bowman, Rand, West: *Textbook of Pharmacology*, 1st ed.; and Bowman, Rand, 2nd ed. Germany: Hauschild: *Pharmakologie und Grundlagen der Toxikologie*, 2nd ed.; Kuschinsky, Lullmann: *Textbook of Pharmacology*, 1st ed.; Forth, Henschler, Rummel: *Allgemeine und Spezielle Pharmakologie und Toxikologie*, 1st throught 4th ed.

smoking issue is not confined to medical education. For example, two current pharmacology texts in nursing of more than 1,000 pages each devote 0% and 0.05% of space to tobacco. Nicotine is only mentioned as an insecticide.)

It is unlikely that the lack of textbook coverage is compensated for by verbal communication or course outlines. Although a comprehensive survey of course hours, such as one that examined drug abuse and alcoholism teaching in US medical and osteopathic schools,[12] has not been made for tobacco, it seems that tobacco is hardly mentioned in medical pharmacology courses. Further support for this impression comes from a recent document that defines the "Knowledge Objectives in Medical Pharmacology."[13] It recommends that approximately three hours (out of a total of 98 lectures) be assigned to topics of drug abuse (two hours for drug dependence and an additional hour or so to cover amphetamines, cocaine, and laws concerning drug abuse) and one hour to "ethanol-alcoholism." In addition, a one-hour clinical round table discussion is devoted to drug abuse. There is not a single lecture designated to address tobacco use. The pharmacology of nicotine is to be dealt with in a traditional manner among "drugs acting at autonomic ganglia," with 15 to 20 minutes of discussion. "Smoking and lung cancer" appears as one of many items in the toxicology section, with the qualification "if not covered adequately in pathology"; the time available for this small segment among the topics listed can hardly amount to more than 10 minutes. (Regrettably, responses to inquiries about medical school courses in pathology suggest that little attention is devoted to tobacco beyond mentions of smoking in connection with lung cancer, emphysema, and coronary heart disease.) The space devoted to tobacco-related diseases in pathology textbooks is less than 0.1%, which is 5 to 10 times less than the coverage of alcohol-related problems. An argument often voiced to defend the minimal attention to the tobacco issue is the assumption that "everyone knows all about it." However, a Federal Trade Commission report[14] published in 1981 revealed that half of the nation's pregnant women were uninformed about the deleterious effect of smoking on the unborn child, that Americans generally were not aware of the relationship between smoking and heart disease, and that the public underestimated the connection with lung cancer.

"IRRATIONAL IMMUNITY"

Wynder's[1] criticism of the scientific professions' neglect of the tobacco issue is poignantly borne out by the symposium in 1972 on the "Contributions of the Biological Sciences to Human Welfare" sponsored by the American Biology Council.[15] In the 144-page proceedings, published eight years after the first Surgeon General's report on smoking and health and replete with data on environmental hazards including noise, smoking is only mentioned in passing: "Lung cancer has been correlated with air pollution as well as with other factors, such as smoking habits." This statement was made despite the pioneering work of Hammond and Horn 14 years earlier[16] that documented the overwhelming influence of smoking on lung cancer, which dwarfs any impact air pollution may have. Clearly, the nonrepresentation and misrepresentation of the tobacco issue by such a prestigious assembly of bioscientists could not have been due to a lack of reputable research in the field of smoking. The proceedings were later published as a paperback (Plenum Press) to serve as course material for college and university students.

In medical school curricula, the present lack of attention to tobacco contrasts sharply with the recognition that "when measured by morbidity and mortality, cigarette smoking is now the most serious, as well as the most widespread form of addiction in the world."[7] According to Fort,[17] "our priorities in the drug field are grossly misplaced" in that "a rational society would start out with alcohol and tobacco . . . our two hardest drugs." The relative priorities are reflected in President Reagan's 1985 budget proposal for health education which allots $472 million for "Alcohol and Drug Abuse" but only $3.5 million for "Smoking and Health."[18] The practice of treating drug addiction and alcoholism while condoning cigarette smoking as if it were unrelated to the underlying problem of poly-drug abuse typifies the prevailing unbalanced approach and may well be responsible for the high rate of recidivism following treatment. Jaffe, who was instrumental in achieving the recent listing of "tobacco use disorder/tobacco dependence" in the International Classification of Diseases (ICD #9, 1979) and the American

Psychiatric Association's Diagnostic and Statistical Manual (DSM-III, 1980), was puzzled as to why tobacco has attained such irrational immunity in contemporary society, a situation that had not existed before World War I and during the last century.[9] Because of this relatively recent change in attitude, the lack of behaviorally disruptive effects seen with alcohol and illicit drug abuse cannot suffice as an explanation. The most compelling answer seems to lie in the unprecedented promotion of cigarettes (now above $1.5 billion annually in the United States), which has succeeded in creating and maintaining for smoking a deceptive imagery of social acceptability[19] and all-around goodness so pervasive and persuasive that it manipulates decision-making of a large cross-section of the population, including scientists.

FROM KNOWLEDGE TO ACTION

The identification by Pollin of "substance abuse as a generic phenomenon that includes tobacco" and of "smoking as a gateway drug to use of stronger or illicit drugs"[20] calls for a fundamental re-orientation of priorities to counteract drug abuse. Whereas tobacco education deserves top priority within the larger field of substance abuse, the total coverage of the latter should be expanded to meet the need for greater emphasis on alcoholism and drug abuse.[14,21,22] A hopeful trend in this direction is reflected in a study advocating that research funding on addictions be brought "more in line with the carnage they wreak."[23] It is hoped that medical colleges and other health professional schools will also be able to make appropriate adjustments in their curricula without having to wait for textbooks to provide the new material.

This article will not spell out a detailed syllabus of tobacco education. Suffice it to say that the mere acquisition of knowledge of the medical aspects of tobacco use is not enough. To be effective in dealing with a problem as complex and ubiquitous as cigarette smoking, especially in light of the onset of smoking in childhood, health professionals must become familiar with the social pressures exerted by the mass media, and by friends and parents who smoke,[24] and must acquire the necessary motivation, attitude, and skills to intervene. As has been highlighted in the efforts of one physicians' organization (DOC—Doctors Ought to Care), it is imperative that physicians and allied health personnel become involved in school health education projects designed to prevent the young from ever starting to smoke.[25,26] Their success will depend on stressing the immediate and long-term effects of smoking and on unmasking the deviousness of tobacco advertising. Chronic illness beginning in childhood might be prevented by early risk identification and intervention, currently a neglected area of pediatric practice.[27] Analogous to epidemic disease, drug abuse, including smoking, can be viewed as transmissible[28]—possibly preventable by "psychologic inoculation" designed to develop resistance to social pressure[29]—but difficult to contain if allowed to spread. Once the propagation is halted, the epidemic often declines rapidly. There is compelling evidence for a link between a variety of eduational programs administered by health professionals and smoking cessation.[30] The effectiveness in promoting smoking cessation of even minimal intervention by the general practitioner has also been documented,[31] but physicians' personal health habits and attitudes appear to affect their initiative in counseling.[4] There is no area in preventive medicine in which emphasis will yield greater returns in better health, saved lives, and reduced medical costs than the prevention and cessation of smoking.[20] "Too many of us," writes Geiger,[32] "still treat the rat bites and ignore the rats; treat the lead poisoning and ignore the plaster on crumbling tenement walls." One should add the following: and treat lung cancer, cardiovascular disease, and stomach ulcers but ignore cigarette smoking and the circumstances that lead to it.

REFERENCES

1. Wynder EL: Interrelationship of smoking to other variables and preventive approaches, in Jarvic ME, Cullen JW, Gritz ER, Vogt TM, West LJ (eds): *Research on Smoking Behavior.* NIDA Research Monograph 17, Washington, DC, US Government Printing Office, 1977.
2. Toynbee P: A smoker's dilemma. *World Medicine* 1982; 16:83.
3. Cohen SB: Are physicians to blame if patients smoke? *NY State J Med* 1983 83:1295.
4. Wells KB, Lewis CE, Leake B, Ware JE: Do physicians preach what they practice? *JAMA* 1984; 252:2846–2848.
5. *Smoking and Health: A Report of the Surgeon General,* Dept of Health, Education, and Welfare publication 79-50066. Public Health Service, 1979.
6. *The Health Consequences of Smoking—Cancer: A Report of the Surgeon General,* Dept of Health and Welfare, Public Health Service, 1982.
7. Pollin W: Tobacco addiction and tobacco mortality. *JAMA* 1984; 252:2849–2854.
8. National Center for Health Statistics, Vital Statistics of the United States, 1972, Vol. II, Washington, DC, US Government Printing Office, 1975.
9. Jaffe JH: Tobacco use as a mental disorder: the rediscovery of a medical problem, in Jarvik ME, Cullen JW, Gritz ER, Vogt TM, West LJ (eds): *Research on Smoking Behavior*, NIDA Research Monograph 17, Washington, DC, US Government Printing Office, 1977.
10. Barker WH, Jonas S: The teaching of preventive medicine in American medical schools. *Prev Med* 1981; 10:674–688.
11. Physicians for the 21st Century. Medical Education in U.S. and Canada. *J Med Educ* 1984; 59:Table 17, p. 53.
12. Pokorny A, Putnam P, Fryer J.: Drug abuse and alcoholism teaching in US medical and osteopathic schools. *J Med Educ* 1978; 53:816–824.
13. *Knowledge Objectives in Medical Pharmacology,* prepared by the Association for Medical School Pharmacology, Committee on Essential Knowledge Base in Pharmacology, Fisher WJ (chairman), January 1985.
14. Marwick C: Medical news. *JAMA* 1984; 252:2797–2799.
15. Contributions of the biological sciences to human welfare. *Fed Proc* 1972; 31:TF1-144.
16. Hammond EC, Horn D: Smoking and death rates—report on forty-four months of follow-up of 187,783 men. *JAMA* 1958; 166:1294–1308.
17. Fort J: Social aspects of research with psychoactive drugs, in Gamage JR, Zerkin EL (eds): *Hallucinogenic Drug Research,* Beloit, WI, Stash Press, 1970.
18. *Health Education Reports* 1984; 6:3.
19. Hoffmann WE: The impact of tobacco advertising and promotion on cigarette consumption, in Ramstrom LM (ed): *The Smoking Epidemic. Proceedings of the Fourth World Conference on Smoking and Health.* Stockholm, Almquist and Wiksell Int, 1980.
20. Pollin W: Foreword, in Jarvik ME, Cullen JW, Gritz ER, Vogt TM, West LJ (eds): *Research on Smoking Behavior.* NIDA Research Monograph 17, Washington, DC, US Government Printing Office, 1977.
21. Pokorny AD, Solomon J: A follow-up survey of drug abuse and alcoholism teaching in medical schools. *J Med Educ* 1983; 58:316–321.
22. Ewan CE, Whaite A: Training health professionals in substance abuse: a review. *Int J Addict* 1982; 17:1211–1229.
23. ADAMHA funding pressed, news and comment: *Science 85* 1985; 227:147–149.
24. Evans RI, Rozelle RM, Mittelmark MB, Hansen WB, Bane AL, Havis J: Deterring the onset of smoking in children: knowledge of immediate physiological effects and coping with peer pressure, media pressure, and parent modeling. *J Appl Soc Psychol* 1978; 8:126–135.
25. Blum A: Medicine vs Madison Avenue, *JAMA* 1980; 243:739–740.
26. Richards JW Jr: School health, in Taylor RB (ed): *Family Medicine: Principles and Practice,* 2nd ed, New York, Springer-Verlag, 1983; 1876–1884.
27. Williams CL, Arnold CB, Wynder EL: Primary prevention of chronic disease beginning in childhood. *Prev Med* 1977; 6:344–357.
28. Bejerot C, Bejerot N: Exposure factors in drug use, abuse and addiction, in Fisherman J (ed): *Basis of Addiction.* Berlin, Dahlem Konferenzen, 1978.
29. McGuire WJ: Inducing resistance to persuasion: some contemporary approaches, in Berkowitz L (ed): *Advances in Experimental Social Psychology,* New York, Academic Press, 1964, vol. 1.
30. Farquhar JW, Magnus PF, Maccoby N: The role of public information and education in cigarette smoking controls. *Can J Public Health* 81; 72:412–420.
31. Russell MAH, Wilson C, Taylor C, Baker CD: Effect of general practitioners' advice against smoking. *Br Med J* 1979; 2:231–235.
32. Geiger J: Hidden professional roles: the physician as reactionary, reformer, revolutionary. *Social Policy* 1971; 1:24–33.

Perspectives on the economic magnitude of cigarette smoking

JAMES M. SHULTZ, MS

The scientific research indicting cigarette smoking as the principal preventable cause of premature death and excessive lifetime morbidity in the United States is vast and unassailable. In contrast, the literature that translates the health toll into economic costs is humble in size but creative and energetic in scope. Health economists have examined the costs of smoking from several vantage points: prevalence-based calculations of national economic costs of smoking; adaptation of national estimates to calculate statewide smoking costs; estimates of the costs of smoking to business; and prospective, incidence-based estimation of the expected costs to individuals who smoke.

NATIONAL COST OF ILLNESS STUDIES

The history of economic costs of illness (COI) calculations has been chronicled.[1,2] The methodology has been formalized and upgraded by Rice and colleagues during the last two decades.[3-5]

COI calculations are aggregated at the level of major diagnosis categories and include estimates of both direct medical care costs and indirect costs. Direct costs are the costs of medical care in the prevention, diagnosis, and treatment of illness and disease, and the rehabilitation of patients. Such costs include personal health expenditures—including hospital admissions, outpatient care, nursing home care, home health care, services of primary physicians and specialists, services of non-physician health practitioners, and medication costs—and support costs for research, public health activities, construction of medical facilities, and administration of health programs.

Indirect costs result from output lost because of cessation or reduction of productivity due to death or disability. The principal measure of indirect costs is lost income. Indirect costs typically account for age-specific and gender-specific life expectancies, patterns of earnings at different ages, labor force participation rates, and the imputed value of housekeeping services.

Psychosocial costs of illness are frequently noted but seldom translated into economic parlance.

In addition to total national COI estimates, detailed disease-specific estimates have been presented which illustrate the considerable sophistication of the methodology.[6] Formal guidelines for prevalence-based COI calculations have been developed to systematize future research in this area.[7]

ECONOMIC COSTS ATTRIBUTABLE TO SMOKING

In the United States. Turning from the relatively pure health economics of disease to the estimation of the costs of cigarette smoking, the bridging of economics and epidemiology becomes necessary. In addition to cost estimates, it is necessry to determine the diseases that are causally linked to smoking and to determine the fraction of costs of these diseases attributable to smoking.

The earliest estimates of smoking-related costs were critiqued in 1977 by Luce and Schweitzer,[8] who noted the "imprecise analytical basis" of these figures. The three estimates cited were those of Soper[9] of $5.3 billion (1966 dollars), including both direct and indirect costs; Williams and Justus[10] of $4.23 billion (1970 dollars) in direct medical care costs; and Walker[11] of $11.5 billion (1974 dollars) in direct health care costs.

Luce and Schweitzer[8] proceeded to calculate the subset of costs attributable to smoking, working from global percentages of all circulatory, neoplastic, and respiratory diseases attributable to smoking (based on Boden,[12] applied to the 1972 cost estimates for these diagnostic categories from Cooper and Rice[4]). Inflated to 1975 dollars, the estimate was $25.9 billion, composed of $7.5 billion in direct costs, $12.3 billion in indirect mortality costs (the costs of lost income due to premature death), $5.9 billion in indirect morbidity costs (the costs of lost income from disability from nonfatal smoking-related disease), and $0.2 billion in cigarette-ignited fire losses. In a subsequent article,[13] the identical methodology was used and was inflated to 1976 dollars, producing an estimate of $27.5 billion.

Kristein[14] calculated the cost of "heavy cigarette smoking" (one pack plus per day) using two strategies. First, based on estimates of excess medical utilization, excess absenteeism, and premature mortality, Kristein estimated smoking-related costs at $20.3 billion (1975 dollars), including direct costs of $5.2 billion, indirect mortality costs of $12.0 billion, and indirect morbidity costs of $3.1 billion. He validated this estimate against a separate calculation using smoking-attributable fractions of respiratory, circulatory, and neoplastic diseases applied to Cooper and Rice[4] cost data to produce an estimate of $20.8 billion. Kristein and Grove[15] translated these large dollar figures into colloquial terms: each cigarette was responsible for 1.35 cents in direct medical costs and 3.15 cents in gross

---

Address correspondence to Mr Shultz, Research Scientist, Minnesota Center for Nonsmoking and Health, Minnesota Department of Health, 717 Delaware St, SE, Minneapolis, MN 55440.

national product loss (1978 dollars), the equivalent of 90 cents per pack sold.

The most recent enhancement to national smoking-attributable cost estimates was performed using 1980 national COI data.[5] Rice, former director of the National Center for Health Statistics (NCHS), and a major force in developing COI methodology, used these 1980 data to estimate smoking costs to the nation.[16] Her method improved on the scheme of Luce and Schweitzer by using gender-specific smoking-attributable risks for individual disease codes and then summing smoking-attributable deaths across all diseases within a diagnostic category. The mortality ratio (smoking-attributable deaths/total deaths) for each diagnostic category was used as a multiplier to determine smoking-attributable costs for each category.

Costs of smoking-attributable disease in 1980 were estimated to total $42.2 billion. This included an estimated $16.1 billion from direct health care costs (7.3% of the national total of $219.1 billion). This also included $19.2 billion in indirect mortality costs and $6.9 billion from indirect morbidity costs. Rice and Hodgson are refining indirect morbidity calculations based on recently analyzed NCHS data which classify disability by smoking status.

Inflating medical and lost income costs separately to 1983 dollars yields an estimate of $55.5 billion in smoking costs. Assuming a 1983 population of 243 million people, this equates to $228 per US citizen or to $1,028 per adult smoker (assuming 54 million smokers).

In recent testimony on smoking-related legislation, Surgeon General C. Everett Koop, MD,[17] referred to the "annual smoking and health bill of $39 billion . . . for personnel and medical technology and insurance and lost wages."

A study conducted by Forbes and Thompson[18] produced estimates of smoking-linked disease costs throughout the lifespan, including the costs of fetal complications and childhood exposure to passive smoking. Direct cost estimates were equivalent to 15% of male and 10% of female per capita health costs in Canada. These authors used a methodology based on morbidity ratios for diseases related to smoking during each of six eras of the lifespan. They constructed a hypothetical nonsmoking population and determined the excess inpatient hospital utilization for cigarette smokers. Annual excess smoking-related costs were equivalent to $36 per man and $29 per woman (1980 dollars). Indirect costs were not estimated.

A second Canadian study[19] examined excess smoking-linked direct costs by cost center, such as hospital admission, rather than by diagnosis category. These authors also included indirect costs and property losses from cigarette-ignited fires. Total costs attributable to smoking were more than 170% of the gross consumer expenditure for tobacco products.

Conversely, a Swiss study[20] indicated that higher annual costs of medical utilization by smokers were compensated by the longer lifetime of medical utilization by nonsmokers. Persons who never smoke cigarettes live longer (a life expectancy increment of 4.1 years at birth) but use medical services heavily in their last years of life. Because of these reciprocal effects, lifetime medical costs per individual were found to be indistinguishable for smokers and nonsmokers. These investigators modeled the concept that persons who smoke are at increased risk due to behavioral factors other than cigarette smoking per se. This study did not estimate the indirect cost associated with smoking, a substantial figure based on the decrement in life expectancy.

On the state level. COI methodology has been adapted to state-level calculations of the costs of smoking. Following Rice and Hodgson,[16] the smoking-attributable costs to the state of Minnesota have been calculated.[21] In 1983 dollars, cigarette smoking cost Minnesota 82 cents per pack sold in direct medical costs and an additional 66 cents per pack sold in indirect mortality costs. In a computational exercise performed by Cady,[22] smoking-attributable direct costs were estimated to be $1.10 per pack sold in Massachusetts. Following the Minnesota methodology, calculations for the state of Washington produced estimates of smoking-related costs of 78 cents per pack sold in 1981.[23] For comparison, national direct costs were equivalent to 77 cents per pack sold in 1983 ($23.0 billion in direct costs/30 billion packs sold).

Spreadsheet softwear will be available from the Minnesota Department of Health which will allow other states to tailor smoking-attributable costs to the age, gender, smoking prevalence, and disease rate characteristics of the state.[24]

Critique of prevalence-based estimates. Prevalence-based COI studies of this genre for national and state populations provide a comprehensive framework for estimating smoking-attributable costs. They allow inclusion of all diseases linked to smoking; they are amenable to direct comparisons with national medical and labor cost indices; they represent the most time-tested economic cost methodology; and formal guidelines for prevalence-based COI calculations have been developed.

The accuracy of using mortality comparisons to approximate morbidity ratios varies by diagnostic category. Use of mortality comparisons suggests that excess smoking-related costs are incurred at the end of life rather than throughout the lifespan.[18] Thompson and Forbes[25] provide a critique of the assumptions that underlie current COI methodology and offer an alternative mathematical strategy for cost calculations. New data using direct morbidity comparisons for smokers and nonsmokers will enhance COI estimates now in progress (Rice, personal communication).

However, use of morbidity comparisons is not a complete solution. Excess morbidity in smokers is not as well documented by age, gender, and diagnosis code as excess mortality; and data are scant on the use of outpatient medical services. The logic of cost estimation assumes that, for a given degree of illness, smokers and nonsmokers will use medical services at comparable rates. This assumption is not supported by Vogt's and Schweitzer's[26] study of smoking characteristics and medical utilization patterns among the Kaiser-Permanente health plan population. Persons who smoked had an excess of 20% more hospital days/1,000 person-years compared to nonsmokers (less than expected), and outpatient utilization did not differ between current smokers and persons who had never smoked. Smokers were less likely to seek preventive medical services. Despite increased rates of illness among children of parents who smoke, children in nonsmoking households used more outpatient services (primarily preventive services) and similar amounts of inpatient medical care compared to children in smoking households.[27] Based on these patterns of differential utilization, the authors calculated excess costs attributable to smoking to be $1.4 billion to $2.1 billion ($6.7 billion to $10.1 billion in 1983 dollars). This range is much smaller than the $23.0 billion (1983 dollars) in direct costs calculated from Rice and Hodgson.[16] The discrepancy stems from several sources: Vogt's use of more conservative estimates of medical utilization; analyses restricted to adult disease only; analyses restricted to hospital and physician services only (about 30% of direct costs are from other cost centers); and nonequivalence of a health maintenance organization population to the total population in utilization rates and casemix.

For indirect cost calculations, the controversial issues of selecting a discount rate for estimating the present value of future earnings and the appropriateness of including non-market costs (such as the imputed value of household services)—issues inherent in the human capital method of attributing economic value to human life—affect smoking-specific calculations.[28]

In summary, sources of error in the application of prevalence-based COI methods to lifestyle behaviors such as cigarette smoking include imprecision in attributable risk estimates, incomplete data on differential morbidity for smokers and nonsmokers, incomplete data on patterns of medical utilization by smoking status, the gross aggregation of economic data which pools costs for large categories of disease rather than for individual diseases, and error in the underlying total COI estimates.

For the employer. Medical cost concerns of the corporate community have spawned estimates of smoking costs at the worksite. Kristein[29] estimated that the average employee who smokes creates between $400 and $800 in excess costs to the employer (1983 dollars). Eight items contribute to this total (in order of magnitude): productivity losses; increased health insurance costs; employer liability for diseases jointly linked to smoking and occupational exposures; absenteeism; passive smoking-induced health costs among nonsmokers; increased life insurance costs; workers' compensation costs; and fire losses.

Regarding productivity, Kristein's high estimate (about $200) was based on a loss of one minute per working hour—8 minutes per working day—due to time spent in smoking rituals. Smokers do waste a finite amount of time performing their smoking behavior. The productivity issue may be a bogus argument, however, because smoker-nonsmoker differences in productivity, controlling for demographic variables, have not been studied.

Included in Kristein's discussion of productivity losses and not reflected in his dollar estimates are other factors: clean-up costs, damage to equipment and furniture, and higher ventilation costs. The issue of ventilation costs is salient because of rising energy costs. Extra ventilation costs per smoking employee were estimated to be $50 per year per smoker (1983 dollars) to maintain air quality standards, with smokers generating 87% of the total demand for ventilation.

At the corporate level, excess direct health care costs attributable to smoking are reflected in inflated health insurance costs. Kristein assigned 75% of excess smoking-related health care costs to businesses. His high estimate of excess health insurance costs—$210 per smoker per year—was based on the national cost estimates of Luce and Schweitzer.[13] His low estimate ($105) was based on the medical utilization study by Vogt and Schweitzer.[26]

Occupational health costs were based on court cases that have set a precedent for finding the employer liable in cases where employees who smoke are disabled by a disease which is synergistically related to smoking and to an occupational exposure. Kristein's estimate regarding absenteeism was based on Surgeon General's reports[30] that smokers have 33% to 45% excess absenteeism compared to their nonsmoking counterparts, the equivalent of two days per smoker per year.

An alternative analysis of smoking costs to employers was conducted by Weis,[31] with a total cost estimate of $4,600 (1981 dollars), a full order of magnitude greater than the estimate by Kristein. Weis addressed many of the same cost factors as Kristein, but his estimates were much higher. As an example, Weis's lost productivity costs were 9 times greater than the corresponding high range estimate from Kristein, the result of attributing more than four times the amount of lost time at twice the dollar value to the smoking ritual.

Weis's estimates have not undergone the scientific scrutiny of the estimates provided by Kristein. Thus, if the estimates by Kristein can be considered to be a reasonable lower bound for costs to employers, those by Weis are probably an extreme upper bound.

A separate calculation by Kristein[32] converted the synergistic relationship among risk factors into costs to employers. Hypertension, hyperlipidemia, and cigarette smoking are independent risk factors for cardiovascular disease. Employees with any one of these risk factors will have higher medical care costs on average. An employee with more than one elevated risk factor is at much greater risk for disease; consequently, the average increase in medical costs is greater than the sum of the costs of individual risk factors. Kristein showed that smoking as an isolated risk factor is costly; smoking by employees with other risk factors increases potential medical costs.

For the individual. It is possible to estimate the costs of smoking over the lifetime of an individual. This prospective, incidence-based approach is singularly suited for estimating the impact of primary prevention efforts.[33] An analytical study by Oster and colleagues at Policy Analysis, Inc[34,35] represents the most sophisticated application of this methodology to the costs of cigarette smoking. Oster et al provide a synopsis of the methodology:

> Our estimates of the costs of smoking are expected values in a statistical or mathematical sense. They represent the likely average outcome of a game of health roulette that every smoker either knowingly or unknowingly plays. These estimates represent an amount that every smoker should expect to lose. Of course, a smoker who is lucky enough not to develop a serious illness over his or her lifetime will not bear significant costs as a result of smoking. Alternatively, a smoker who does eventually develop a serious disease will incur costs far in excess of those that are expected on average.[34]

These investigators examined the major diseases related to smoking: coronary heart disease, lung cancer, and chronic obstructive pulmonary disease. Smokers were considered to be engaging in a series of gambles based on their higher risks of developing any one of these diseases in any given year compared to nonsmokers. Medical costs for treatment of these diseases and the lost income costs from death or long-term disability were estimated. From this set of calculations, Oster et al estimated the expected lifetime costs of smoking, by amount smoked, in present-valued dollars to the individual for persons of different ages.

As an illustration, for male smokers aged 40 to 44, the costs of smoking ranged from $20,000 for light smokers to $57,000 for heavy smokers (1980 dollars). For females, the range was from $5,000 to $19,000. These costs include both direct health care costs and indirect future lost income costs, and they represent the average cost for each smoker.

This method is suited to personalizing the economic risks of smoking both to the individual smoker and to the employer who ultimately pays the majority of the direct health care costs.

COMMENTS ON COI METHODOLOGY

Focusing on economic costs in no way devalues the human costs of physical suffering, disabling illness, and loss of life from smoking-related disease. These costs are individually and collectively compelling, but they are difficult to value monetarily.

All methods for calculating costs of illness and attributing a portion of the costs to smoking have definable strengths and weaknesses. A critique of the prevalence-based methods has been presented. Estimates of costs to employers are primarily exercises in reasoning; they do not represent quantitative analysis. The sets of assumptions contained in these estimates rely on consensus-building until controlled studies of smoker-nonsmoker differentials in medical utilization costs and productivity are performed.

The incidence-based studies of Oster et al represent the method of choice for evaluating the potential cost impact of interventions on smoking behavior. These calculations are epidemiologically based and account for disease pattern variation by age and sex. They are limited by the dearth of disease incidence data for all but the most prominent causes of death. Thus, there is a trade-off: incidence-based methodology provides a comprehensive description of a limited set of smoking-related diseases. A further limitation for direct cost calculations is the ability to define the treatment protocol for a typical case.

Calculations by Richter and Gori[36] pose a challenge to health professionals committed to smoking control. Using econometric models, those authors found that successful smoking control and successful health promotion will produce positive economic outcomes in the short run. But ultimately, if no policy changes are made in the age of retirement and in the mechanisms for the financial support of the retired population, the effect of smoking control—and the larger arena of health promotion—could be recessionary. Under this theory, Social Security and pension plans as presently constituted could not easily support an influx of older retired adults with an extended life expectancy.

FUTURE DIRECTIONS

Future smoking-attributable COI calculations can benefit from several improvements in methodology:

- Future costs of smoking studies should be compatible with established guidelines for COI estimates[7] when estimating total disease costs.
- Additional guidelines should be established for the use of epidemiologic data when estimating the subset of costs for behavioral risk factors such as cigarette smoking.
- Calculations based on mortality comparisons should be validated or upgraded by calculations based on morbidity comparisons. This should take the form of direct medical care utilization data categorized by smoking status.
- Because patterns of medical utilization, lifestyle risk behaviors, and disease incidence and mortality patterns differ by gender, wherever possible smoking-attributable cost calculations should be performed separately by gender.
- Similarly, cost calculations should be performed separately by age category, particularly when the prevalence of the lifestyle risk behavior or the relative risk varies with age. These two parameters—prevalence and relative risk—are entered into the attributable risk calculations. For example, intricacies and accuracy of description of the smoking-CHD relationship are lost by pooling all ages.
- COI guidelines should be expanded to the societal level to account for all major economic effects. Direct cost estimates should be accompanied by both indirect mortality and indirect morbidity cost estimates. Ideally, one set of indirect mortality calculations should include, for each age and gender category, the present value of future earnings adjusted for non-market components (such as imputed value of household earnings), average per capita pension disbursements (non-prepaid pension plans only), and average per capita Social Security payments (with all values appropriately discounted).
- Prospective epidemiologic-economic models should be developed that predict the impact of smoking cessation, primary prevention of smoking, extended longevity, and other expected outcomes of smoking control measures using different scenarios.
- Collaboration among the disciplines of health economics, epidemiology, and health behavior research should be encouraged to promote increasingly sophisticated economic analyses of behavioral health problems such as cigarette smoking. An example of such collaboration is a workshop on the costs of smoking, which was convened in April 1985 by the Office of Technology Assessment, US Congress.
- Smoking-attributable COI calculations should be packaged into spreadsheet formats suitable for updating as new data become available. These spreadsheets should be well documented for use by state health departments and other agencies needing to calculate the costs of smoking. This would reduce duplicate labor and tap the expertise of different disciplines.
- Networking of national and state-level health professionals is recommended as a means of continually updating smoking cost estimates.

CONCLUSION

The enormity of the costs of cigarette smoking to society, to business, and to the individual has been estimated. Economic estimates will be crucial in future health care programs, and in corporate and societal decisions with regard to smoking control policies. Current best estimates place the annual costs of smoking at $39 billion to $55 billion to the United States and $400 to $800 in excess costs to employers per smoking employee (1983 dollars).

Methodology for COI calculations is undergoing constant refinement. The compounding of error in economic calculations with error in epidemiologic estimates of attributable risk poses a significant threat to the precision of smoking cost estimates.

See also Warner KE, NY State J Med *1983;83:1273–1274.*

REFERENCES

1. Hodgson TA, Meiners MR: Cost-of-illness methodology: a guide to current practices and procedures. *Milbank Memorial Fund Quarterly/Health and Society* 1982; 60:429–462.
2. Hodgson TA: The state of the art of cost-of-illness estimates. *Advances in Health Economics and Health Services Research* 1983; 4:129–164.
3. Rice DP: *Estimating the Cost of Illness.* Health Economics, series no 6. Public Health Service, publication no. 947-6. Washington, DC, US Government Printing Office, 1966.
4. Cooper BS, Rice DP: The economic cost of illness revisited. *Social Security Bulletin* 1976; 39:21–36.
5. Hodgson TA, Kopstein AN: Health care expenditures for major diseases in 1980. *Health Care Financing Review* 1985; 5:1–12.
6. Rice DP, Hodgson TA: *Social and Economic Implications of Cancer in the United States, Vital and Health Statistics,* series 3, no. 20, DHHS publication no. (PHS) 81-1404. Washington, DC, US Public Health Service, 1981.
7. Hodgson TA, Meiners MR: *Guidelines for Cost of Illness Studies in the Public Health Service.* Bethesda, Public Health Task Force on Cost of Illness Studies, 1979.
8. Luce BR, Schweitzer SO: The economic costs of smoking-induced illness. *Research on Smoking Behavior,* NIDA Research Monograph series no. 17, Washington, DC, US Department of Health, Education, and Welfare, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, DHEW publication no. (ADM) 79-581, 1977.
9. Soper FA: The economics of smoking. *Rehabilitation* (Bonn) 1972; 25:48–50.
10. Williams JR, Justus CG: Evaluation of nationwide health costs of air pollution and cigarette smoking. *J Air Pollution Control Assoc* 1974; 24:1063–1066.
11. Walker WJ: Government subsidized death and disability. *JAMA* 1974; 230:1529.
12. Boden LI: The economic impact of environmental disease on health care delivery. *J Occupational Med* 1976; 18:467–472.
13. Luce BR, Schweitzer SO: Smoking and alcohol abuse: a comparison of their economic consequences. *N Engl J Med* 1978; 298:569–571.
14. Kristein MM: Economic issues in prevention. *Prev Med* 1977; 6:252–264.
15. Kristein MM, Grove DA: Who pays health costs of alcohol and tobacco? (letter) *N Engl J Med* 1978; 299:606–607.
16. Rice DP, Hodgson TA: Economic costs of smoking: an analysis of data for the United States. Presented at Allied Social Science Association annual meetings, San Francisco, CA, 1983.
17. Koop CE: Statement to the committee. Presented to the Health and Human Services Committee, Minnesota House of Representatives, Minneapolis, March 14, 1985.
18. Forbes WF, Thompson ME: Estimating the health care costs of smokers. *Can J Pub Health* 1983; 74:183–190.
19. Collingshaw NE, Myers G: Dollar estimates of the consequences of tobacco use in Canada, 1979. *Can J Pub Health* 1984; 75:192–199.
20. Leu RE, Schaub T: Does smoking increase medical care expenditure? *Social Science in Medicine* 1983; 17:1907–1914.
21. Dean AG, Shultz JM, Kottke TE, Gust SW, Harty KC: The Minnesota plan for nonsmoking and health: ideas for statewide action. *Minnesota Med* 1985; 68:371.
22. Cady B: Cost of smoking. (letter) *N Engl J Med* 1983; 308:1105.
23. Paulozzi L: The costs of smoking for Washington. *Wash Morbid Report* 1984; 1:1–5.
24. Minnesota Department of Health: *The Minnesota Plan for Nonsmoking and Health: Report and Recommendations of the Technical Advisory Committee on Nonsmoking and Health.* Minnesota Dept of Health, 1984.
25. Thompson ME, Forbes WF: The methodology of estimating economic benefits and losses associated with cigarette smoking. *Mathematical Scientist* 1983; 8:53–61.
26. Vogt TM, Schweitzer SO: Medical costs of cigarette smoking. Final report for grant no. 5 RO1 HS 03601. Washington, DC National Center for Health Services Research, Dept of Health and Human Services, 1982.
27. Vogt TM: Effects of parental smoking on medical care utilization by children. *Am J Pub Health* 1984; 74:30–34.
28. Landefeld JS, Seskin EP: The economic value of life: linking theory to practice. *Am J Pub Health* 1982; 72:555–566.
29. Kristein MM: How much can business expect to profit from smoking cessation? *Prev Med* 1983; 12:358–381.
30. *Smoking and Health: A Report of the Surgeon General.* United States Department of Health, Education, and Welfare, Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health, publication no (PHS) 79-50066, 1979.
31. Weis WL: Can you afford to hire smokers? *Personnel Administrator* 1981; May:71–78.
32. Kristein MM: "How much is chronic disease costing the typical American company?" mimeographed. New York, American Health Foundation, 1980.
33. Hartunian NS, Smart CN, Thompson MS: The incidence and economic costs of cancer, motor vehicle injuries, coronary heart disease, and stroke: a comparative analysis. *Am J Pub Health* 1980; 70:1249–1260.
34. Oster G, Colditz GA, Kelly NL: *The Economic Costs of Smoking and Benefits of Quitting.* Lexington, MA, Lexington Books, 1984.
35. Oster G, Colditz GA, Kelly NL: The economic costs of smoking and benefits of quitting for individual smokers. *Prev Med* 1984; 13:377–389.
36. Richter BJ, Gori GB: Demographic and economic effects of the prevention of early mortality associated with tobacco-related diseases, in *Banbury Report: A Safe Cigarette?* Cold Spring Harbor Laboratory, 1980.

SAVING OR SMOKING

Tobacco companies have reason to be concerned about the growing involvement of the medical profession in efforts to eliminate cigarette smoking. A single physician who is successful in convincing even one person a day to stop smoking or not to start is responsible for approximately $125,000 per year in lost sales to the tobacco industry.

Money can be a major motivating factor in smoking cessation and prevention (which may also explain why the most expensive cessation programs claim the highest success rates). Few persons are aware of how much a cigarette habit costs. Paul Leichty of Jamestown, North Dakota, has calculated the amount a person can save by depositing in a 10% daily savings account the money that would have gone to pay for cigarettes. This table can be a useful reference in educating patients and the general public. One Tennessee family physician uses a personal computer to calculate the precise amount one can save, based on the cost of cigarettes, packs bought per day, the interest rate one is receiving on savings, and the frequency of compounding of interest. The print-out is given to the patient.

Dollars Saved Per Two Pack Year When Invested at a 10% Interest Rate*

Year	Balance	Year-to-Date Interest
1	922.20	46.20
2	1,941.69	143.49
3	3,068.72	251.03
4	4,314.64	369.92
5	5,691.99	501.35
6	7,214.63	646.64
7	8,897.89	807.26
8	10,758.72	984.83
9	12,815.84	1,181.12
10	15,089.96	1,398.12
11	17,603.98	1,638.02
12	20,383.20	1,903.22
13	23,455.59	2,196.39
14	26,852.08	2,520.49
15	30,606.86	2,878.78
16	34,757.73	3,274.87
17	39,346.47	3,712.74
18	44,419.26	4,196.79
19	50,027.17	4,731.91
20	56,226.65	5,323.48
25	98,527.20	9,359.90
30	168,369.20	16,024.38
35	283,684.59	27,028.04
40	474,080.60	45,196.06

* Initial investment of $2.40.

An insurance company perspective on smoking

MICHAEL J. COWELL

In April of 1964, within three months of the publication of the first report of the Surgeon General on *Smoking and Health,* State Mutual Life Assurance Company took what was then considered a bold step in the insurance industry. The company pioneered the concept of using a person's smoking habits as an insurance risk factor and introduced a special life insurance policy at discounted rates for those who did not smoke cigarettes.

State Mutual based its action on the belief that persons who choose not to smoke should not be made to subsidize insurance costs resulting from cigarette smokers' extra death claims. As a mutual company owned by policyholders, it had a tradition stretching back to the early 19th century of providing life insurance at cost.

State Mutual did not take this action out of a belief that smoking was immoral. Rather, the statistical evidence of much higher death rates among persons who smoke was so overwhelming that the company could no longer ignore it in pricing insurance.

This article discusses the key implications of incorporating the smoking risk in insurance underwriting; the principal findings from State Mutual's experience and that of other companies; and the changes that the company's efforts have brought about in the awareness of the smoking risk among insurance consumers.

1964: "IT CAN'T BE DONE"

In hindsight this move may not seem that bold. However, viewed in light of smoking habits in 1963, just before the Surgeon General's first report was published, it was a radical step. At that time, more than half the adult men and over 30% of adult women in the US smoked cigarettes. State Mutual's market 20 years ago was still predominantly male—like many traditional institutions, the company was only beginning to cultivate the newly emerging female insurance market. The concept of a premium discount for what was a minority of the market was viewed by many in the business as a risky venture at best, and reactions ran the gamut from healthy skepticism to outright ridicule. As recently as the year prior, the Society of Actuaries had sponsored a panel discussion on using smoking as a risk factor, the general consensus being that this was totally impracticable in underwriting life insurance.

Fortunately for State Mutual, in spite of all the experts who said it could not be done—or that it could not be done cost effectively—the company's new policy was well-received in the marketplace. Within a year non-smoker policies accounted for 30% of State Mutual's new individual life insurance sales. Soon other companies began to recognize that smoking was a significant factor in life and health insurance claims. With increased public attention in the late 1960s and early 1970s on the health effects of smoking, State Mutual's action was coming to be viewed as more than just a change in underwriting and pricing. What the company had pioneered was much more than a reclassification of risk. It was, in fact, the beginning of a change in the way that insurance companies would come to view smoking. Also, the offer was a financial incentive for those who had given up smoking, and an opportunity for those who had never started, to benefit from their favorable health habits.

By the mid 1970s, State Mutual had extended non-smoker pricing to virtually all its individual life and disability business and was beginning to develop statistically credible claims data. But other factors were also emerging. For one thing the nation's smoking habits were undergoing rapid change. Throughout the late 1960s and continuing into the 1970s, smoking among men declined by about one percentage point a year, falling below 40% by 1975 and below 33% by 1980 (*Statistical Abstract of the US 1984)*. Among women, following a slight increase in the late 1960s, the incidence of smoking began to decline, and by 1975 had fallen below 30%. Data from the National Center for Health Statistics suggest that the incidence of cigarette smoking in 1984 was only slightly above 30% for men and below 30% for women.

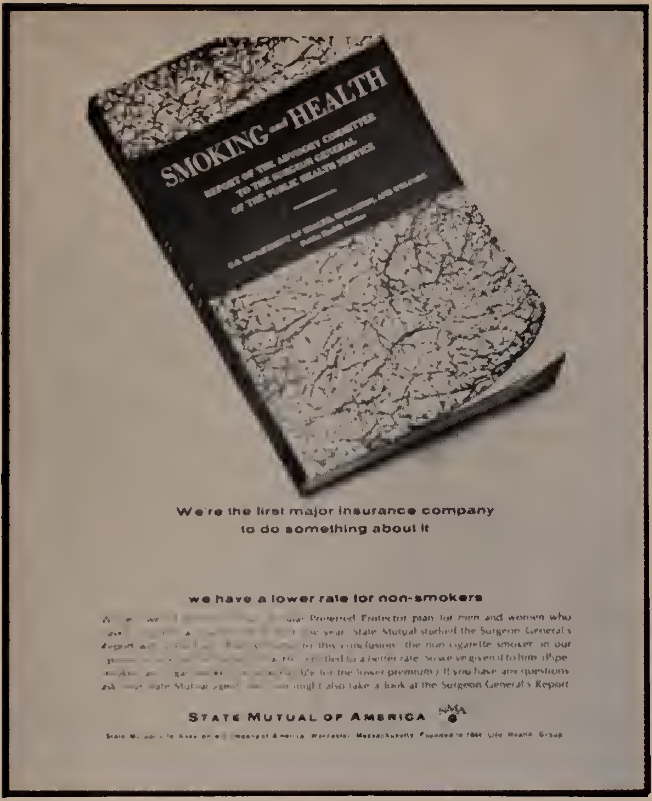
Another trend was the increasing number of major insurance companies offering non-smoker discounts—from a handful in the late 1960s to several dozen by the late 1970s. In addition to solid statistical data on mortality differences between smokers and non-smokers, companies were accumulating valuable experience in how to underwrite the smoking risk.

One of the most common questions about underwriting practices is the extent to which people misrepresent their smoking habits. No doubt a small percentage of clients are less than honest with themselves—and with the company. But, if anything, with the change in smoking habits over the past 20 years, there are far fewer casual smokers—those smoking less than 10 cigarettes a day. And since men had traditionally smoked much more than women, it was they who led in quitting. Men make up the majority of the 33 million former smokers, who comprise 21% of the adult population. Another 75 million, or 46% of the population, claim to have never smoked, leaving 52 million, or about 32%, as current smokers. On the basis of the 600 billion cigarettes consumed in the US in 1984, it is estimated that these 52 million people smoked, on the average, about a pack-and-a-half a day. At that level of consumption it is difficult—though not impossible—to escape detection from an experienced underwriter.

STATE MUTUAL REPORT OF 1979

With that background in mind, it is worthwhile to con-

Address correspondence to Mr Cowell, vice president and actuary, State Mutual Life Assurance Company of America, 440 Lincoln Street, Worcester, MA 01605.


SMOKING AND HEALTH
REPORT OF THE ADVISORY COMMITTEE TO THE SURGEON GENERAL OF THE PUBLIC HEALTH SERVICE

We're the first major insurance company
to do something about it

we have a lower rate for non-smokers

STATE MUTUAL OF AMERICA

Original advertisement that State Mutual ran in 1964 when the nonsmoker discount was introduced.

sider the key findings of State Mutual's 1979 study that became the basis for its report, "Mortality differences between smokers and non-smokers."* First, the company analyzed the death claims between 1973 and 1978 of individuals who had purchased policies issued between 1964 and 1968 on a so-called "regular" basis, meaning they didn't qualify as nonsmokers. State Mutual compared these to the deaths of those who were similar in all other respects except that they qualified for the nonsmoker policy. The analysis showed the following:

- Cigarette smokers were subject to mortality significantly higher than nonsmokers—specifically, two to two-and-a-half times, in the aggregate.
- Compared to nonsmokers, death rates for smokers were 15 times as high for respiratory cancer; almost four times as high for other respiratory diseases; and three times as high for heart disease.
- These differences emerged at early durations. Contrary to what the company had believed, the extra death claims were not deferred to older ages.
- The differences were statistically significant at a level above 99.99%.
- They were far too large to be ignored for insurance underwriting and pricing purposes.

After the company published its report a number of other companies also presented their statistics. The only surprise was how closely their results paralleled State Mutual's. It came as quite a surprise to some of them that the mortality differences between the smokers and nonsmokers were as large as they were. Ironically, two other companies had taken a look at the statistics, but were reluctant to publish them. Until State Mutual issued its report, they simply could not believe the magnitude of the differences they were coming up with. Even now, more than 20 years after State Mutual made the distinction, executives of other companies who have only recently separated out nonsmokers express amazement at the extra mortality among the smokers.

One of the most talked about statistics emerging from the study was the difference in life expectancy between smokers and nonsmokers. State Mutual had for some time taken the position that aggregate life expectancy figures, based on a composite population of smokers and nonsmokers, were meaningless, because they did not reflect the experience of any homogeneous group. On the basis of population data and government studies in the US, Canada, Britain, Sweden, and Japan, the Surgeon General concluded that a nonsmoking man age 32 could expect to live eight to nine years longer than his smoking counterpart. State Mutual calculated that a healthy male nonsmoker age 32 just underwritten for life insurance would outlive an average smoker the same age by almost seven years.

Data on women were far more limited. On the basis of a much smaller sample size, similar mortality patterns were found between smoking and nonsmoking women, although the absolute numbers and the differences in longevity were not as significant as for men. Some of this has to do with the differences in smoking habits between the sexes. The typical woman smoker consumes fewer cigarettes than her male counterpart. This is not to say that smoking is not a problem among women; recent studies by the US Surgeon General have indicated that women smokers are, indeed, subject to the same risks as men. It is simply that smoking among women was not perceived as being as serious until they began to take up the habit in record numbers in the decades following World War II. State Mutual was also interested to learn from the Surgeon General's reports that smoking among women is slightly more prevalent at middle and upper income levels. In the managerial and professional ranks, female smoking patterns come closer to matching those of males. For example, a 1975 government survey found almost 40% of nurses smoking—one of the highest percentages among a group of predominantly female professionals. Among men, on the other hand, the incidence of cigarette smoking falls off at higher income and educational levels.

In their 1979 study of mortality differences between smokers and nonsmokers, the State Mutual authors acknowledged that they had only scratched the surface of what they believed would be a vast new area for investigation. Their data base is derived from the experience of healthy individuals selected for life insurance, a group whose life expectancies would be generally more favorable than those of the overall population. While their study should thus be interpreted with this caution in mind, they do demonstrate remarkable parallels between the excess mortality of smokers in the insurance population to that found in the general population.

REWARDING HEALTHY HABITS

These patterns of cigarette usage by occupational class

---

* Cowell MJ, Hirst BL: Mortality differences between smokers and nonsmokers. *Transactions of the Society of Actuaries* 1980; 32:185-261.

have considerable significance for insurance companies like State Mutual that target their markets to specific socioeconomic segments of the insurance buying public. One insurance company has gone so far as to announce that it is no longer actively soliciting life insurance among smokers. State Mutual is not convinced of the business wisdom of such a move. In 1961, State Mutual introduced discounts for nonsmokers as a means of pricing insurance more fairly. It was not the company's intention to encourage an underwriting system that excluded a significant segment of the market. Given their higher risk of sickness and death, it would seem that smokers have an even greater need for insurance than nonsmokers. State Mutual simply wanted to make sure that smokers paid for the higher insurance risk related to their habit.

The company foresees several additional insurance classifications that reward favorable health habits, without denying coverage to those who need it the most. State Mutual has extended nonsmoker discounts to individual disability insurance and to small groups of persons underwritten individually. It has also incorporated smoking habits as a risk factor in a new group health insurance program called "Wellcare." This novel approach to containing health care costs and helping people stay healthy encourages employers to pass along to employees the lower health costs that result from not smoking and from other favorable habits.

By following these examples, the insurance industry is accomplishing several objectives. First, it is providing financial incentives to those who maintain good health habits. Second, the types of risk classification on which these incentives are based put primary emphasis for health under the personal control of the individual. A third objective—one that most insurance managers find appealing—is that segmenting the market along smoking lines has turned out to be a profitable business decision in terms of the quality and quantity of insurance that is sold to nonsmokers (Table).

Current Mortality Charges Per $1,000 of Insurance at Four Ages for State Mutual's Popular Universal Life Insurance Policy

	Male		Female	
Age	Non-Smoker	Smoker	Non-Smoker	Smoker
25	$1.22	$ 1.71	$0.93	$1.23
35	$1.35	$ 2.11	$1.21	$1.84
45	$2.66	$ 5.03	$2.40	$3.93
55	$5.49	$11.81	$4.61	$7.08

Lastly, there may well be a role for the insurance industry that goes beyond the traditional function of passively observing trends in sickness and death and putting a price on them in the form of life and health insurance premiums.

Emphasis on preventive medicine and greater acceptance of personal responsibility for one's health—staying healthy, as contrasted with seeking treatment for illness—will present insurance companies with a new challenge.

INSURANCE INCENTIVES FOR NOT SMOKING

In December 1984, the National Association of Insurance Commissioners (NAIC) recommended that insurance companies voluntarily offer a 25% to 50% insurance discount on health insurance for nonsmokers who keep their weight and blood pressure down.

The proposal represents a shift in the attitudes of the insurance industry and the belief that health care costs will not decrease until people take responsibility for their own health. Currently, many companies offer lower rates to nonsmokers for life insurance. But a great majority of health insurance companies have not lowered their rates for nonsmokers despite the known ill health effects to those who do smoke. Moreover, according to William P. Daves Jr, a member of the Texas State Board of Insurance and chairman of the NAIC Task Force on Health Promotion, "group health insurance is the only form of insurance left that doesn't calculate its rates according to risk factors."

The NAIC proposal is being tested by the Blue Cross and Blue Shield Company of Southwestern Virginia; for community rated groups, a 3% discount is offered if 80% of the subscribers are nonsmokers and a 7% discount is offered if all the subscribers do not smoke.

To promote the idea of adjusted rates for nonsmokers, Action on Smoking and Health (ASH) Executive Director John Banzhaf appeared at a press conference sponsored by the NAIC. He presented evidence that shows that smoking greatly increases health care costs by, for example, increasing hospital stays and worsening less serious health conditions. A George Washington University task force testifying with Banzhaf cited figures that showed how smoking increases the nation's annual health care costs by a minimum of $11 billion.

Banzhaf said that the current policy was not only unfair but possibly illegal. He said the differential rates would end the mandatory subsidy of nonsmokers and provide smokers with an incentive to stop. Banzhaf also offered the NAIC three criteria concerning risk factors: the risk factor must cause a substantial increase in health care costs; it must be subject to easy and objective verification; it must be subject to change by the insured and, if changed, result in lower health care costs.

The NAIC consists of commissioners from all 50 states who regulate insurance rates and the insurance industry. Because their approval is required for most rate increases, and they are influential in proposing insurance legislation, the NAIC is capable of encouraging the adoption of the proposal by the health insurance companies.

In Congress, Senator David Durenberger (R-Minnesota) has put forward a bill entitled Medicare Cost Incentives for Non-Smokers (S-357), which calls for lower Medicare premiums for elderly persons who do not smoke than for those who do.

MICHAEL WROBLEWSKI

(Sources: *ASH Smoking and Health Review, Family Practice News, The New York Times*)

TOXICOLOGY

Changes in overwrap and butt length of American filter cigarettes

An influence on reported tar yields

NEIL E. GRUNBERG, PHD, DAVID E. MORSE, PHD, VIRGINIA A. MAYCOCK,
LYNN T. KOZLOWSKI, PHD

ABSTRACT. Reported tar and nicotine yields of cigarettes, determined by official government assays, are used by the public and by health professionals to evaluate and compare cigarette brands. Many smokers use these reported values to help them decide what brands to smoke. In addition, health-care professionals often encourage smokers to switch to presumably less hazardous low tar brands. Health researchers also use reported yields to examine changes in smoking behavior with changes in cigarette brands and health hazards associated with smoking certain brands. Unfortunately, some of these reported values are misleading.

The present study examined the relationship between reported tar yields and changes in physical characteristics (overwrap length and butt length) of filter cigarettes. Of the 161 American filter cigarette brands examined, 18% showed an inverse relationship between changes in overwrap length and reported tar yield. When this relationship exists, reported yields may be misleading and inaccurate. To improve the meaningfulness of the reported tar and nicotine yields, the number of puffs taken by smoking machines on each cigarette should be reported. (NY State J Med 1985; 85:310–312)

INTRODUCTION

Since 1967 the public has used reported tar and nicotine yields of cigarettes to make decisions about what cigarettes to smoke. Unfortunately, some of this information is quite misleading. For example, official assays in the United States and Canada show that tar deliveries of cigarettes have declined over the years.[1,2] However, the value of these reported figures has been criticized on the basis that smokers can compensate for reduced tar and nicotine content of cigarettes by taking more puffs or by blocking ventilation holes.[3–9] Smokers may thereby self-administer substantially more tar and nicotine from each cigarette than official tar and nicotine reports indicate.

Smokers and health-care professionals can only compare cigarette brands on the basis of reported tar and nicotine yields. No other information is available.

Reported tar and nicotine yields are determined from smoking machines (Fig 1) that smoke each cigarette to a prescribed butt length established in 1967 by the Federal Trade Commission (FTC). This butt length criterion (23 mm or filter plus overwrap plus 3 mm, whichever is greater) has been debated since cigarette testing began because, following this criterion, cigarettes will be smoked to different butt lengths—depending on their filter and overwrap lengths—by the smoking machines and therefore will give incomparable tar and nicotine yields. In fact, two of the five commissioners of the FTC who established this criterion disagreed with it from the beginning, arguing that because different cigarettes are smoked by the smoking machines to different butt lengths, "the comparative value of the tests is doubtful."[10]

It is clear that these tests, as they now are performed, have little comparative value over time, because butt lengths have increased markedly over the years. Figure 2 illustrates the dramatic increase in the percent of cigarettes smoked by smoking machines to a butt length in excess of 30 mm from 1967 to 1979 (correlation coefficient, $r = 0.975$). Increases in butt length such as these may lead to decreases in the number of puffs taken by smoking machines which, in turn, would lead to decreases in reported tar and nicotine yields. In contrast, people can change their rate and number of puffs to compensate for increased butt lengths. To improve the smoking machine analyses and the value of tar and nicotine reports, it is essential to report the number of puffs taken by smoking machines on each cigarette brand.

Previous research has found that the number of puffs taken by smoking machines on popular brands of Canadian cigarettes decreased from 1969 to 1974.[7] This decline in number of puffs appears to be a major cause of reported reductions in tar yields for those cigarettes. That study also included two American cigarette brands. As with the Canadian cigarettes, a decline in the number of puffs over the years appeared to be a major cause of the reported reductions in tar yields for these two brands. It is impossible to know from a study of only two cigarette brands whether changes in number of puffs is an important factor affecting reported yields for US cigarette brands. A more complete analysis is necessary to reach conclusions about the importance of reporting the number of puffs on US cigarette brands. Unfortunately, the US Federal Trade Commission

---

From the Department of Medical Psychology (Drs Grunberg and Morse and Ms Maycock), Uniformed Services University of the Health Sciences, Bethesda, and the Addiction Research Foundation (Dr Kozlowski), Toronto.

Address correspondence to Dr Grunberg, Associate Professor, Department of Medical Psychology, Uniformed Services University of the Health Sciences, 4301 Jones Bridge Rd, Bethesda, MD 20814.

The opinions or assertions contained herein are the private ones of the authors and are not to be construed as official or reflecting the views of the DOD or the USUHS. Portions of this paper were presented at the annual meeting of the American Psychological Association in Los Angeles, California, August, 1981.

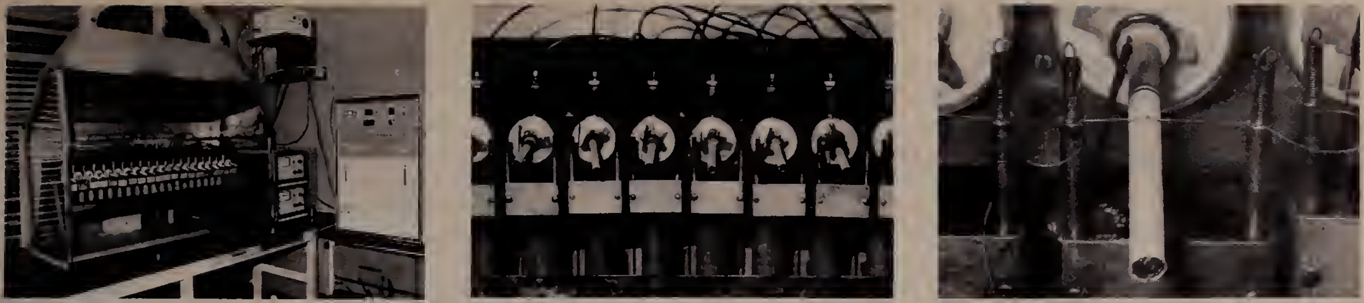


FIGURE 1. Three views of the smoking machine used by the Federal Trade Commission; the machine smokes cigarettes up to the string. (Photographs by Greg Holmes, Uniformed Services University Audio Visual Center).

Laboratory has not maintained records of the number of puffs taken per cigarette in their tests (Dr Harold Pillsbury, Federal Trade Commission, personal communication). Therefore, we were unable to examine directly the relationship between number of puffs and reported yields of US cigarette brands.

There is, however, an indirect way to determine whether the US smoking machines changed the number of puffs taken of filter cigarettes tested. Smoking machines take a 2-second 35-mL puff once each minute, until the remaining butt length equals the length of the filter and overwrap plus 3 mm. The overwrap is a piece of paper that connects the filter to the rest of the cigarette (Fig 3). If the length of the filter or the length of the overwrap increases, thereby increasing the butt length, the smoking machine will take fewer puffs, assuming that the burn rate does not change. Therefore, an increase in either filter or overwrap length can result in a decreased reported tar and nicotine yield by the smoking machine, because the machine would take fewer puffs.

It is important to note that the overwrap is a smokable portion of the cigarette; it is filled with tobacco, it burns, and cigarettes can be smoked through the overwrap right up to the filter. People are not governed by the FTC definition of how to smoke each cigarette. Therefore, increases in overwrap length that result in smoking machines smoking less of a cigarette do not necessarily decrease tar and nicotine delivery to human smokers. Whenever there are increases in overwrap length, the reported tar yield probably underrepresents what human smokers actually get from a given cigarette brand. Because puff data and burn rate information are not available for US cigarette brands, we had to rely on overwrap length as an indirect indication of number of puffs. Assuming that the burn rate did not change, butt length (which includes the overwrap) would determine the number of puffs. This study evaluated changes in overwrap length with respect to reported tar delivery for all US cigarette brands for which data were available.

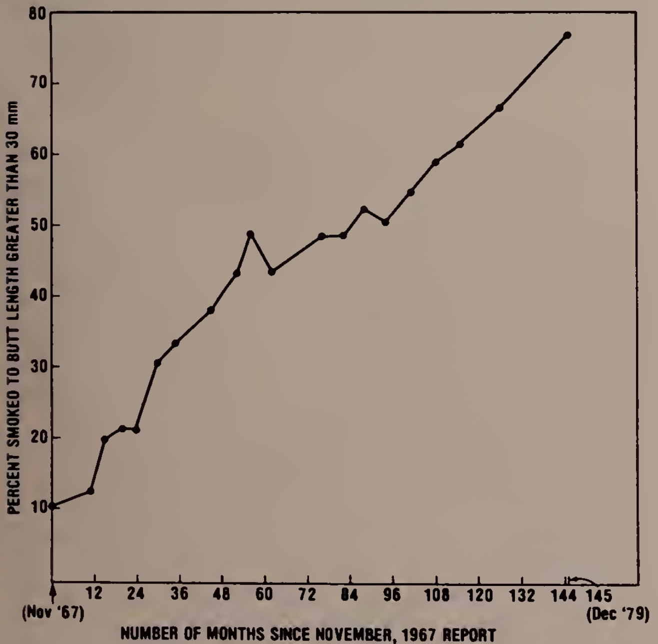


FIGURE 2. Percent of US cigarette brands smoked by smoking machines to a butt length greater than 30 mm (from 1967 to 1979 FTC reports).

METHODS

The physical dimensions—filter length, overwrap length, and cigarette length—of US cigarette brands tested by the FTC Laboratory from 1967 through 1979 were obtained from the laboratory's files. These data are not included in the FTC reports on cigarettes, and have never been evaluated. Because there has been no reason for the FTC to record this information, the data set is incomplete. The tar yield data come from the published reports of the Federal Trade Commission.

For all filter cigarettes for which there were more than three data points (physical dimensions recorded for more than three of the cigarette analysis periods), two raters examined graphs and tables of overwrap lengths and reported tar values and judged whether the overwrap length either did not change, increased as reported tar yield decreased, decreased as reported tar yield decreased, or changed but not in any regular fashion with changes in reported tar yields. Disagreements in judgments were settled by a third rater.

RESULTS

The physical dimensions of 346 cigarette brands were examined. There were too few data points to classify 149 of these brands. In addition, there were 36 nonfilter cigarette brands. Therefore, only 161 filter cigarette brands could be classified.

Of these 161 brands, the two raters agreed in classifying 158 (98.1%). Twenty-nine brands (18%) showed increases in overwrap corresponding to decreases in reported tar yield (Table). The two raters agreed in classifying all of these brands. One hundred and twenty-one brands showed no changes or irregular changes in overwrap corresponding to changes in reported tar yield, and 11 showed decreases in overwrap corresponding to decreases in reported tar yield. All of these latter 11 brands showed increases in filter lengths corresponding to decreases in reported tar yields. In addition, for five of these 11, the increases in filter lengths were greater than the decreases in overwrap length. Therefore, the butt lengths of these five cigarette brands increased, smoking machines would have puffed less, and the reported tar yields may be misleading.

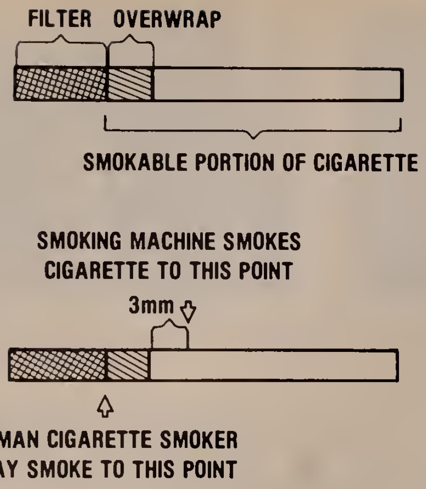

FILTER
OVERWRAP
SMOKABLE PORTION OF CIGARETTE
SMOKING MACHINE SMOKES CIGARETTE TO THIS POINT
3mm
HUMAN CIGARETTE SMOKER MAY SMOKE TO THIS POINT

FIGURE 3. Cross-sectional diagram of a filter cigarette.

DISCUSSION

These results indicate that changes in overwrap may have had an artifactual effect on reported tar yields for 18% of the US filter cigarette brands evaluated. These particular filter cigarettes accounted for at least 18%, and possibly as much as 30%, of the cigarettes sold in the United States between 1967 and 1979.[11]

Smokers who base their decision to smoke particular cigarettes on the reported tar yields alone are relying on incomplete, misleading information. In addition, cigarette smoking research that included these particular cigarette brands may have come to false conclusions—conclusions regarding the relationship between tar yields and morbidity may be inaccurate.

These findings further support the argument that the number of puffs taken on cigarettes by smoking machines should be reported.[7] The smoking machines in current use automatically count puffs, so that information could be published along with tar and nicotine deliveries, making these reports more meaningful. Individual smokers could use this information and compare it to their own smoking behavior to determine whether switching brands is actually beneficial. For instance, if low tar brand A reports 10 puffs per cigarette and brand B reports the same low tar content but 6 puffs, then the smoker who finds that he or she takes 8 puffs on both A and B probably should smoke brand A. Health professionals also could use puff data along with tar and nicotine reports to help direct smokers to cigarette brands that are less harmful. Further, smokers could be encouraged to take the same or fewer puffs as the smoking machines took on a given cigarette brand.

Although the percentage of the US adult population who smokes cigarettes has decreased, 54 million US citizens continue to smoke. Information that can help prevent and reduce the death and illness associated with smoking, such as accurate and meaningful tar yield data, is vitally important. Unfortunately, the very statistics that were meant to help people may instead misguide them.

The Federal Trade Commission did not wish to comment on the subject at this time. —*Editor*

Filter cigarette brands that showed increases in overwrap corresponding to reported decreases in tar content.

Brand	Company
Alpine Menthol Kingsize Filter Softpack	Philip Morris
Benson & Hedges Kingsize Filter Hardpack	Philip Morris
Chesterfield Kingsize Filter Softpack	Liggett & Myers
Chesterfield 101's Filter Softpack*	Liggett & Myers
Edgeworth Exports Kingsize Filter Hardpack†	Larus & Brother
Galaxy Kingsize Filter Softpack‡	Philip Morris
Kent Kingsize Filter Hardpack	Lorillard
Kent Kingsize Filter Softpack	Lorillard
Kent Menthol 100's Filter Softpack	Lorillard
Kool Menthol 100's Filter Softpack	Brown & Williamson
Lucky Strike 100's Filter Softpack†,‡	American
Marlboro 100's Filter Softpack†	Philip Morris
Marvels Menthol Kingsize Filter Softpack‡	Stephano Brothers
Newport Menthol Kingsize Filter Hardpack	Lorillard
Newport Menthol 100's Filter Softpack*	Lorillard
Old Gold Kingsize Filter Hardpack†	Lorillard
Old Gold Kingsize Filter Softpack	Lorillard
Pall Mall Kingsize Filter Softpack	American
Pall Mall 95's (100's) Filter Hard Wide Pack*	American
Pall Mall Menthol 100's Filter Softpack*	American
Raleigh Kingsize Filter Softpack	Brown & Williamson
St. Moritz Menthol 100's Filter‡	Turmac Tobacco
Salem 100's Filter Softpack*	RJ Reynolds
Tareyton 100's Filter Softpack*	American
Tempo Kingsize Filter Softpack*	RJ Reynolds
True 100's Filter Softpack	Lorillard
Viceroy Kingsize Filter Softpack	Brown & Williamson
Winston Kingsize Filter Hardpack	RJ Reynolds
Winston Kingsize Filter Softpack†	RJ Reynolds

* Noticeable increases in filter length accompany increases in overwrap length.
† Relationship between overwrap length and reported tar is weak.
‡ For these cigarettes there are decreases in filter length that may offset the increased overwrap length as far as the smoking machine is concerned.

Acknowledgment. This work was supported by the Uniformed Services University of the Health Sciences Protocol No. C07217. Dr Harold Pillsbury provided the overwrap length data. Andrew Baum and Nancy Grunberg assisted in the preparation of the manuscript.

REFERENCES

1. Report of "tar" and nicotine content of the smoke of 176 varieties of cigarettes. Washington, DC, Federal Trade Commission, 1979.
2. Tar, nicotine levels show some substantial reductions. (news release), Ottawa, Health and Welfare Canada, March 29, 1974.
3. Ashton H, Watson DW: Puffing frequency and nicotine intake in cigarette smokers. *Br Med J* 1970; 3:679–681.
4. Green SJ: Ranking cigarette brands in smoke delivery, in Thornton RE: *Smoking Behaviour: Physiological and Psychological Influences.* Edinburg, Churchill Livingstone, 1978, pp 380–388.
5. Kozlowski LT, Frecker RC, Khouw V, Pope MA: The misuse of 'less hazardous' cigarettes and its detection: hole-blocking of ventilated filters. *Am J Public Health* 1980; 70:1202–1203.
6. Kozlowski LT, Jarvik ME, Gritz ER: Nicotine regulation and cigarette smoking. *Clin Pharmacol Ther* 1975; 17:93–97.
7. Kozlowski LT, Rickert WS, Robinson JC, Grunberg NE: Have tar and nicotine yields of cigarettes changed? *Science* 1980; 209:1550–1551.
8. Sutton SR, Feyerabend C, Cole PV, Russell MAH: Adjustment of smokers to dilution of tobacco smoke by ventilated cigarette holders. *Clin Pharmacol Ther* 1978; 24:395–405.
9. Benowitz NL, Hall SM, Herning RI, Jacob P, III, Jones RT, Osman A: Smokers of low-yield cigarettes do not consume less nicotine. *N Engl J Med* 1983; 309:139–142.
10. Letter from FTC Secretary to Chairman, Committee on Interstate and Foreign Commerce, House of Representatives, Nov 22, 1967. In *Report of tar and nicotine content of the smoke of 127 varieties of cigarettes.* Washington, DC, Federal Trade Commission, 1968.
11. Market shares of cigarette brands. JC Maxwell, Jr (1967–1970 data) and Philip Morris Market Research Department (1971–1979 data).

The "less hazardous" cigarette: a deadly delusion

G. H. MILLER, PHD

The quest for a "safer" or "less hazardous" cigarette received its initial thrust after the 1964 report of the Surgeon General, which reviewed studies that showed the impact of cigarette smoking. Despite reports in 1968[1] and 1970[2] of the potential benefits of low tar and low nicotine cigarettes, an analysis of studies reported in the past 15 years appears to demonstrate the difficulty of attempting to produce a safer cigarette.

The major justification for the development of a less hazardous cigarette was the assumption that most individuals who smoke cannot stop. Proponents of this premise have contended that a less hazardous cigarette should be developed to reduce the incidence of cigarette-related diseases for smokers.

FILTERED FACTS

The components first considered in cigarettes were nicotine and tar (presently defined as the total particulate matter of the cigarette retained by a glass fiber filter, excluding water and nicotine). Early research was based on the assumption that reduction in tar and nicotine would lead to a less hazardous cigarette. This thesis was accepted by most health professionals and the public. Soon cigarette companies, which have never admitted that smoking causes any harm whatsoever, began advertising numerous brands that claimed to offer less tar and nicotine. Indeed, low tar claims have become one of the major themes of cigarette advertising, with each major manufacturer offering at least one low tar cigarette brand (Fig 1). No mention has recently been made by the cigarette companies of the other harmful components in cigarette smoke (But see Fig 2).

The first major studies to develop a "safer" cigarette were supported by the National Cancer Institute. The researchers reported findings that appeared to show lower mortality rates with lower tar and nicotine levels. Analyses of these studies, however, produced questionable results.[3] The use of small sample sizes resulted in data unsuitable for age-adjusted analysis. The studies compared individuals who smoked filter cigarettes for more than 10 years with individuals who were lifetime nonfilter cigarette smokers and with those who had smoked filter cigarettes for less than 10 years. This comparison introduces a bias, because those who smoked filter cigarettes only are almost exclusively from the lower age groups. Therefore, such differences in age cohorts would lead to a lower cancer mortality rate for filter cigarette smokers. Also, tables showing mortality rates for ex-smokers that were higher than those of smokers make such studies unreliable.

A series of studies investigated the potential effect of carbon monoxide, which forms a large portion of the gaseous components in cigarette smoke. In Denmark, reports based on animal studies[4,5] demonstrated the potentially damaging effects of carbon monoxide on the cardiovascular system.

Studies in the United States confirmed these effects on the cardiovascular system. Increased levels of carbon monoxide were found to reduce arterial flow,[6] increase the incidence of angina attacks,[7,8] and increase deaths from cardiovascular disease. In England it was reported that those who smoked the supposedly safer filter-tipped cigarettes had higher levels of carboxyhemoglobin in their blood compared with those who smoked unfiltered cigarettes.[9] These studies provide evidence that low tar-low nicotine filter cigarettes, which produce increased levels of carbon monoxide, can be more hazardous to the smoker's overall health.

USE OF "CRITICAL VALUES"

During the mid-1970s, a spirited controversy arose concerning the less hazardous cigarette following the publication of two articles.[10,11] The first, published in *Science*,[10] purported to estimate the safe levels of exposure to certain components in cigarette smoke. The analysis of the data was used to justify the continued production of the less hazardous cigarette. Certain "critical values" were defined as safe levels for these components or as "the maximum number of pre-1960 cigarettes that may be smoked daily without detectable increase of the average smoker's risk of mortality beyond that of the nonsmoker."

A month before the second article was published in *JAMA*,[11] the purported results were given widespread publicity, a situation decried by the journal's editor, William R. Barclay, MD, in an accompanying editorial.[12] Barclay also took pains to diminish the significance of the publication of the article, describing the finding as of "small benefit" to individuals who do not follow the medical profession's good advice not to smoke.

Nonetheless, reports in the mass media on the safety levels of certain low tar-low nicotine cigarettes included tables listing the number of cigarettes of specific brands that could be smoked "safely" (Fig 3). For example, 21 "low-tar" Merit cigarettes were supposedly equal to two regular "high-tar" cigarettes. These tables of the "safe" numbers of cigarettes that could be smoked were not contained in the original article. The source of these tables remains unclear.

The *JAMA* article[11] used "critical values" to estimate the safety levels of six compounds found in cigarette smoke: carbon monoxide, tar, nicotine, nitrogen oxides, hydrogen cyanide, and acrolein. However, the statements concerning the possible safety of the less hazardous ciga-

---

Address correspondence to Dr Miller, Director, Studies on Smoking, 125 High St, Edinboro, PA 16412.

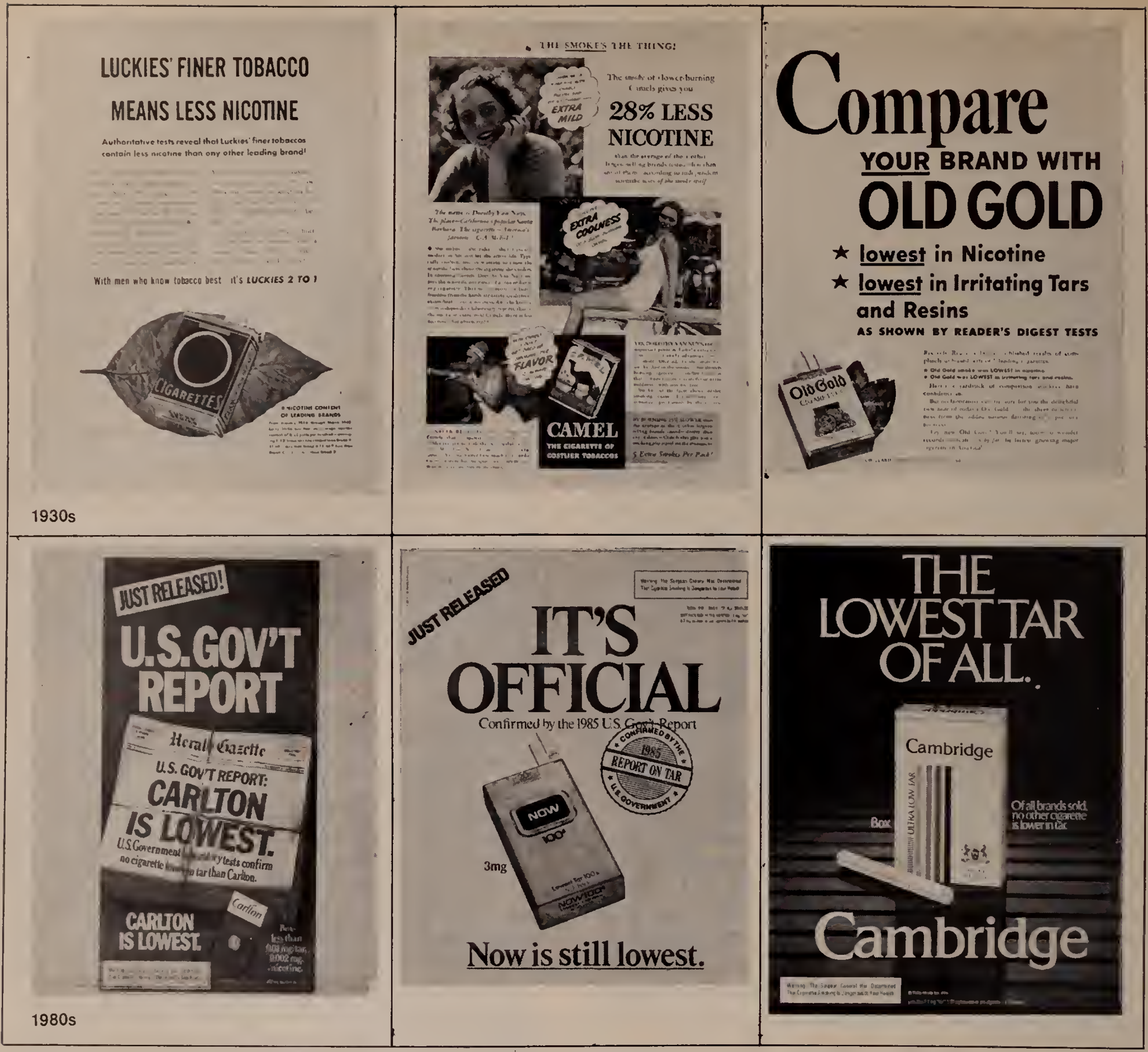

LUCKIES' FINER TOBACCO MEANS LESS NICOTINE
Authoritative tests reveal that Luckies' finer tobaccos contain less nicotine than any other leading brand!
With men who know tobacco best it's LUCKIES 2 TO 1
THE SMOKE'S THE THING!
EXTRA MILD
28% LESS NICOTINE
EXTRA COOLNESS
FLAVOR
CAMEL
THE CIGARETTE OF COSTLIER TOBACCOS
Compare
YOUR BRAND WITH
OLD GOLD
★ lowest in Nicotine
★ lowest in Irritating Tars and Resins
AS SHOWN BY READER'S DIGEST TESTS
1930s
JUST RELEASED!
U.S. GOV'T REPORT
Herald Gazette
U.S. GOV'T REPORT:
CARLTON IS LOWEST.
CARLTON IS LOWEST
JUST RELEASED
IT'S OFFICIAL
Confirmed by the 1985 U.S. Gov't Report
CONFIRMED BY THE 1985 REPORT ON TAR U.S. GOVERNMENT
NOW 100's
3mg
Now is still lowest.
THE LOWEST TAR OF ALL.
Cambridge
Of all brands sold, no other cigarette is lower in tar.
Cambridge
1980s

FIGURE 1. Plus ça change. For decades cigarette makers have vied to take out ingredients they have never acknowledged to be harmful. (Illustrations in this article courtesy DOC archive.)

rettes were dependent upon assumptions of theoretical safety limits if the estimates were correct and if the proposed reduction in these components could actually be made. These assumptions, as well as the handling of the data, were made without experimental substantiation.[13-15] Unfortunately, due to the amount of pre-publication publicity, the public was presented with information assumed to be based on fact, whereas it was based on speculation.

People vs Machines

Reports concerning the potential hazards of low tar-low nicotine cigarettes have appeared for the last decade.[16-18] Each has noted compensatory behavior, such as an increase in the number of cigarettes smoked and an adjustment of the smoking pattern to changes in length of the cigarette. Ventilated cigarettes (cigarettes with small holes near the end of the filter) were developed ostensibly to reduce the amount of carbon monoxide inhaled by the smoker. However, compensatory behavior appears to offset any theoretical benefits of the ventilated cigarette, since many smokers cover up the holes, in effect making the ventilated cigarette no different from any other. An excessive amount of carbon monoxide inhaled by smokers of ventilated cigarettes compared with smokers of unfiltered cigarettes has been documented.[19]

The effectiveness of the measurement of nicotine obtained by cigarette smoking machines has also been questioned. Benowitz et al[19] have shown that smoking machines do not allow for human factors such as deeper inhalation, increased puffs per minute, and other types of compensatory behavior with reference to low tar-low nicotine cigarettes. Whether the cigarette brand had a high or low nicotine rating—as established by smoking machine mea-

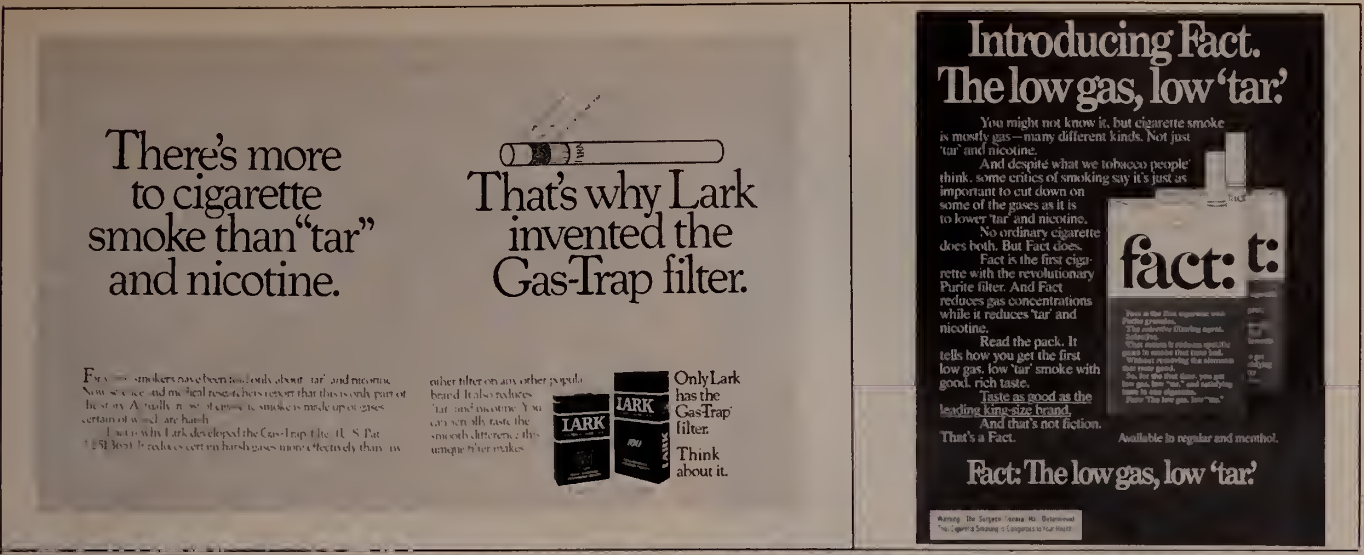
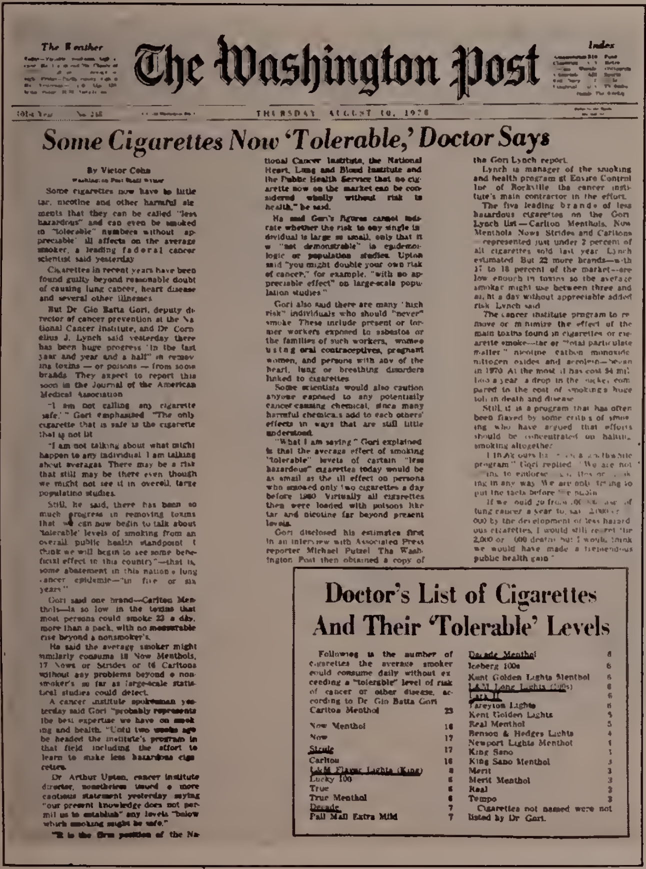

There's more to cigarette smoke than "tar" and nicotine.
That's why Lark invented the Gas-Trap filter.
LARK
Only Lark has the Gas-Trap filter.
Think about it.
Introducing Fact.
The low gas, low 'tar.'
You might not know it, but cigarette smoke is mostly gas—many different kinds. Not just 'tar' and nicotine.
And despite what we tobacco people think, some critics of smoking say it's just as important to cut down on some of the gases as it is to lower 'tar' and nicotine.
No ordinary cigarette does both. But Fact does.
Fact is the first cigarette with the revolutionary Purite filter. And Fact reduces gas concentrations while it reduces 'tar' and nicotine.
Read the pack. It tells how you get the first low gas, low 'tar' smoke with good, rich taste.
Taste as good as the leading king-size brand.
And that's not fiction. That's a Fact.
fact:
Available in regular and menthol.
Fact: The low gas, low 'tar.'

FIGURE 2. At least two brands have alluded to components of smoke other than tar and nicotine. LARK in the 1960s and fact in the late 1970s promised less "gas." Carbon monoxide and cyanide were not mentioned by name.

surements—the smoker still absorbed approximately the same amount of nicotine. Also, measurements from the chemical analysis of tobacco in 15 brands of cigarettes showed little differences in nicotine content, but smoking machines reported much greater differences. Benowitz and colleagues[19] concluded that "the tobacco in low yield cigarettes does not 'contain' less nicotine than higher yield cigarettes and that smokers of these cigarettes do not consume less nicotine."



The Weather

The Washington Post

THURSDAY AUGUST 10, 1978

Index

Some Cigarettes Now 'Tolerable,' Doctor Says

By Victor Cohn

Washington Post Staff Writer

Some cigarettes now have so little tar, nicotine and other harmful elements that they can be called "less hazardous" and can even be smoked in "tolerable" numbers without appreciable ill effects on the average smoker, a leading federal cancer scientist said yesterday.

Cigarettes in recent years have been found guilty beyond reasonable doubt of causing lung cancer, heart disease and several other illnesses.

But Dr. Gio Batta Gori, deputy director of cancer prevention at the National Cancer Institute, and Dr. Cornelius J. Lynch said yesterday there has been huge progress "in the last year and year and a half" in removing toxins — or poisons — from some brands. They expect to report this soon in the Journal of the American Medical Association.

"I am not calling any cigarette safe," Gori emphasized. "The only cigarette that is safe is the cigarette that is not lit.

"I am not talking about what might happen to any individual. I am talking about averages. There may be a risk that still may be there even though we might not see it in overall, large population studies.

Still, he said, there has been so much progress in removing toxins that we can now begin to talk about 'tolerable' levels of smoking from an overall public health standpoint. I think we will begin to see some beneficial effect in this country"—that is, some abatement in this nation's lung cancer epidemic—"in five or six years."

Gori said one brand—Carlton Menthols—is so low in the toxins that most persons could smoke 23 a day, more than a pack, with no measurable rise beyond a nonsmoker's.

He said the average smoker might similarly consume 18 Now Menthols, 17 Nows or Strides or 16 Carltons without any problems beyond a nonsmoker's so far as large-scale statistical studies could detect.

A cancer institute spokesman yesterday said Gori "probably represents the best expertise we have on smoking and health." Until two weeks ago he headed the institute's program in that field, including the effort to learn to make less hazardous cigarettes.

Dr. Arthur Upton, cancer institute director, nonetheless issued a more cautious statement yesterday saying "our present knowledge does not permit us to establish" any levels "below which smoking might be safe."

"It is the firm position of the National Cancer Institute, the National Heart, Lung and Blood Institute and the Public Health Service that no cigarette now on the market can be considered wholly without risk to health," he said.

He said Gori's figures cannot indicate whether the risk to any single individual is large or small, only that it is "not demonstrable" in epidemiologic or population studies. Upton said "you might double your own risk of cancer," for example, "with no appreciable effect" on large-scale population studies."

Gori also said there are many "high risk" individuals who should "never" smoke. These include present or former workers exposed to asbestos or the families of such workers, women using oral contraceptives, pregnant women, and persons with any of the heart, lung or breathing disorders linked to cigarettes.

Some scientists would also caution anyone exposed to any potentially cancer-causing chemical, since many harmful chemicals add to each others' effects in ways that are still little understood.

"What I am saying," Gori explained, "is that the average effect of smoking 'tolerable' levels of certain "less hazardous" cigarettes today would be as small as the ill effect on persons who smoked only two cigarettes a day before 1960. Virtually all cigarettes then were loaded with poisons like tar and nicotine far beyond present levels.

Gori disclosed his estimates first in an interview with Associated Press reporter Michael Putzel. The Washington Post then obtained a copy of the Gori-Lynch report.

Lynch is manager of the smoking and health program at Enviro Control Inc. of Rockville, the cancer institute's main contractor in the effort.

The five leading brands of less hazardous cigarettes on the Gori-Lynch list—Carlton Menthols, Now Menthols, Nows, Strides and Carltons—represented just under 2 percent of all cigarettes sold last year, Lynch estimated. But 22 more brands—with 17 to 18 percent of the market—are low enough in toxins so the average smoker might use between three and eight a day without appreciable added risk, Lynch said.

The cancer institute program to remove or minimize the effect of the main toxins found in cigarettes or cigarette smoke—tar or "total particulate matter," nicotine, carbon monoxide, nitrogen oxides and acrolein—began in 1970. At the most it has cost $4 million a year, a drop in the bucket compared to the cost of smoking's huge toll in death and disease.

Still, it is a program that has often been flayed by some critics of smoking who have argued that efforts should be concentrated on halting smoking altogether.

[illegible] program," Gori replied. "We are not [illegible] to endorse [illegible] or [illegible] ing in any way. We are only trying to put the facts before the public.

If we could go from [illegible] of lung cancer a year to, say, 2,000 [illegible] by the development of less hazardous cigarettes, I would still regret the 2,000 or [illegible] deaths but I would think we would have made a tremendous public health gain."

Doctor's List of Cigarettes And Their 'Tolerable' Levels

Following is the number of cigarettes the average smoker could consume daily without exceeding a "tolerable" level of risk of cancer or other disease, according to Dr. Gio Batta Gori.

Carlton Menthol	23
Now Menthol	18
Now	17
Stride	17
Carlton	16
L&M Flavor Lights (King)	8
Lucky 100	8
True	8
True Menthol	8
Decade	7
Pall Mall Extra Mild	7
Decade Menthol	6
Iceberg 100s	6
Kent Golden Lights Menthol	6
L&M Long Lights (100s)	6
Lark II	6
Tareyton Lights	6
Kent Golden Lights	5
Real Menthol	5
Benson & Hedges Lights	4
Newport Lights Menthol	4
King Sano	3
King Sano Menthol	3
Merit	3
Merit Menthol	3
Real	3
Tempo	3

Cigarettes not named were not listed by Dr. Gori.

FIGURE 3. Store display advertisement distributed by Liggett in 1978 used a newspaper article with a doctor's seeming approval of certain "tolerable" brands. The doctor referred to was Gio Gori, who holds a PhD but not an MD.

The accuracy of smoking machines in establishing true estimates of tar and nicotine in cigarettes is challenged again by Grunberg et al[20] in this issue of the *Journal*. They note that as the butt length of cigarettes has become longer, the reported number of puffs has become fewer. These differences could account for the lower ranking of tar and nicotine of different cigarette brands by the rating agencies. Thus, smoking machines report lower tar and nicotine content than that actually consumed by the smoker.

Another example of the inability of smoking machines to provide accurate measurements of how much tar and nicotine smokers absorb is demonstrated in an ongoing legal battle brought about by competing American cigarette companies over the advertising claims for the Actron filter of British American Tobacco's Barclay cigarette ("99% tar free") (Figs 4, 5). In the United States, the Federal Trade Commission noted in an injunction against Brown and Williamson Tobacco Company that the ventilation channels near the top of the cigarette, which were supposed to reduce tar and carbon monoxide intake, were blocked by the smoker's lips but not by the smoking machine. Advertisements have claimed a nicotine level of 0.1 mg; however, independent testing supported by the FTC showed a measurement of 0.3 to 0.7 mg. This legal altercation has caused considerable concern in England, where the Department of Health and Social Security is investigating the possibility of eliminating smoking machine data and is reevaluating the recommendation that smokers shift to a low tar-low nicotine cigarette.

ADDITIVES

Another serious problem with the low tar-low nicotine cigarette is the recent evidence that a number of the com-

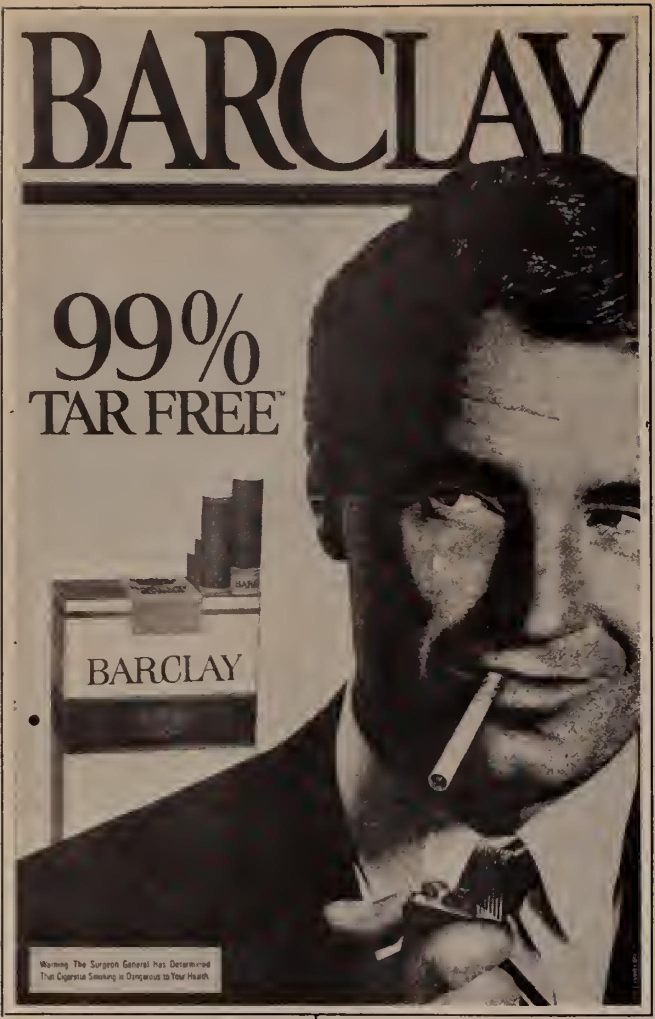
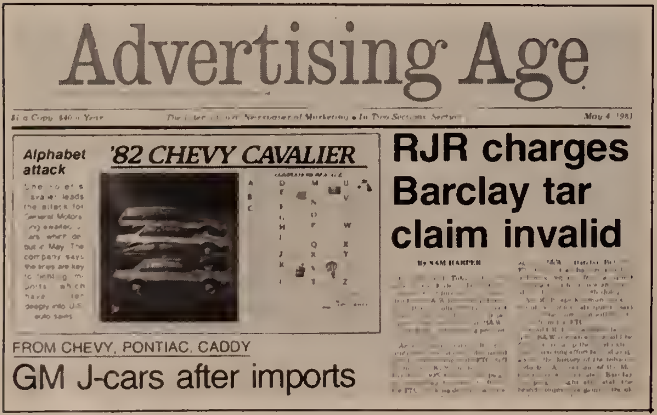

BARCLAY
99%
TAR FREE
BARCLAY
Warning: The Surgeon General Has Determined That Cigarette Smoking Is Dangerous to Your Health

FIGURE 4. Only 1% poison remaining?

pounds being added to cigarettes to make them more palatable are dangerous to the health of the smoker. For example, cocoa is found to increase the incidence of cancer in mice, and deer tongue is found to cause liver damage.[18] Since these additives are trade secrets, even the recent cigarette labeling bill passed by Congress holds little promise that the public will learn much about the chemicals that go into cigarettes.


Advertising Age

May 4 1981

Alphabet attack

'82 CHEVY CAVALIER

RJR charges Barclay tar claim invalid

FROM CHEVY, PONTIAC, CADDY

GM J-cars after imports

FIGURE 5. Low-tar claims are a front-page concern to the advertising profession.

The most damaging evidence concerning the hypothetical benefits of a "less hazardous" cigarette has been produced by recent epidemiologic studies. In Connecticut, the incidence of lung cancer is 10% higher for women in the 35 to 44 year age cohort than for men of the same age cohort.[22] In this age group the number of years women have smoked approaches that of men, a condition not true for older age cohorts. Since most women now smoke low tar-low nicotine cigarettes, the Connecticut study provides no evidence of the reduced cancer incidence of the newer cigarettes. A report by the Northwestern Pennsylvania Study on Smoking and Health[23] showed that those who smoke filter cigarettes die earlier than those who smoke nonfilter cigarettes. The most recent report of the Framingham study,[24] which shows that those who smoked filter cigarettes had a higher incidence of cardiovascular disease than those who smoked nonfilter cigarettes, corroborates the evidence of the Pennsylvania study.

A review of research in 1983 and 1984 on the low tar-low nicotine cigarette, as reported in the *Bibliography on Smoking and Health* (Washington, DC, Office on Smoking and Health) shows that all experimental studies presented evidence that the so-called less hazardous cigarettes are either no less hazardous or are simply not safe. It is interesting to note that beginning with the 1981 *Bibliography on Smoking and Health,* the major heading of "Cigarettes, Less Hazardous" has been deleted and is now replaced by the categories "Cigarettes—Low Nicotine, High Nicotine, High Tar, Low Tar, and Low Yield."

The realities of the smoking process make the production of a "less hazardous" cigarette highly improbable. The combustion of any types of compounds that have been used in cigarettes will produce the following: carbon monoxide, which is detrimental to the cardiovascular system; tars, which are saturated with carcinogenic compounds; nitrogen oxides, which form acids capable of dissolving lung tissue and causing bronchitis and emphysema; and hundreds of other compounds such as cyanide and acrolein which are potentially dangerous to the health of both smokers and the people around them.

ENDING "LESS HAZARDOUS" CIGARETTE RESEARCH

The less hazardous cigarette is a hypothetical construct developed from earlier unsubstantiated research and wishful thinking. Based on the optimistic preliminary reports, cigarette companies began to echo these claims and have continued to use this information to advertise the safety of cigarettes based on the curtailment of tar and nicotine. The evidence presented in this article shows that inaccurate information is being used to advertise cigarettes.

The following measures are recommended to redress this situation:

- All funding for the development of a "less hazardous" cigarette should be discontinued.*
- Funding for smoking machine research should also be dis-

---

* According to an article in *American Medical News* (Gapen P: A safer tobacco is target of USDA. Apr 5, 1985: 2, 35), the US Department of Agriculture has continued to spend $5.3 million a year on research designed to develop safer tobacco. —Editor

continued, since their data have been shown to be highly inaccurate.

- Funding should be available from all levels of government to support and publicize "stop smoking" clinics with verifiable success rates for those who wish to quit smoking.
- Funding should be made available for advertising to counter the advertising efforts of the cigarette industry, especially in regard to low tar-low nicotine claims.
- The business community should expand efforts to encourage the workforce to stop smoking by providing funding to develop its own programs to assist in smoking cessation.

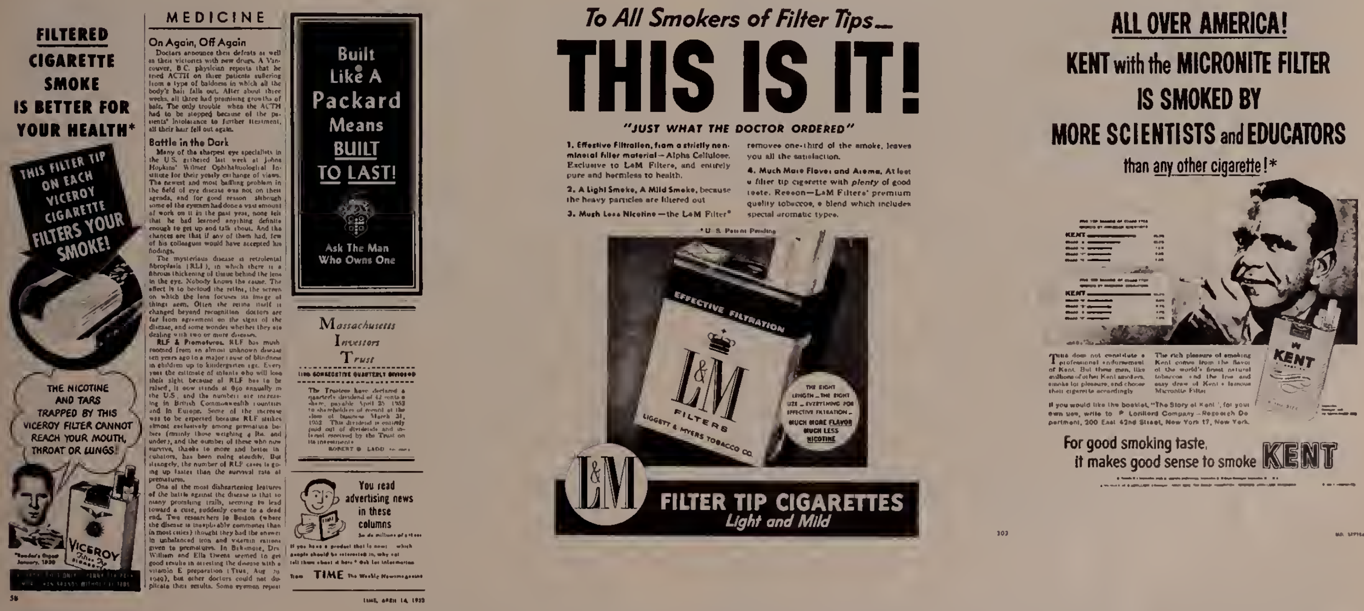
It is imperative that physicians realize that there will never be a "less hazardous" cigarette. Instead of recommending that the smoker change to low tar-low nicotine cigarettes, physicians should advise all patients to stop smoking. For those unable to stop on their own, physicians should undertake to develop skills in better educating patients on how to stop smoking and analyze the methods and verifiable success rates of the various clinics before referring patients to them.

The evidence should now be clear: the "less hazardous" cigarette is a deadly delusion.

REFERENCES

1. Bross ID, Gibson R: Risks of lung cancer in smokers who switch to filter cigarettes. *Am J Public Health* 1968; 58:1396-1403.
2. Wynder EL, Mabuchi K, Beattie EJ Jr: The epidemiology of lung cancer. *JAMA* 1980; 213:2221-2228.
3. Miller GH: The filter cigarette controversy. *J Indiana State Med Assoc* 1979; 72:903-904.
4. Astrup P, Kjeldsen K, Wanstrup J: Enhancing influence of carbon monoxide on the development of atheromatosis in cholesterol-fed rabbits. *J Atheroscler Res* 1967; 7:343-354.
5. Kjeldsen K, Wanstrup J, Astrup P: Enhancing influence of arterial hypoxia on the development of atheromatosis in cholesterol-fed rabbits. *J Atheroscler Res* 1968; 8:835-845.
6. Ayres SM, Giannelli S Jr, Mueller H: Myocardial and systemic responses to carboxyhemoglobin. *Ann NY Acad Sci* 1970; 174:268-293.
7. Aronow WS: Carbon monoxide and cardiovascular disease, in Wynder EL, Hoffman D, Gori GB (eds): *Proceedings of the 3rd World Conference on Smoking and Health, vol 1, Modifying the risk for the smoker.* DHEW publication no. (NIH) 76-1221. 1976:321-328.
8. Kurt TL, Mogielnicki PR, Chandler JE: Association of the frequency of acute cardiorespiratory complaints with ambient levels of carbon monoxide. *Chest* 1978; 74:10-14.
9. Wald N, Boreham J, Bailey A: Relative intakes of tar, nicotine, and carbon monoxide from cigarettes of different yields. *Thorax* 1984; 39:361-364.
10. Gori GB: Low risk cigarettes: a prescription. *Science* 1976; 194:1234-1246.
11. Gori GB, Lynch CJ: Toward less hazardous cigarettes. *JAMA* 1978; 240:1255-1259.
12. Barclay WR: Smoke screens. (editorial) *JAMA* 1978; 240:1271.
13. Warner KE: Toward less hazardous cigarettes. (letter) *JAMA* 1979; 241:2143-2144.
14. Gart JJ, Schneiderman MA: "Low risk" cigarcttes: the debate continues. (letter) *Science* 1979; 240:688.
15. Miller GH, Schumaker JA: Less hazardous cigarettes debated. (letter) *JAMA* 1979; 242:1029.
16. US Public Health Service: *Health Consequences of Smoking: The Changing Cigarette, a report of the Surgeon General.* DHHS(PHS) 81-50156, 1981.
17. Gerstein DR, Levison PK (eds): *Reduced Tar and Nicotine Cigarettes: Smoking Behavior and Health.* Washington DC, National Academy Press, 1982.
18. Rickert WS: "Less hazardous" cigarettes: fact or fiction? *NY State J Med* 1983; 83:1269-1272.
19. Benowitz NL, Hall SM, Herning RI, Jacob P III, Jones RT, Osman AL: Smokers of low-yield cigarettes do not consume less nicotine. *N Engl J Med* 1983; 309:139-142.
20. Grunberg NE, Morse DE, Maycock VA, Kozlowski LT: Changes in overwrap and butt length of United States filter cigarettes: an influence on reported tar yields. *NY State J Med* 1985; 85:310-312.
21. Mintz J: Low tar, high risk. *Mother Jones,* Feb-Mar 1983, pp 54-56.
22. Meigs JW: Epidemic lung cancer in women. *JAMA* 1977; 238:1055.
23. Miller GH: World smoking health report: filter or non-filter cigarettes—which is safer? report no. 4, *J Breathing.* Illinois Lung Assoc 1977; 40.
24. Castelli WP: Epidemiology of coronary heart disease: The Framingham study. *Am J Med* 1984; 76:4-12.

HEALTH CLAIMS MADE FOR FILTER CIGARETTES



FILTERED CIGARETTE SMOKE IS BETTER FOR YOUR HEALTH*

THIS FILTER TIP ON EACH VICEROY CIGARETTE FILTERS YOUR SMOKE!

THE NICOTINE AND TARS TRAPPED BY THIS VICEROY FILTER CANNOT REACH YOUR MOUTH, THROAT OR LUNGS!

*Reader's Digest January, 1950

VICEROY
Filter Tip

58

MEDICINE

On Again, Off Again

Doctors announce their defeats as well as their victories with new drugs. A Vancouver, B.C. physician reports that he tried ACTH on three patients suffering from a type of baldness in which all the body's hair falls out. After about three weeks, all three had promising growths of hair. The only trouble: when the ACTH had to be stopped because of the patients' intolerance to further treatment, all their hair fell out again.

Battle in the Dark

Many of the sharpest eye specialists in the U.S. gathered last week at Johns Hopkins' Wilmer Ophthalmological Institute for their yearly exchange of views. The newest and most baffling problem in the field of eye disease was not on their agenda, and for good reason: although some of the eyemen had done a vast amount of work on it in the past year, none felt that he had learned anything definite enough to get up and talk about. And the chances are that if any of them had, few of his colleagues would have accepted his findings.

The mysterious disease is retrolental fibroplasia (RLF), in which there is a fibrous thickening of tissue behind the lens in the eye. Nobody knows the cause. The effect is to becloud the retina, the screen on which the lens focuses its image of things seen. Often the retina itself is changed beyond recognition: doctors are far from agreement on the signs of the disease, and some wonder whether they are dealing with two or more diseases.

RLF & Prematures. RLF has mushroomed from an almost unknown disease ten years ago to a major cause of blindness in children up to kindergarten age. Every year the estimate of infants who will lose their sight because of RLF has to be raised. It now stands at 650 annually in the U.S., and the numbers are increasing in British Commonwealth countries and in Europe. Some of the increase was to be expected because RLF strikes almost exclusively among premature babies (mainly those weighing 4 lbs. and under), and the number of these who now survive, thanks to more and better incubators, has been rising steadily. But strangely, the number of RLF cases is going up faster than the survival rate of prematures.

One of the most disheartening features of the battle against the disease is that so many promising trails, seeming to lead toward a cure, suddenly come to a dead end. Two researchers in Boston (where the disease is inexplicably commoner than in most cities) thought they had the answer in unbalanced iron and vitamin rations given to prematures. In Baltimore, Drs. William and Ella Owens seemed to get good results in arresting the disease with a vitamin E preparation (TIME, Aug. 29, 1949), but other doctors could not duplicate their results. Some eyemen report

Built Like A Packard Means BUILT TO LAST!

Ask The Man Who Owns One

Massachusetts Investors Trust

110th CONSECUTIVE QUARTERLY DIVIDEND

The Trustees have declared a quarterly dividend of 42 cents a share, payable April 25, 1952 to shareholders of record at the close of business March 31, 1952. This dividend is entirely paid out of dividends and interest received by the Trust on its investments.

ROBERT W. LADD [illegible]

You read advertising news in these columns

So do millions of others

If you have a product that is news which people should be interested in, why not tell them about it here? Ask for information from TIME The Weekly Newsmagazine

TIME, APRIL 14, 1952

To All Smokers of Filter Tips—

THIS IS IT!

"JUST WHAT THE DOCTOR ORDERED"

1. Effective Filtration, from a strictly non-mineral filter material—Alpha Cellulose. Exclusive to L&M Filters, and entirely pure and harmless to health.

2. A Light Smoke, A Mild Smoke, because the heavy particles are filtered out.

3. Much Less Nicotine—the L&M Filter* removes one-third of the smoke, leaves you all the satisfaction.

4. Much More Flavor and Aroma. At last a filter tip cigarette with *plenty* of good taste. Reason—L&M Filters' premium quality tobaccos, a blend which includes special aromatic types.

*U. S. Patent Pending

EFFECTIVE FILTRATION
L&M
FILTERS
LIGGETT & MYERS TOBACCO CO.
THE RIGHT LENGTH—THE RIGHT SIZE—EVERYTHING FOR EFFECTIVE FILTRATION—MUCH MORE FLAVOR MUCH LESS NICOTINE

L&M FILTER TIP CIGARETTES
Light and Mild

ALL OVER AMERICA!

KENT with the MICRONITE FILTER IS SMOKED BY MORE SCIENTISTS and EDUCATORS than any other cigarette!*

This does not constitute a professional endorsement of Kent. But these men, like millions of other Kent smokers, smoke for pleasure, and choose their cigarette accordingly.

The rich pleasure of smoking Kent comes from the flavor of the world's finest natural tobaccos and the free and easy draw of Kent's famous Micronite Filter.

If you would like the booklet, "The Story of Kent", for your own use, write to P. Lorillard Company—Research Department, 200 East 42nd Street, New York 17, New York.

For good smoking taste, it makes good sense to smoke KENT

In the 1950s, confronted with declining cigarette sales following the publication of studies linking smoking to lung cancer, tobacco companies began producing filter-tips that were claimed to selectively remove certain components of smoke that manufacturers to this day have never acknowledged to be harmful. Left, for several years, Brown & Williamson bought the advertising space alongside the Medicine section of *TIME* to claim that Viceroy cigarettes offered double-barrel health protection (as in this ad from 1952) and that persons with a cold should switch to the company's menthol brand, KOOL. Middle, "JUST WHAT THE DOCTOR ORDERED" claimed advertisements in 1953 in magazines and on radio and television for Liggett & Myers' L & M cigarettes. Right, Lorillard's advertisement for KENT appeared in the medical publication *MD* in 1960.

Cipollone v Liggett Group, Inc

The application of theories of liability in current cigarette litigation

MARC Z. EDELL, ESQ, STEWART M. GISSER, ESQ

Liggett Group, Inc, Lorillard, Philip Morris, American Brands, Brown and Williamson, RJ Reynolds Tobacco Co, and other cigarette producers, as well as the Tobacco Institute, are defending lawsuits brought by individuals suffering and dying from lung cancer and other cigarette-related diseases. Executive officers and advertising representatives of these companies can no longer evade public scrutiny by means of prepared statements issued by public relations departments. They are now testifying under oath at depositions, and, as a result of court orders, are answering interrogatories and producing documents that may provide the factual bases to support jury findings of compensatory as well as punitive damages against their companies.

Rose Cipollone began smoking in 1942, when she was 16 years old. She smoked one and a half to two packs of cigarettes per day until 1981, when a lesion was discovered in her lung. A lobectomy was performed in 1981; the pathologic report was that of small-cell carcinoma. She continued to smoke cigarettes while in the hospital. A lawsuit was brought on her behalf in United States District Court for the District of New Jersey in August 1983 against the manufacturers of the cigarettes smoked by Mrs Cipollone. Rose Cipollone died in 1984, at the age of 58, as a result of the metastasis of her disease. An amended complaint has been filed on behalf of her estate.

Although this article concentrates on the legal theories espoused in the Cipollone case, the claims and defenses of those claims are similar to those that will be litigated throughout the country. An analysis of this case thus offers a paradigmatic view of present cigarette litigation.

"RISK-UTILITY ANALYSIS"

Early unsuccessful cases brought against the cigarette companies were premised on product-liability law, which was still in its embryonic stages. Recent changes in the law regarding "design defect" and "failure to warn" have now given plaintiffs the means to prevail. The basic theory of strict liability predicates recovery on a showing that the product causing the injury is defective—that is, not reasonably fit, suitable, and safe for its intended and foreseeable purposes.[1] A manufacturer of a product has a duty to warn foreseeable users of the risks inherent in its use, and not to place defective products on the market.[2] The standard employed for determining whether a particular product is defective is paramount.

In cases such as *Cipollone,* involving claims of design defect and inadequate warnings, one standard by which the product would be measured is whether the product is so dangerous that it creates a risk of harm that outweighs its usefulness. This benchmark, never before applied in cigarette litigation, is known as "risk-utility analysis."

In the recent decision of *O'Brien v Muskin,* the New Jersey Supreme Court stated the following:

> [O]ther products, including some for which no alternative exists, are so dangerous and of such little use that under the risk-utility analysis, a manufacturer would bear the cost of liability of harm to others. That cost might dissuade the manufacturer from placing the product on the market, even if the product has been made as safely as possible.[3]

The court in *O'Brien* adopted the following criteria as relevant in determining whether a product's risk outweighs its utility:

- The usefulness and desirability of the product—its utility to the user and to the public as a whole*;
- The safety aspects of the product—the likelihood that it will cause injury, and the probable seriousness of the injury;
- The availability of a substitute product which would meet the same need and not be as unsafe;
- The manufacturer's ability to eliminate the unsafe character of the product without impairing its usefulness or making it too expensive to maintain its utility;
- The user's ability to avoid danger by the exercise of care in the use of the product;
- The user's anticipated awareness of the dangers inherent in the product and their avoidability, because of general public knowledge of the obvious condition of the product, or of the existence of suitable warnings or instructions;
- The feasibility, on the part of the manufacturer, of spreading the loss by setting the price of the product or carrying liability insurance.[4]

Mr Edell is a principal of Porzio, Bromberg & Newman, Morristown, NJ. Mr Gisser is an associate of the firm.

Address correspondence to Mr Edell, Counsellor at Law, Porzio, Bromberg & Newman, 163 Madison Ave, Morristown, NJ 07960-1997.

The authors, in association with David J. Novack, ESQ of Budd, Larner, Kent, Gross, Picillo, Rosenbaum, Greenberg & Sade, and Alan M. Darnell, ESQ of Wilentz, Goldman & Spitzer have brought eight lawsuits against various manufacturers of cigarettes. These cases are pending in state and federal courts in New Jersey. The authors have also been involved in "toxic tort" litigation involving exposures to arsenic, asbestos, lead, polyvinyl chloride, and urea foam formaldehyde.

* Products are not removed from the market based on a "risk-utility" analysis. Risk-utility analysis is a standard used to determine whether or not a product is defective, as such a finding is a predicate for recovery under strict liability. Thus, there are no concrete examples of products that have been removed from the market based on such an analysis.

The New Jersey Supreme Court recognized that the plaintiff need not produce evidence with regard to all of the above criteria to establish that a product is defective.[5] Rather, the standard is flexible. The court in *O'Brien* explained that the evaluation of the utility of a product takes into consideration the relative need for the product and acknowledged that a luxury item may be viewed differently under the above analysis than would, for example, a therapeutic device.[6] A court may be more likely to find utility in a therapeutic device, than in a non-essential product.

In applying the above analysis to cigarettes, it seems clear that with regard to the first two criteria, the suffering and death of 350,000 Americans per year as a result of cigarette smoking, as well as the economic costs to the public of lost work time and increased medical insurance premiums, outweigh any social utility claimed to accompany cigarette smoking. The cigarette companies claim that cigarette smoking provides enjoyment to millions in this country.

Moreover, in 1969, Joseph F. Cullman, III, then chairman of the Board of Directors and chief executive officer of Philip Morris, Inc, and chairman of the Executive Committee of the Tobacco Institute, testified before Congress as spokesman, in his words, for the manufacturers of virtually all cigarettes produced in this country, as to the importance of the tobacco industry to the national economy. Cullman referred to the farmers that derive their livelihood from tobacco's contribution to the balance of trade in the United States, the income tobacco sales provide to merchants, and the state and federal tax revenues tobacco sales generate. Cullman warned Congress that "All these factors point to the conclusion that extreme care should be exercised before any action is taken which could seriously disrupt this important industry."[7]

With regard to the third, fourth, and fifth criteria, the literature supports the view that there exists neither a "safe" cigarette, nor a "safe level" of smoking. As to the sixth criterion, the Federal Trade Commission has made annual reports to Congress since 1967 pursuant to the mandate of the Cigarette Labeling Act, regarding the effectiveness of the cigarette warning label on packages of cigarettes. The Federal Trade Commission has concluded that the health warning label that appears on packages of cigarettes and in advertising is inadequate and ineffective in conveying to the public the "true hazards" attendant to cigarette smoking. Regarding the last of the factors to be considered, there is no question that cigarette manufacturers can spread their losses both by obtaining general liability insurance and by increasing the cost of cigarettes. Based upon the foregoing, the plaintiff in *Cippolone* will argue that the risks of cigarette smoking outweigh any social utility, and that therefore, cigarette products are defective as a matter of law.

"FAILURE TO WARN"

Other significant changes in product liability law have occurred in "failure to warn" cases. In *Feldman v Lederle,*[8] a case involving a pharmaceutical company, the court held that the "state of the art" defense—a defense based on knowledge of the industry at the time the product was manufactured and marketed—was relevant in a failure to warn case involving strict product liability. The court, however, placed a heavy burden on the defendant in that case, holding that "the defendant should properly bear the burden of proving that the information was not reasonably available or obtainable and that it, therefore, lacked actual or constructive knowledge of the defect."[9]

In cases associated with cigarettes, assuming that the court will apply the law as stated in *Feldman,* the burden of proving that there were no scientific data or other pertinent information that would "give rise to a reasonable inference that the danger is likely to exist" is placed upon the tobacco company defendants. Thus, the cigarette companies' usual litany of reasons for failing to warn, such as "it hasn't been proven," "causal relationship can never be established through epidemiologic study," and "one cannot extrapolate from animal studies to human beings to show a causal relationship," are deficient under the above standard. The standard is not *certainty* before the manufacturer has a duty to warn, but rather the availability of data and information that would give rise to a *reasonable inference* that the danger existed. Cigarette manufacturers would be hard pressed to take the position that, at least as of the 1950s, there was no scientific and medical literature available that "gave rise to a reasonable inference" that there were dangers attendant to the use of their product.

Recent cases have held that inadequate warnings in and of themselves may constitute design defects.[10] Plaintiffs in the *Cipollone* case claim that inadequate health warnings were provided by the defendants. The plaintiffs present the novel allegation that the defendants promoted, marketed, and advertised their products in such a way as to neutralize or negate the limited warnings that were provided.

In support of the plaintiff's claims is the fact that no warnings appeared on cigarette packages or were displayed with cigarette advertising prior to 1966. Even after 1966, despite the defendants' knowledge of countless studies linking cigarette smoking with disease, and, in particular, with lung cancer, defendants failed to warn with specificity and without qualification of the established risks attendant to cigarette smoking, including the risk of addiction.

In addition, the plaintiff will introduce expert testimony as to the effect on Rose Cipollone of the advertising and promotion utilized by defendants. The plaintiff contends that the defendants negligently and/or intentionally, through their advertising and promotion, induced Rose Cipollone to begin smoking, negated and neutralized antismoking warnings and health literature, and reinforced her cigarette smoking, all of which resulted in her dependence on cigarettes.

CONSPIRACY TO MISLEAD?

The plaintiff also alleges a conspiracy between the Tobacco Institute and tobacco manufacturers to mislead the public. In support of such a claim, the plaintiff brought to the court's attention a Federal Trade Commission report to Congress, documenting attempts by the Tobacco Institute to mislead the public by disseminating medical articles containing half-truths and misrepresenting scientific evidence to physicians, lawyers, civic leaders, and other citizens through a public relations firm hired by the institute.[11]

The articles (one of which was entitled, "Cigarette-cancer link is bunk") appearing in *True Magazine* and the *National Enquirer,* were written by a freelance sports writer who had been approached by the then editor of *True* and a public relations man retained by Brown and Williamson to write an article on the tobacco company's position regarding the relationship between cigarette smoking and health. Background material for the story was provided to the writer by an attorney retained by one of the tobacco companies. The note accompanying the reprints sent around the country gave the impression that the mailings came from the editors of *True,* when they had in fact been sent by Tiderock Corp, a public relations firm hired by the Tobacco Institute, and paid for by individual tobacco companies.

The importance and the relevance of this type of information to a plaintiff's claims is clear. The New Jersey Supreme Court stated the following in *Board of Education, Asbury Park v Hoek*:

> . . . it is well known that the nature of a conspiracy is such that more often than not the only type of evidence available to prove the conspiracy is circumstantial evidence. And although the verdict must not be the result of pure speculation [citations omitted], circumstantial evidence will suffice [citations omitted].[12]

Among the defenses to the plaintiff's claims relied on by the cigarette companies is the defense of contributory negligence. In order to prevail on that defense, the defendants must show that the plaintiff voluntarily encountered a known danger. Defendants have argued that the plaintiff was aware of the "alleged" dangers of cigarette smoking through antismoking commercials and warnings placed on cigarette packages and voluntarily chose to continue smoking.

The basic flaw in the defendants' argument is the fact that there was no voluntary encounter of a known danger. First, the defendants deny that any danger exists. How, then, can an individual voluntarily encounter a nonexistent danger? The defendants will argue that, although they do not accept the causal connection between cigarette smoking and disease, most people do. The fact of the matter, however, is that most smokers do not believe the link between cigarettes and disease. This position was even taken by the tobacco industry before Congress.[13]

Rose Cipollone began smoking more than 20 years before there was a warning on packages of cigarettes. This fact is significant for a number of reasons, the first of which deals with the subject of addiction. Because the plaintiff was addicted to cigarettes before there were warnings on the packages, the 1966 warning had little effect on her "voluntary" assumption of the risk attendant to cigarette smoking. Moreover, for the purposes of liability, the question with regard to addiction is not whether there are people who can stop, but, rather, whether it is reasonably foreseeable to the cigarette manufacturers that there is a significant number of people who can not stop.

The fact that the plaintiff's smoking habits predated the cigarette health warnings by more than 20 years is also important because it resulted in her unwillingness to accept the proposition set forth by the Surgeon General. The plaintiff believed that if cigarette smoking were harmful, then the federal government would not permit the sale of cigarettes, and the cigarette companies would not sell them. (This is based on her assumption that the government was acting in her best interest.)

In addition to addiction removing the "voluntary" aspect of the plaintiff's assumption of the risks of cigarette smoking, the cigarette companies, through their advertising, negated and neutralized the health warning by presenting images and messages totally inconsistent with the health warning. This permitted the plaintiff to disassociate herself from the health warning and associate with the picture of health, well being, vitality, glamour, sophistication, and excitement depicted in advertisements.

Although the cigarette companies claim that peer group pressure causes people to smoke, social acceptability of the practice of smoking was created by cigarette advertising and is continually reinforced. Before cigarette advertising existed, it was not a sign of adulthood, maturity, or sophistication to smoke cigarettes. Rather, it is the plaintiff's position that it was through the use of movie stars and other celebrities in promotional materials that the smoking of cigarettes achieved an aura of respectability and glamour. Mrs Cipollone stated this position at her deposition:

> Q: How did you come about starting to smoke?
> A: Well, I used to see ads . . . .[14]
> Q: How did you happen to select Chesterfields?
> A: I thought it was glamorous. I used to see the ads of these pretty girls and movie stars. I use to go to the candy store on the corner and there were ads. I thought it was ladylike.[15]

"PREEMPTION" DEFENSE

One of the cigarette manufacturers' key defenses has been that of "preemption." The defendants claimed that certain provisions of the 1965 Cigarette Labeling Act precluded product liability actions such as those that have been filed. They argued that if the courts permitted the plaintiff to proceed on a theory of inadequate warning, a concept developed through state common law, then the state would indirectly be imposing additional requirements on the cigarette manufacturers in contravention of the following express provisions of the act:

> A. No statement relating to smoking and health other than the statement required by Section 1333 of this Title, shall be required on any cigarette package.
> B. No requirement or prohibition based on smoking and health shall be imposed under State law with respect to the advertising or promotion of any cigarettes the packages of which are labeled in conformity with the provisions of this Chapter.

Prior to the *Cipollone* case, there had never been a decision by any court in the United States regarding these provisions. Recognizing the importance of the issue, the plaintiff's counsel filed a motion seeking an order striking the defense premised on the preemption provision. After extensive briefing by all parties, the Honorable H. Lee Sarokin, a federal judge of the United States District Court of New Jersey, filed an opinion and accompanying order on September 20, 1984 granting the plaintiff's motion to strike the defense that the preemption provision precluded the plaintiff's cause of action. The court ruled as follows:

> Congress did not, despite its solicitousness for the cigarette industry, deprive citizens of this recourse. Nor shall the court, in deference to Congress and with respect for the state common law and individuals' right to invoke it, do so. . . .
> In almost every instance, government standards are meant to fix

> a level of performance below which one should not fall. However, legal minimums were never intended to supplant moral maximums . . . . The Court here concludes that the warning of the Surgeon General fixes the minimum. Indeed indications are that the Surgeon General himself does not view the warning as adequate.[16]

The court found that the specter of tort liability does not regulate conduct under the Cigarette Labeling Act, but rather puts a choice to the cigarette manufacturers:

> [T]he producer of a defective product, who has been found liable in tort is put to a choice: it may avoid the risk of future liability by remedying the defect in its product or, at the extreme, by withdrawing such product from the market, or it may confront such risk, hoping that future juries, acting in light of different sets of facts, will find for it. Which course it takes depends upon a complex combination of economics, morality and psychology. In this sense, tort liability does not *regulate* at all. . ." [Emphasis supplied].[17]

By ruling that cigarette manufacturers were not precluded from placing their own more stringent warnings on their cigarette products, the court cleared the way for additional plaintiffs' suits based on claims of inadequate warnings. The defendants are appealing Judge Sarokin's ruling to the Third Circuit Court of Appeals.

It should be noted, however, that even in the event that defendants would prevail on appeal, the plaintiff's claims that defendants neutralized their warnings and misled the public through their advertising would survive. This was put forth by the court in *Cipollone*:

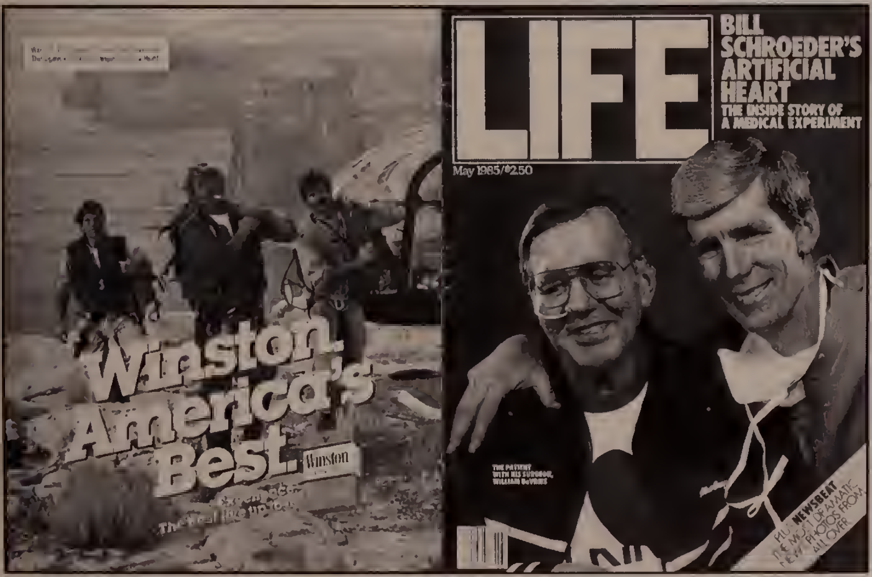
> Efforts to convince the public that the risks do not exist or that they are minimal or unsupported by medical or scientific data may in and of themselves give rise to a cause of action; indeed they may even constitute a violation of the very statute which defendants brandish as a shield.[18]

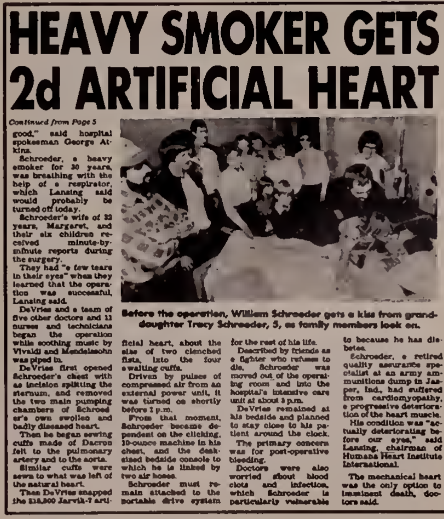
A major hurdle in the path of all those contemplating lawsuits against cigarette manufacturers has been overcome by the court's preemption ruling in *Cipollone*. The state of product liability law is more favorable to the potential plaintiff in a case involving cigarettes than it has ever been.

If plaintiffs establish causation and defeat the assumption of voluntary risk defense by competent evidence with regard to addiction, inadequacy of warnings, and the neutralizing of warnings in advertising, the jury verdicts against cigarette manufacturers will begin to appear. Those victimized by the tortious acts and omissions of the cigarette industry will at last be justly compensated.

REFERENCES

1. *Feldman v Lederle*, 97 NJ 429 (1984); *O'Brien v Muskin Corp*, 94 NJ 169 (1983).
2. *Feldman v Lederle*, 97 NJ 429 (1984); *O'Brien v Muskin Corp*, 94 NJ 169 (1983); *Michalko v Cooke Color & Chemical Corp*, 91 NJ 386 (1982); *Cepeda v Cumberland Engineering Co, Inc*, 76 NJ 152 (1978); reversed on other grounds, *Suter v San Angelo Foundry & Machine Co*, 81 NJ 150, 177 (1979).
3. *O'Brien v Muskin Corp*, 94 NJ 169, 184 (1983).
4. *O'Brien v Muskin Corp*, 94 NJ 169, 182 (1983); *Cepeda v Cumberland Engineering Co, Inc*, 76 NJ 152, 174 (1978); reversed on other grounds, *Suter v San Angelo Foundry & Machine Co*, 81 NJ 150, 177 (1979).
5. *O'Brien v Muskin Corp*, 94 NJ 169, 185 (1983).
6. *O'Brien v Muskin Corp*, 94 NJ 169, 184-185 (1983).
7. *Cigarette Labeling and Advertising—1969:* Hearings on HR 643, HR 1237, HR 3055, HR 6543 before the Committee on Interstate and Foreign Commerce House of Representatives, Ninety-First Congress, First Session (1969) (Statement of Joseph Cullman, III, p 552).
8. *Feldman v Lederle*, 97 NJ 429 (1984).
9. *Feldman v Lederle*, 97 NJ 429, 455–456 (1984).
10. *See Freund v Cellofilm Properties, Inc*, 87 NJ 229 (1981). *See also, Suter v San Angelo Foundry and Mach Co*, 81 NJ 150 (1979).
11. *Congressional Record*, March 27, 1968; *Federal Trade Commission Report to Congress* of June 30, 1968.
12. *Board of Education, Asbury Park v Hoek*, 38 NJ 213, 239 (1962).
13. *Smoking Prevention Education Act:* Hearings on HR 1824 before the Subcommittee on Health and the Environment of the Committee on Energy and Commerce, House of Representatives, Ninety-Eighth Congress, First Session March 9 and 17, 1983 (Statement of Curtis H. Judge, p 327).
14. Deposition of Rose Cipollone, *Cipollone v Ligget Group, Inc*, January 26, 1984, p 126.
15. Deposition of Rose Cipollone, *Cipollone v Liggett Group, Inc*, January 26, 1984, p 129.
16. *Cipollone v Liggett Group, Inc*, 593 F. Supp. 1146, 1170 (DNJ 1984).
17. *Cipollone v Liggett Group, Inc*, 593 F. Supp. 1146, 1156 (DNJ 1984).
18. *Cipollone v Liggett Group, Inc*, 593 F. Supp. 1146, 1148 (DNJ 1984).


Winston America's Best
Winston
LIFE
BILL SCHROEDER'S ARTIFICIAL HEART
THE INSIDE STORY OF A MEDICAL EXPERIMENT
May 1985/$2.50
THE PATIENT WITH HIS SURGEON, WILLIAM DeVRIES
PLUS NEWSBEAT THE MOST DRAMATIC NEW PHOTOS FROM ALL OVER



HEAVY SMOKER GETS 2d ARTIFICIAL HEART

Continued from Page 5

good," said hospital spokesman George Atkins.

Schroeder, a heavy smoker for 30 years, was breathing with the help of a respirator, which Lansing said would probably be turned off today.

Schroeder's wife of 32 years, Margaret, and their six children received minute-by-minute reports during the surgery.

They had "a few tears in their eyes" when they learned that the operation was successful, Lansing said.

DeVries and a team of five other doctors and 11 nurses and technicians began the operation while soothing music by Vivaldi and Mendelssohn was piped in.

DeVries first opened Schroeder's chest with an incision splitting the sternum, and removed the two main pumping chambers of Schroeder's own swollen and badly diseased heart.

Then he began sewing cuffs made of Dacron felt to the pulmonary artery and to the aorta.

Similar cuffs were sewn to what was left of the natural heart.

Then DeVries snapped the $18,500 Jarvik-7 artificial heart, about the size of two clenched fists, into the four awaiting cuffs.

Driven by pulses of compressed air from an external power unit, it was turned on shortly before 1 p.m.

From that moment, Schroeder became dependent on the clicking, 10-ounce machine in his chest, and the desk-sized bedside console to which he is linked by two air hoses.

Schroeder must remain attached to the portable drive system for the rest of his life.

Described by friends as a fighter who refuses to die, Schroeder was moved out of the operating room and into the hospital's intensive care unit at about 3 p.m.

DeVries remained at his bedside and planned to stay close to his patient around the clock.

The primary concern was for post-operative bleeding.

Doctors were also worried about blood clots and infection, which Schroeder is particularly vulnerable to because he has diabetes.

Schroeder, a retired quality assurance specialist at an army ammunitions dump in Jasper, Ind., had suffered from cardiomyopathy, a progressive deterioration of the heart muscle.

His condition was "actually deteriorating before our eyes," said Lansing, chairman of Humana Heart Institute International.

The mechanical heart was the only option to imminent death, doctors said.

Before the operation, William Schroeder gets a kiss from granddaughter Tracy Schroeder, 5, as family members look on.

Juxtaposition of the year: LIFE magazine, May 1985. Although artificial heart recipient William Schroeder called for a Coors beer soon after surgery, news reports have never mentioned his brand of cigarettes. Mrs Barney Clark, widow of the recipient of the first artificial heart, has appeared at Congressional hearings to publicize the fact that Dr Clark had smoked cigarettes for most of his life, resulting in emphysema and hastening the development of coronary heart disease.

Product liability in cigarette-caused fires

DONALD W. GARNER, ESQ

No tobacco company has ever been held responsible for any death, disability, or disease due to smoking.[1] However, the invincibility of the industry is being challenged in several recent personal injury lawsuits. Tobacco companies have lost lawsuits in cases in which truly weird things like snakes or toes were found in chewing tobacco.[1] But they have yet to pay compensation to the family of anyone who suffered a smoking-induced disease or who died or was disfigured in a fire started by a cigarette.

During the late 1950s and early 1960s, a number of unsuccessful lawsuits were filed on behalf of persons who claimed to have been made ill by cigarettes.[2] At that time, product law was not as favorable to the plaintiff, and the advocacy in some of the cases lacked vigor. But the most important factor was the well-financed and sustained defense of the tobacco companies. The most nearly successful case, *Green v American Tobacco,*[3] lasted 12 years, comprising six different appeals and two jury verdicts.

Today, plaintiffs' lawyers are better prepared to fight these cases to the finish. In addition, a plaintiff now has two alternative legal theories to sustain a product suit. The first and older theory is based on the concept that a consumer should be told about a product's dangers in order to choose whether to confront this risk. Individual choice, personal responsibility, and free will are the theoretical underpinnings of such liability. Thus, if a drug prescribed for arthritis causes blindness, and the manufacturer knew of this risk, liability will be imposed if the patient was not warned of the drug's danger.[4] This type of product law is called "the consumer expectation test" of liability or "failure to warn."

It is this theory that would be most likely to lead to a victory in a suit to recover damages for lung cancer, heart disease, or the myriad ailments attributed to cigarette smoking. Millions of individuals began smoking in an environment that portrayed cigarettes as a positive, even healthful custom. Cigarette manufacturers did not discharge their legal responsibility to warn consumers of the dangers of cigarette smoking, and the bland warnings required by federal law were insufficient.

The second and newer theory of liability is one that could be used by a plaintiff injured in a cigarette-caused fire. This theory questions whether the manufacturer could have eliminated the foreseeable dangers from the product. It does not depend on whether the consumer was ignorant of these dangers. Products that are subject to liability under this theory are those that contain "defects in design." Thus, if a patient is paralyzed when a doctor drills too deeply in the patient's cervical vertebrae during surgery because the bone cutter is too slippery, liability will be imposed on the manufacturer, since the product could have been designed to be safer.[5] Similarly, cigarettes should be designed so as not to set fires when accidently dropped or deliberately discarded.

While the defendant is the same in each type of liability case, lawsuits brought by a victim of fire and a victim of disease from smoking present an interesting contrast. Manufacturing a fire-safe cigarette long appears to have been feasible,[6] while making a cigarette safe for smoking is difficult or impossible.[7] According to the commissioner of the US Consumer Products Safety Commission, cigarette manufacturers add citrates to the paper that make cigarettes burn even when they are not being smoked.[8] In other words, there has been a conscious effort on the part of cigarette manufacturers to design cigarettes that will continue to burn without being smoked and will thereby ignite couches and beds when cigarettes are dropped. The safety of the consumer, and, even more importantly, the safety of innocent bystanders such as children in the neighboring apartment, must be factored into the design of the cigarette. Congress has established a commission to study the feasibility of producing a relatively fire-safe cigarette. The report is due at the end of 1985.

When a victim of lung cancer sues, however, it is doubtful he will be able to prove that the tobacco industry can market a cigarette that will not cause lung cancer. None of the current lawsuits assumes this difficult burden. The plaintiff with a disease alleged to be caused by smoking can point to the refusal of the cigarette companies ever to apprise the prospective smoker that cigarettes, in the words of William Pollin, MD, director of the National Institute on Drug Abuse, are "a powerfully addictive drug." Teenagers who begin smoking are not only filled with a sense of invincibility, but they also receive no reinforced message that cigarettes are dangerous and addictive. The cigarette companies' failure to discharge their responsibility to warn potential customers will be the lynch-pin of the lung cancer lawsuits.

A second distinguishing factor between a victim of fire and a victim of lung cancer is the relative simplicity of proving who did what to whom. In a fire case, a specific cigarette marketed by a specific defendant will cause specific damage to the plaintiff. The identity of the manufacturer, the brand of the cigarette, the cause of the fire, and the extent of the resulting injury will often be a matter of public record through the fire marshal's investigation and may not be difficult to prove. In a case of lung cancer, however, there likely will be a multitude of potential defendants and several different brands, without a clear as-

---

Address correspondence to Mr Garner, Professor of Law, Southern Illinois University at Carbondale, School of Law, Carbondale, IL 62901.

The author recently drafted the Cigarette Accountability Act which was introduced in the 1985 session of the Illinois Senate. The act would require the cigarette manufacturers to compensate family members who have suffered pecuniary loss as a result of cigarette-induced lung cancer or emphysema and to reimburse the state Medicaid fund for payments made to treat these diseases.

sociation between one particular brand and the disease. Other possible contributing factors such as environmental pollution and occupational exposures may be raised by the defendants. An increasing number of multi-defendant, multi-causation cases have been won, but a lung cancer suit will be considerably harder to unravel than that of a fire case.

Another important difference arises from the types of plaintiffs. In a fire case, three parties may often be involved: the cigarette manufacturer, the smoker, and an innocent third party. A child sleeping in the next apartment may be a victim in addition to the smoker (often drunk) who decides to have one last smoke before retiring. Both the individual who drops the cigarette that ignites the bedding and the smoker who develops lung cancer, emphysema, or heart disease that is claimed to be caused by smoking will be met with a barrage of defenses asserting their irresponsibility, culpability, negligence, assumption of risk, and misuse of the product. But an innocent third party injured in a fire started by a cigarette will have all of the benefits of product law and none of its limitations. Whereas everyone, including the smoker, may know that a cigarette dropped on a bed may start a fire, that is an irrelevant consideration given the capacity of the cigarette manufacturers to market a cigarette that will not start a fire.

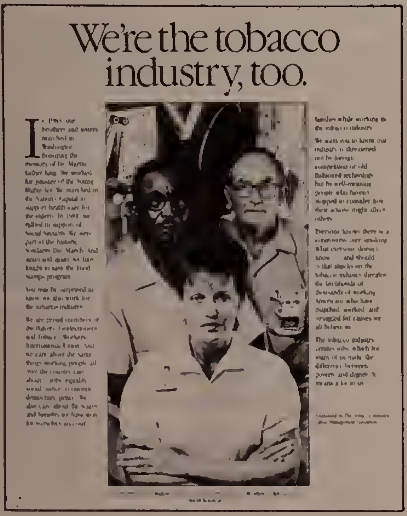
A final and critical difference between cases brought by victims of cigarette-caused fires and those brought by persons with smoking-related diseases is the magnitude of potential liability. The number of fire deaths (2,100) and injuries (9,500) each year in the United States is small compared to the 350,000 to 500,000 annual deaths attributable to cigarette smoking.[9] Cigarette manufacturers will be able to cover the damages to victims of fire, but if the initial cigarette disease cases are successful, then a flood of litigation will be provoked.

The consequence of massive cigarette litigation may prove to be similar to that which may befall the asbestos industry. When several very large judgments were awarded against Johns Manville and other asbestos manufacturers, Manville moved to preserve its substantial assets by declaring preemptive bankruptcy and by asking Congress for protection.[10] Several bills are before Congress that call for compensation to victims by the asbestos manufacturer. In addition, there has been a recent nationwide effort to reduce exposure to asbestos.

Asbestos litigation offers an example of how an independent judiciary can promote public health. Cigarette product liability litigation offers a similar hope of provoking a more sensible public policy on smoking and greater accountability by the tobacco industry in matters of public health.

REFERENCES

1. Garner D: Cigarette dependency and civil liability of cigarette manufacturers: a modest proposal. *S Cal L Rev* 1980; 53:1423–1425.
2. Garner D: Cigarettes and welfare reform. *Emory LJ* 1977; 26:269, 297–300.
3. *Green v American Tobacco,* 304 F. 2d 70 (5th Cir. 1962), *question certified on rehearing,* 154 So. 2d 169 (Fla.), *rev'd and remanded,* 325 F. 2d 673 (5th Cir. 1963), *rev'd and remanded on rehearing,* 391 F.2d 97 (5th Cir. 1968), *rev'd per curiam;* 409 F.2d 1166 (5th Cir. 1969) (en banc), *cert. denied,* 387 U.S. 911 (1970).
4. *Schenebeck v Sterling Drug, Inc;* 423 F.2d 919 (8th Cir. 1970).
5. *Grubb v Albert Einstein Medical Center,* 255 Pa. Super. Ct. 381, 387 A.2d 480 (1978).
6. McGuire A: Cigarettes and fire deaths. *NY State J Med* 1983; 83:1296–1298.
7. Rickert WS: Less hazardous cigarettes: fact or fiction? *NY State J Med* 1983; 83:1269–1272.
8. Cigarette Safety Act: Hearings on HR 1880 before the Subcommittee on Health and the Environment of the House Committee on Energy and Commerce, 98th Cong. 1st Sess. 80-85 (1983) (statement of Stuart Statler, Comm. CPSC).
9. Ravenholt RT: Addiction mortality in the United States, 1980: tobacco, alcohol, and other substances. *Population and Development Review* 1984; 10:697–724.
10. Ford SS: Who will compensate the victims of asbestos-related diseases? Manville's chapter 11 fuels the fire. *Environmental Law* 1984; 14:465–496.



We're the tobacco industry, too.

LOSS OF JOBS DUE TO *NOT* SMOKING?

In late 1984, an advertisement placed in magazines sympathetic to labor unions warned that jobs were being threatened by "attacks on the tobacco industry" by "well-meaning people who haven't stopped to consider how their actions might affect others." The ad's sponsor was listed as The Tobacco Industry Labor/Management Committee, which is funded by The Tobacco Institute—which is funded by cigarette manufacturers. Although *The Progressive* accepted the ad, its editor likened it to the buttons worn at an aerospace plant in the 1960s: "DON'T BITE THE WAR THAT FEEDS YOU." One letter-writer wondered if the next ad would be from workers who built and loaded the ovens at Belsen. Writing in *The Washington Post,* George Silver, MD, emeritus professor of public health at Yale University, pointed out that "the union is asking us, in effect, to honor their job need by sacrificing our lives."

Cigarette marketing: ethical conservatism or corporate violence?

JOE B. TYE

A free enterprise society can only flourish when business voluntarily takes measures to protect the public from risk of harm. Much of the government regulation that impinges on business freedom today is a result of corporate failure to exercise responsible behavior in the past. Despite the existence of substantial evidence linking cigarette smoking with many debilitating and often lethal diseases, cigarettes are one of the least regulated, most promoted, and most profitable products on the market.

It is reasonable to expect that when significant evidence suggests that a product is dangerous, those engaged in selling it should adhere to a standard of conservatism, whether they believe the evidence or not. They should not depart from this standard until the safety of the product has been determined. The degree of conservatism should be related to the magnitude, severity, and irreversibility of the risk. In the case of cigarettes, hundreds of millions of people around the world smoke, and thousands more start every day, so the potential risk is of great magnitude; the diseases cigarettes have been implicated in are frequently fatal, so the risk is severe; and for many smokers cigarettes create a powerful dependence, making the risk irreversible.

A proposed Standard of Ethical Conservatism for cigarette marketing includes three duties. First is the duty to warn people of potential risks they assume by initiating or continuing smoking, so they can make an informed decision. Second is the duty not to undertake promotional activities that might encourage an increase in smoking. Third is the duty not to exploit new markets in which the prevalence of smoking is not yet widespread. Given the seriousness of the diseases with which smoking has been linked, the near unanimity of the scientific and medical communities that cigarettes are causally related, and the well-known habituating nature of cigarettes, this is a modest standard of behavior to expect.

MARKETING ACTIVITIES

As evidence concerning the risks of smoking has mounted, the cigarette industry has expanded many of its activities.[1] Substantial resources have been committed to advertising and promotion, development of new markets, and political activities to fight initiatives that would restrict cigarette companies' freedom to market cigarettes.

Warning consumers. To assure that consumers can make informed decisions about using a product, manufacturers have a duty to inform them of any potential risk. Risk in this sense does not imply inevitability of harm; rather, "it describes a scenario in which possible losses are present." [2] This duty arises as soon as there is credible evidence that the risk is real, not when the last shred of doubt in the manufacturer's mind is resolved. In a recent example of ethical behavior, aspirin manufacturers have agreed to warn consumers of the potential link between aspirin and Reye's syndrome in children, even though they have challenged the validity of epidemiologic studies showing this linkage.

The tobacco industry maintains that cigarettes might in fact cause disease, but that current evidence is not definitive and more research is needed to prove or disprove a causal relationship. The industry not only has refused to voluntarily warn consumers of the risk associated with cigarette smoking, but it has effectively limited government attempts to require warnings. When legislation was finally enacted in 1965, according to Fritschler, "it was of dubious value to the public; it contained a mild health warning requirement for packages but prohibited government agencies from imposing other health rules on cigarette advertising or labeling." [3] Recently, the industry was able to remove any reference to the words "death," "miscarriage," and "addiction" from the rotating health warnings approved by Congress in 1984. Also removed was a requirement that exported cigarettes carry health warnings.

In fact, the cigarette industry still does not warn consumers of the risks associated with smoking. The warnings are attributed to the Surgeon General—not the cigarette manufacturer. This gives the industry a high degree of flexibility in its attempts to vitiate the warning's effectiveness. Through newspaper advertisements and pseudoscientific publications of its public relations arm, the Tobacco Institute, the industry has conducted major publicity campaigns to create doubt in the public mind concerning the relationship between smoking and disease. One report critical of these campaigns said that the cigarette industry "has adopted . . . a strategy [of] citing evidence selectively and quoting scientists out of context" to make it appear that there is a controversy within the scientific community when none really exists.[4]

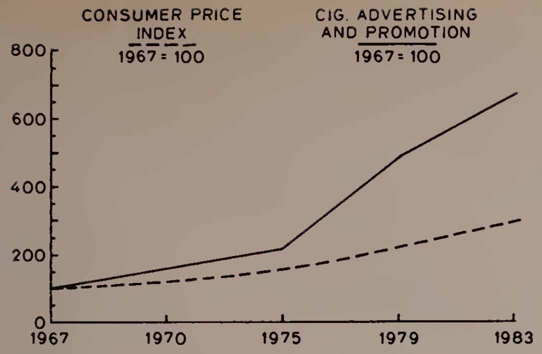
Advertising and promotion. A Standard of Ethical Conservatism would require that special efforts be made to prevent advertising and promotional activities from influencing people who might be particularly susceptible to cigarette advertising imagery. A good example of an ethical concern for the impact of advertising is reflected in Abbott Laboratories' "Code of Marketing Ethics for Developing Countries":

> We believe that unsupervised, direct promotion of infant feeding products to mothers can unjustly impel them to make decisions

---

Mr Tye worked on the administrative staff of the University of Iowa Hospitals and Clinics from 1976 to 1983 and served as Director of Planning from 1981 to 1983. In 1985 he received a Masters degree in Business Administration from the Stanford University Graduate School of Business.

Address correspondence to Mr Tye, 105C Escondido Village, Stanford, CA 94305.



US cigarette advertising and promotional expenses compared with the consumer price index (1967 to 1983). Sources: CPI data from the *Statistical Abstract of the US* for each respective year; 1967 cigarette expenditures from Miles R: *Coffin Nails and Corporate Strategy* (Englewood Cliffs; Prentice-Hall Inc); p 83; 1970–1979 cigarette expenditures from Whelan E: *A Smoking Gun* (Philadelphia, GF Stickley Co), p 178; and 1983 cigarette expenditures estimated by The Tobacco Institute.

> concerning the care and nutrition of their babies for which they may lack adequate medical or nutritional knowledge. Therefore, we do not advertise our products through general circulation magazines, directories, newspapers, radio, television, billboards, and other public mass media.[5]

From the time credible evidence linking cigarette smoking with disease began to appear, cigarette promotional activities have shown explosive growth, and cigarettes are now the most heavily advertised product in the world. As shown in the Figure, between 1970 and 1983 cigarette company advertising and promotion expenditures increased at a rate far in excess of inflation.

Of particular concern is the potential impact of cigarette advertising and promotion on young people. According to the Public Health Service, nearly one quarter of high school seniors smoke on a daily basis, and 63% of them started before the 10th grade.[6] Although the cigarette industry's official position is that no advertising or promotion should be directed at anyone under 21 years of age, many cigarette marketing activities are directed at teenagers. Cigarette company representatives give away free cigarettes to young people attending rock concerts and sponsor a variety of youth-oriented athletic and musical events.[7]

Much of the thematic content of cigarette advertising has been developed with young people in mind. It is inconceivable that cigarette marketers, who employ costly and sophisticated research techniques, develop role model images like cowboys and high fashion models—the very essence of teenage dreams—without being cognizant of the potential impact on young people. The vulnerability of teenagers to the role model imaging used to market cigarettes is acute, because many do not have a tangible understanding of the health risks. Young people tend to discount the possibility of dying of chronic diseases sometime in the distant future when compared to the perceived glamour of smoking. A recent survey showed that 40% of the nation's high school seniors do not believe that there is a great health risk associated with even heavy smoking.[6]

Some cigarette advertising is designed to neutralize evidence of the harmful nature of the product. In its 1981 report to Congress,[8] the Federal Trade Commission charged that "cigarette ads . . . attempt to allay anxieties about the hazards of smoking and to associate smoking with good health, youthful vigor, social and professional success, and other attractive ideas." The report also charged that "a number of campaigns imply that smoking a particular brand solves the health problem or at least minimizes the risk," and that others "capitalize upon the erroneous consumer perceptions that there is a health benefit to smoking mentholated cigarettes."

New markets. When large numbers of men stopped smoking as a result of health concerns, the cigarette industry began to look for potential growth markets to offset the loss. The women's market was given renewed emphasis with cigarette brands and massive advertising campaigns directed specifically at young women. As a result, during the 1970s there was an increase in the number of teenage girls who smoked.

The cigarette companies are dividing ethnic markets into ever-finer segments in order to maximize their penetration in each. One group that has recently been targeted is Hispanics. The smoking prevalence among Mexican-American women is 70% lower than among Anglo-women, indicating the potential for growth in this market.[9] Because of language and literacy factors, it is likely that many people in this market are not aware of the risks associated with smoking. Philip Morris has initiated a focused advertising campaign for Hispanics,[10] and Liggett & Myers used test marketing to develop its Dorado brand of cigarettes, aimed at the Hispanic population in the Southwest.[11]

Cigarette companies have also promoted the development of foreign markets. Between 1974 and 1979, exports of American tobacco products increased by 230%.[12] Much of this activity has been in developing countries, where the rate of cigarette smoking is expanding rapidly. The cigarette companies advertise heavily in many of these countries and do not provide health warnings unless required by local law.[13,14]

Other activities. The cigarette companies have invested heavily in the design and production of lower tar and nicotine cigarettes. Ironically, however, they insist that they have not developed these new cigarettes out of a concern for the health of their consumers, but simply in response to economic demands of the marketplace. The probable truth of this motivation is borne out by the introduction by Brown & Williamson of a new high tar/nicotine brand (Eli Cutter) with an advertising image of a 19th century outlaw even more macho than the Marlboro cowboy,[15] and the current trend toward 25-cigarette packages which, according to John Maxwell, an investment analyst who specializes in the tobacco industry, is intended to increase the daily consumption of current smokers.[16]

The tobacco industry points out that it spends a lot of money on research concerning the health implications of smoking. Between 1954 and 1983, the industry-supported Council for Tobacco Research spent $83 million on research.[17] At the current rate, however, cigarette companies spend that much on advertising and promotion every 20 days. Furthermore, there is no evidence that findings from research supported by the industry have resulted in any changes in the marketing practices of cigarette companies.

In essence, the cigarette industry has done everything possible to avoid warning consumers of the risks associated with smoking and to confuse the evidence underlying the warnings that are provided by the Surgeon General. It has developed sophisticated marketing programs, elements of which are intended to influence young people to start smoking. It has developed new markets, frequently exploiting people's ignorance of the risks associated with smoking. Many of the activities which the industry singles out as examples of its social responsibility are subterfuges to distract attention from the real purposes of its marketing activities.

CIGARETTE MARKETING AS CORPORATE VIOLENCE

The marketing practices of cigarette companies diverge from the Standard of Ethical Conservatism proposed above. As a result, many more people are now smoking cigarettes than would be had the industry adopted ethically conservative marketing practices. These people have been placed at risk of serious physical harm. Many will be unable to stop and some will suffer painful, protracted illnesses and premature death. In fact, the ultimate consequences of cigarette marketing practices are so pernicious that they constitute corporate violence, which has been defined as follows:

> Behavior producing an unreasonable risk of physical harm to consumers, employees, or other persons as a result of deliberate decision-making by corporate executives or culpable negligence on their part.[18]

Corporate violence is not a legal concept, but rather it implies violation of an ethical standard (though it may result in liability for civil damages and, in extreme cases, criminal charges). It is typically distinguished from more familiar forms of violence by the absence of malevolent intent. Corporate violence usually does not entail a deliberate conspiracy to cause injury, but results from subordination of concern for human safety to monetary considerations.

Although the cigarette industry acknowledges its own uncertainty about the safety of its product, its response to increasingly damning medical research findings has been aggressive promotion of cigarette smoking. Although an analysis of what motivates those in the cigarette industry and the advertising agencies that create its selling programs is beyond the scope of this article, a variety of motives might be postulated for the failure of executives and board directors of cigarette companies and advertising agencies to adopt ethically conservative marketing practices. It has been argued that cognitive dissonance avoidance is an important factor in the behavior of cigarette company executives. One social scientist argues that cigarette company executives avoid the psychological conflict between their perceptions of themselves as decent human beings and the possibility that they are contributing to the early deaths of a great many people by rationalizing away the evidence linking cigarettes with disease. They convince themselves that cigarettes are not harmful (partly by smoking heavily themselves)—indeed, that they might even be beneficial.[19]

There may also be cultural factors at work. For example, a former chief executive officer of Philip Morris described his company's "Masada complex," referring to the courageous stand of a small group of first century Jews against the Roman Legions, stating that the company's embattled psychology had drawn its people together and created a high degree of loyalty.[20] This would explain how a group of people can come to view "do-gooder" organizations like the American Cancer Society as "the enemy." Whatever the reasons, cigarette company executives have subordinated concern for human safety in their quest for growth and profits. Because their marketing programs are targeted at those most vulnerable to smoking's appeal and least knowledgeable about the risk, a great many people have been, and will continue to be, subjected to an unreasonable risk of harm.

BUSINESS COMMUNITY INVOLVEMENT

Although a growing number of businesses have acknowledged the health hazards of cigarette smoke by establishing smoke-free work environments and incentive programs for employees to stop smoking, overall the business community has been willing to support, and in some cases to profit from, the sale of cigarettes. The most direct collaborators in cigarette marketing have been the advertising agencies that develop cigarette company promotional campaigns, and the mass media corporations that convey these messages.

Arguments by the advertising and media industries that cigarette advertisements are not intended to build primary demand do not relieve these industries of responsibility for the likelihood that this is a foreseeable result. Likewise, their argument that the advertising of any legal product should not be restricted overlooks the fact that cigarettes are not legal because they have been proven safe, but because the tobacco industry has used its political power to obtain specific exemption from every law that might have been used to regulate them. Neither the American Association of Advertising Agencies nor the American Advertising Federation has developed ethical standards for marketing dangerous products like cigarettes or has spoken out against cigarette marketing excesses.

The business press, which is in the best position to understand and report on the marketing practices of the cigarette industry, has instead contributed to perpetuating the myth that cigarettes are just another consumer product and that the people who promote them are ethically no different than their counterparts in other industries. Cigarette companies are praised for their acumen in increasing profits each year despite the "health scare," are bestowed awards for their advertising achievements, and are recommended as vehicles for investors to increase their wealth. An extensive literature search failed to locate a single article in the business press that specifically addressed the ethics of cigarette marketing.

Corporations in other lines of business have also lent considerable support to the cigarette industry's attempt to maintain both profitability and social respectability. For example, senior officials of American companies such as IBM, Monsanto, NCR, and Citicorp have served on the boards of directors of cigarette companies. Similarly, annual reports of tobacco companies highlight not-for-profit organizations that have accepted donations. For example, in 1984, American Brands, makers of Lucky Strike, Carlton, and Pall Mall, funded a mobile advanced life support ambulance for a New York City hospital (The New York Infirmary-Beekman Downtown Hospital). Perhaps the most disturbing silence on the subject of cigarette marketing has been that of health organizations and trade associations that have both the societal responsibility and the resources to influence the business community. The American Hospital Association and the American Medical Association, which are on record as opposing smoking, have not played a major role in efforts to counteract cigarette advertising.

The response of the nation's business schools to the issue of cigarette marketing ethics has been disappointing. Most textbooks on business ethics do not even mention cigarette marketing, although it is one of the most serious and controversial ethical problems in business today. Although most teaching at business schools is based on the case method, there are not case studies that deal specifically with the ethics of marketing cigarettes.

In addition to neglecting the problem, some business schools have actively supported the cigarette industry. The Wharton School of Business,* for example, completed a study for the tobacco industry on the economic contributions of tobacco to the American economy.[21] This study has been used by the cigarette industry to further its political aims. More a marketing tool than a rigorous academic analysis, the Wharton study does not consider the societal costs of cigarette smoking—costs that have been well documented. When more stringent health labeling requirements were being considered by Congress, several marketing professors from Wharton and Ohio State University business schools testified in behalf of the Tobacco Institute that marketing techniques—the very strategies they teach—would be ineffective in raising consumer awareness concerning the health hazards of smoking.

PROTECTING THE PUBLIC

When corporations refuse to act in a socially responsible manner, and informal pressure is ineffective, government bears a responsibility to protect the public from the consequences. The primary reaction of the US government to the marketing excesses of the cigarette industry has been to require health warnings. Unfortunately, Congress succumbed to industry influence and expurgated from these warnings reference to important elements of risk, and eliminated warning requirements altogether for people in other nations whose ignorance of smoking's hazards places them at particularly high risk. The government has also allowed the cigarette industry great freedom to undermine the effectiveness of warnings that are provided.

The cigarette industry has not shown a willingness to adhere to a Standard of Ethical Conservatism, and government has been unwilling or unable to impose one. Fortunately, society provides another mechanism—the judicial system—for those who are injured as a result of corporate violence to seek compensation and to impose incentives on corporations to behave in an ethical manner. The courts should recognize that the pattern of corporate violence in current cigarette marketing has inflicted an unreasonable risk of harm on many people who were not informed of the addictive and harmful nature of cigarettes or were influenced by the implicit health assurances present in most cigarette advertising. It may be that the most effective way to induce cigarette companies and their advertising agencies to behave in an ethically conservative manner is by making them compensate people who have been harmed by the industries' failure to adopt socially responsible marketing practices.

REFERENCES

1. Miles R: *Coffin Nails and Corporate Strategies.* Englewood Cliffs, NJ, Prentice-Hall, 1982.
2. Moore P: *The Business of Risk.* New York, Cambridge University Press, 1983:2.
3. Fritschler A: *Smoking and Politics.* Englewood Cliffs, NJ, Prentice-Hall, 1983:38.
4. Ernster V, Burns D: A rebuttal to the tobacco industry's document "cigarette smoke and the nonsmoker." *J Pub Health Policy* 1984; 5:368–375.
5. Post J: *Nestle Boycott* (A), Graduate School of Business, Stanford University, 1981:32.
6. Johnston L, O'Malley P, Bachman J: *Highlights from Drugs and American High School Students, 1975–1983.* National Institute on Drug Abuse, 1984:59.
7. ABC News 20/20: "Growing Up in Smoke," transcript from broadcast, October 20, 1983.
8. *Staff Report on the Cigarette Advertising Investigation.* Federal Trade Commission, May 1981:2–8.
9. Holck S, Warren C, Rochat R, Smith J: Lung cancer mortality and smoking habits: Mexican-American women. *Am J Pub Health* 1982; 72:38–42.
10. Rangel J: Ads for Hispanic consumers. *New York Times,* September 3, 1984:19.
11. Revamped tobacco firm targets new consumer segments. *Marketing News,* July 6, 1984:8.
12. Heubner A: Tobacco's lucrative Third World invasion. *Business and Society Review* 1980; 35:49–53.
13. Taylor P: *The Smoke Ring.* New York, Pantheon Books, 1984:248.
14. Muller M: *Tobacco and the Third World: Tomorrow's Epidemic?,* London, War on Want, 1978:54.
15. New B & W brand debuts wild west character. *Advertising Age,* November 8, 1984.
16. Gloede W: Philip Morris gives Marlboro 25s more country. *Advertising Age,* December 24, 1984.
17. *Report of the Council for Tobacco Research—USA,* New York, 1982:5.
18. Monahan J, Novaco J, Geis G: Corporate violence: research strategies for community psychology, in Adelson D, Sarbin T (eds): *Challenges for the Criminal Justice System,* 1979. Cited in Clinard M, Yeager P, *Corporate Crime.* New York, The Free Press, 1980:9.
19. Aronson E: *The Social Animal.* San Francisco, WH Freeman & Co, 1980:102–107.
20. Gibson P: The George Weissman road show. *Forbes* Nov 10, 1980:179–188.
21. *A Study of the U.S. Tobacco Industry's Economic Contribution to the Nation, Its Fifty States, and the District of Columbia.* Philadelphia, Applied Research Center, The Wharton School, University of Pennsylvania, 1979.

---

* In 1980 the National Association of Tobacco Distributors (NATD) raised $1 million to establish an endowed chair of social responsibility in business at the Wharton School of the University of Pennsylvania. The chair was named for a longtime managing director of the NATD, Joeseph Kolodny, in tribute to his "services to the association, the trade, the industry and the field of humanitarian endeavors" (*US Tobacco and Candy Journal,* July 22, 1982). Among the major contributors to the endowment were the six major American cigarette manufacturers, Consolidated Cigar Co, US Tobacco Co, Hershey Chocolate Co, M&M/Mars, Wm Wrigley Jr Co, Life-Savers, and Warner-Lambert Co. —*Editor*

Diversification in the tobacco industry

The following tables represent a sequel to the organizational profiles of the six American cigarette manufacturers published in the December 1983 issue of the *New York State Journal of Medicine* (83; 1324–1332) and updated in *The Cigarette Underworld* (Secaucus, NJ, Lyle Stuart, 1985). The earlier tables listed the subsidiaries, US cigarette brands, advertising themes, advertising agencies, and sponsorships and promotions. Also noted were figures on overall advertising expenditures.

Cigarettes are by far the most advertised product in society. More than $2.7 billion is spent each year in the US by the six tobacco companies to promote cigarette smoking. In addition to maintaining or increasing overall cigarette sales, advertising creates social acceptability for the product and helps reinforce complacency of those who do not smoke (or do not yet smoke) and who may not be fully aware of the negative economic and health impact of smoking.

In spite of the belief that there is a powerful "anti-smoking" effort, the fact remains that cigarette manufacturers control virtually all of the contemporary imagery and terminology of smoking and continue to be successful in staving off major legislative measures that will adversely affect profits.

Some health professionals believe that criticism of tobacco companies for promoting cigarette smoking should be tempered because they have become conglomerates that are diversifying into non-tobacco products and services. By encouraging such diversification, it is reasoned, health professionals can help expedite the phasing out of smoking while tobacco companies can have an opportunity to replace the resultant lost revenue.

Such a belief on the part of individuals working to eliminate smoking may be misguided. In fact, the percentage of total profit accounted for by tobacco sales is still the highest of all sources of revenue for tobacco companies. The figures for all but the two lowest ranking cigarette makers (American Brands and Grand Metropolitan USA) are especially striking. Whereas tobacco sales account for 70.9% of the net sales of Philip Morris, tobacco profits account for 93.8% of the company's total profits. For Loews, whose tobacco sales are just 25.5% of total sales, cigarette profits account for 64.4% of the company's total profits. (Table I)

Thus it is wrong to conclude that in the forseeable future the diversification strategy of tobacco companies includes decreasing the investment in cigarette manufacturing. Indeed, RJ Reynolds whose tobacco profits account for 79.5% of total profits (versus sales of 59.2% of total sales) has opened a $1 billion cigarette manufacturing facility in Tobaccoville, North Carolina. Philip Morris has similarly expanded its facilities.

Although cigarette sales in the United States have flattened in recent years, they are not declining substantially, and profits have risen to record levels. The US still has the highest per capita cigarette consumption, and sales are more than double what they were 30 years ago at the time of publication of the first studies implicating cigarette smoking as a major cause of lung cancer. Even were domestic cigarette profits to slip, they would be more than made up for by the dramatic growth in cigarette sales in developing nations, where tobacco companies have been welcomed by numerous governments.

Perhaps the most important strategic aspect of diversification is one that is seldom discussed: geographic expansion. This tactic has been best applied by defense industry contractors, which have established weapons-related research and manufacturing facilities in numerous cities throughout the US, effectively integrating into the local economy. Congressional opponents of an arms build-up are hard-pressed to vote for cuts in defense spending for fear of causing loss of jobs in their communities.

A trend in the tobacco industry is to acquire companies in non-tobacco-growing states (and countries) that manufacture popular consumer products such as soda and candy. Executives of these seemingly disinterested and unrelated companies then take the lead in the local business community in opposing legislative restrictions on public smoking or tobacco advertising, which could reduce cigarette sales. This occurred in San Francisco with RJ Reynolds' Del Monte subsidiary.

In Table II that follows is an attempt to illustrate the diversity of holdings of the tobacco industry. Presented on a state by state basis, the table provides an opportunity to identify the tobacco interest that may help explain the success of the tobacco lobby in such seemingly non-tobacco states as New York.

However, this table represents only the tip of the iceberg of the network of supporting industries and services connected to the tobacco industry. These include thousands of farmers, tobacco distributors, food stores, drug stores, shipping companies and port authorities that are involved in the transport of tobacco (eg, Maersk Line, NY; Sea-Land, NJ; American President Lines, VA, GA; United States Lines, NJ), tobacco flavoring manufacturers (eg, International Flavors and Fragrances, NY; Bell Flavors and Fragrances, IL), banks and transfer agents (eg, Morgan Guaranty Trust, Manufacturers Hanover Trust Co, NY), chemical companies that manufacture herbicides and pesticides (eg, CIBA-Geigy, NJ, NC), cigarette lighter manufacturers (eg, BIC, CT; Ronson, NJ; Scripto, GA), paper and container manufacturers (eg, Kimberly-Clark, PA; Union Camp, VA; International Paper, NY), and cellophane makers (eg, Mobil). Nor do the tables include American holdings of foreign tobacco companies such as Imperial Group, which owns Howard Johnson's and Peoples Drugs.

Nor can the tables include another important aspect of diversification: the presence of public figures and leaders of other unrelated corporations on the boards of directors of tobacco companies. The board of RJ Reynolds includes Vernon E. Jordan, Jr, former director of the National Urban League and Juanita M. Kreps, former Secretary of Commerce in the Carter administration. A Former Secretary of Defense in the same administration, Harold Brown, sits on the board of Philip Morris along with executives of Citibank and Bankers Trust Company. Former Vice-President Walter Mondale has joined the board of CNA Financial, division of Loews.

Also not included in the table are the numerous cultural organizations funded in part by tobacco companies (and co-sponsors of tobacco company-controlled sporting events) as part of the strategy for creating social acceptability for smoking. A partial list of such sponsorships is included in the organization profiles published in the December 1983 issue of the *Journal*, as well as in other articles in this issue.

Although on the whole diversification by the tobacco industry (which began around the time of the early scientific reports indicting smoking as a cause of death) has been profitable, the proportion of profit derived from the sale of tobacco has always been and remains the foundation of these companies. Even in instances in which diversification has not been profitable—as in the case of Philip Morris' Miller Brewing Co and Seven-Up—the high visibility and large advertising budgets on television (where cigarette ads are banned) for products made by these companies has helped to insulate tobacco profits by buying immunity from journalistic scrutiny into the practices of cigarette advertisers. In this regard, the significance of RJ Reynolds' announced purchase of Nabisco Brands Inc for $5 billion is that Reynolds will become the leading national advertiser—spending $1 billion a year in the mass media. An analysis of the mass media will be the subject of a future table. The following tables were based on research by Michael Wroblewski, a student at Adelphi University.

—Alan Blum, MD

TABLE I. Financial Review of the American Tobacco Industry, 1984 (source: annual corporate reports)

	American Brands, Inc	BATUS, INC	Grand Met USA	Loews Corp	Philip Morris, Inc	RJ Reynolds Industries, Inc	United States Tobacco Company
Employees	74,000	61,800	10,408	29,000	68,000	97,551	3,476
Cigarette Market Share[1] (approximate)	8.1%	11.2%	5.9%	8.4%	35.2%	31.3%	—
Net Sales (in millions)	$6,995.2	$6,211.1	—	$5,603.3	$13,813.7	$12,974.0	$443.8
Tobacco Sales (in millions)	$4,229.0	$2,264.1	—	$1,430.8	$9,801.7	$7,685.0	$380.2
% Tobacco Sales of Net Sales	60.4	37	—	25.5	70.9	59.2	85.6
Total Profit* (in millions)	$892.2	$729.2	$181.4	$437.2	$2,280.2	$1,619.0	$172.1
Net Profit	$414.1	$313.5	—	$328.6	$888.5	$843.0	$83.7
Tobacco Profit (in millions)	$496.7	$427.8	$96.3	$281.9	$2,140.8	$1,288.0	$168.6
% Tobacco Profit of Total Profit	55.7	59	53	64.4	93.8	79.5	98

* Total profit is defined as operating income—net sales less operating expenses. It does not include income after the payment of interest or taxes and other non-operating expenses.

[1] Source: *US News and World Report* Oct 22, 1984: 49.

TABLE II. Tobacco Company Holdings in the United States

State	American Brands, Inc	BATUS, Inc	Grand Metropolitan USA	Loews Corporation	Philip Morris, Inc	RJ Reynolds Industries, Inc	United States Tobacco Company
Alabama			Diversified Products (fitness products); Quality Care, Inc (home health services)			RJR Archer-Filmco (plastic film wrap)*	
Alaska			Nine permanent pipeline pump-station accommodation camps			*	
Arizona		Breuners (home furnishing stores); Saks Fifth Avenue (department stores) (SFA)	Quality Care	Loews Paradise Valley Resort; Loews Ventana Canyon Resort	†	*	
Arkansas			Quality Care			Del Monte-Morton Frozen Foods (processing plant)*	
California	Bell Brand Foods, Inc; Taylor Food Products, Inc	Breuners; SFA	Children's World, Inc (157 child care centers in 10 states); Diversified Products (manufacturing facility); Inter-Continental Hotel, San Diego and San Francisco; Pepsi-Cola San Joaquin Bottling Co; Express Foods (cheese plant); Quality Care		Aliso Viejo Co; Jack G. Raub Co; Miller brewery; Mission Viejo Co; MVC Escrow Corp; MVC Financial Corp; Mission Viejo Realty Group, Inc; Ventura Coastal Corp; Warner-Jenkison Land Co	Aminoil (operations offices, oil and gas facilities)*[1]; Beaulieu Vineyard; Del Monte Corp (and research facility); Del Monte Frozen Foods, Inc; Heublein Spirits (processing facility); Heublein Wines; Inglenook Vineyards; International Grocery Products; US Dry Grocery and Beverage Products	
Colorado			Children's World; Quality Care		Highlands Ranch Real Estate Development	Aminoil (operations office)[1]*	
Connecticut		SFA; Gimbels (department stores)	Quality Care		†	Del Monte, Specialty Grocery Products; Heublein Spirits & Wine Co; Heublein Spirits (processing facility); International Vintage Wines*	*Corporate headquarters*; Babco Associates, Ltd; Honduran Cigar Imports, Ltd; United States Tobacco International, Inc

Delaware					State of incorporation for 15 US and overseas subsidiaries[a]		
Florida	Pinkerton's (private security)	SFA; Ivey's (department stores)	Quality Care	Loews Theatres	Miller distributor and warehouse; Southern Gold Citrus Products, Inc	Del Monte Banana Co; Del Monte World Wide Fresh Fruit operating unit*	
Georgia		Brown & Williamson Tobacco (tobacco facility); SFA	Atlantic Soft Drink Co (bottlers and distributors); Express Foods (whey protein processing plant); Quality Care		Miller brewery and can-making facility	Del Monte Soft Drink Unit Headquarters (Canada Dry, Sunkist, Hawaiian Punch)*	
Hawaii						Del Monte farmland*	
Idaho			Quality Care			Boise Truck & Equipment Inc*	
Illinois	James B. Beam Distilling Co; The Franklin Life Insurance Co; Wilson Jones Co (office products)	Marshall Field's (department stores); SFA; Thimbles (fashion specialty stores)	Alpo Petfoods, Inc (processing facility); Children's World; Quality Care	CNA Financial Corp		Skolniks Bagel Bakeries*	Tobacco facility
Indiana			Quality Care	Loews Theatres	Koch Label Co (beer labels)	*	
Iowa			Quality Care		†	*	
Kansas		SFA	Quality Care			*	
Kentucky	American Tobacco Co (tobacco facility); James Beam distillery	*Corporate headquarters*; Brown & Williamson Tobacco Co	Express Foods Group USA, Inc (dairy-based food ingredients); Atlantic Soft Drink Co; The Pinkerton Tobacco Co (chewing tobacco); Quality Care	Lorillard (tobacco facility)	Tobacco facilities (two)	Kentucky Fried Chicken (and training center; approximately 4,500 US stores, of which about 1,000 are company owned); Zantigo Mexican Restaurants; Tobacco facility*	Tobacco facility
Louisiana		SFA	Inter-Continental Hotel, New Orleans; Quality Care		Miller distributor and warehouse	Aminoil (operations office, natural gas processing plant)[1]*	

Maine			Quality Care			*	
Maryland		SFA	Children's World; Quality Care			Del Monte, Specialty Grocery Products (processing plant)*	
Massachusetts	Acushnet Co (golf equipment)	SFA	Inter-Continental Hotel, Boston; Quality Care		†	*	
Michigan	Sugar Beet Products Co	SFA; Thimbles	Children's World; Quality Care		Plainwell Paper Co, Inc	Heublein Spirits (processing plant)*	WPBN-TV and WTOM-TV, Inc
Minnesota		Thimbles	Alpo Petfoods, Inc (processing facility); Children's World; Quality Care			Zantigo Mexican Restaurants*	
Mississippi			Alpo Petfoods, Inc (processing facility); Quality Care			*	
Missouri		SFA	Quality Care		The Seven-Up Co; Taylor Group, Inc	Zantigo Mexican Restaurants*	
Montana						Aminoil (land interests)[1]*	
Nebraska			Alpo Petfoods, Inc (processing facility)			*	
Nevada		Breuners; SFA	Quality Care			Aminoil (land-geothermal leases)[1]*	
New Hampshire						*	
New Jersey	Corporate office	Gimbels; SFA; Thimbles	*Corporate headquarters*; Scanticon Corp (video conferencing); Quality Care	Loews Glenpointe Hotel; Loews Theatres	■	Del Monte (frozen food plant); Sea-Land Corp (container shipping)[2]*	
New Mexico			Alpo Petfoods, Inc (processing facility); Quality Care		†	Aminoil (land-geothermal leases)[1]*	
New York	*Corporate headquarters*; American Cigar; American Tobacco Co; MCM Products, Inc (cutlery, light bulbs, auto products); Pinkerton's, Inc (private security service); Sunshine Biscuits, Inc; Swingline, Inc (office products)	Batus Retail Group; Gimbels; SFA	Carillon Importers (liquor); Intercontinental Hotels Corp; Inter-Continental Hotel, NYC; Quality Care; The Paddington Corp (liquor)	*Corporate headquarters*; Loews Hotels; Loews Theatres; Loews Representation International, Inc (reservation service for about 300 hotels); Lorillard Tobacco Co; Bulova Watch Co	*Corporate headquarters*; Miller brewery and glass-making plant (Fulton and Sennett)	Canada Dry Corp[3]; Del Monte Franchise Beverage Products; Service Systems Corp (restaurant and vending machine services)[4] *	

North Carolina	Golden Belt Manufacturing Co (printing and paper-foil lamination for product packaging)	Brown & Williamson tobacco facility and export leaf processing facility; Ivey's	Atlantic Soft Drink Co; Liggett & Myers Tobacco Co and facilities; Quality Care	Lorillard (tobacco facility)	Miller brewery and can-making facility; tobacco facility	*Corporate headquarters*; RJR Archer, Inc (packaging); RJ Reynolds Development Corp; RJ Reynolds Tobacco Co and tobacco facilities*	Henry, Leonard & Thomas, Inc; Mastercraft Pipes, Inc
North Dakota						Aminoil (operations office and natural gas processing plant)[1]*	
Ohio	The Andrew Jergens Co (body lotions)	Appleton Papers; SFA	Children's World; Quality Care	Loews Theatres	Miller brewery (not in use)	RJR Archer-Combibloc, Inc (aseptic packaging); Filmco (plastic film wrap);* Skolniks; Zantigo	
Oklahoma			Quality Care			Aminoil (operations office and natural gas processing plant)[1]; Aminoil Marketing, Inc[1]*	
Oregon		Frederick & Nelson (department stores)	Quality Care		Miller distributor and warehouse; Oregon Freeze Dry Foods, Inc	Bear Creek Corp (retail mail order of fruit and garden plants; parent company of Harry & David, and Jackson & Perkins Co); Aminoil (land-geothermal leases)[1]*	
Pennsylvania	Case WR Sons Cutlery Co	Appleton Papers, Inc (pulp and paper mill and converting plant)	Alpo Petfoods, Inc; Quality Care			Skolniks Bagel Bakeries*	House of Windsor, Inc
Rhode Island			Quality Care			*	
South Carolina		Ivey's	Atlantic Soft Drink Co; Inter-Continental Hotel, Hilton Head Island; Quality Care			*	
South Dakota						*	
Tennessee			Atlantic Soft Drink Co; Quality Care	CNA (administrative facility)		*	National Pen & Pencil Co; tobacco facilities (three)

Texas	Southland Life Insurance Co; Perma Products Co, Inc	Marshall Field's; SFA	St Anthony Inter-Continental Hotel, San Antonio; Inter-Continental Hotel, Houston; oil and gas exploration; Quality Care	Loews Anatole Hotel, Dallas; Loews Theatres	Miller brewery and can-making facility[†]	American Independent Oil Co[1]; Aminoil International, Inc[1]; Aminoil (operation offices and natural gas processing plant)[1]; Aminoil USA, Inc[1]; Skolniks*	
Utah					Miller distributor and warehouse	Aminoil (land-geothermal leases)*	
Vermont			Express Foods (whey protein processing plant)			*	
Virginia	American Tobacco Co (tobacco facility)	Brown & Williamson Tobacco Co (tobacco facility); Thimbles	Children's World; Quality Care	Lorillard (tobacco facility)	Tobacco facilities (seven); engineering center; research facility[†]	Del Monte-Morton Frozen Foods (processing plant)*	Tobacco facility
Washington		The Crescent (department stores); Frederick & Nelson	Children's World; Quality Care		Miller distibutor and warehouse	*	Ste. Michelle Vinters Inc
West Virginia			Quality Care			*	
Wisconsin	Master Lock Co	Appleton Papers, Inc (pulp and paper mill); Gimbels; Thimbles	Quality Care		Miller Brewing Co (brewery and can-making facility); Waterloo Malting Co, Inc; Nicolet Paper Co; Wisconsin Tissue Mills	Del Monte, Specialty Grocery Products (processing plant)*	
Wyoming						Aminoil (oil production and land)[1]*	
Washington, DC			Willard Hotel	L'Enfant Plaza Hotel		Dental Care of America; Skolniks*	

---

[a] Continental Equity Investments, Inc; Philip Morris Asia Inc; PMCC Leasing Corp; Philip Morris Credit Corp; Philip Morris Europe SA; Philip Morris Export Corp; Philip Morris Industrial Inc; Philip Morris International Finance Corp; Philip Morris Limited; Philip Morris Marketing SA; Philip Morris Overseas, Inc; Philip Morris Services, Inc.; Seven-Up International, Inc.; The Seven-Up Co; Wisconsin Tissue Mills, Inc. Divisions of other tobacco companies are also incorporated in Delaware.

[1] Sold in October 1984.

[2] Sold in June 1984.

[3] Moved to Georgia in 1985.

[4] Sold in January 1985.

* Indicates Kentucky Fried Chicken retail outlets.

† Indicates site of company owned bottlers of Seven-Up.

■ In May 1985, purchase of Nabisco Brands, Inc, Parsippany, NJ, for $5 billion announced by RJ Reynolds Industries, Inc. Nabisco has 69,700 employees and in 1984 had a net income of $309 million on revenues of $6.25 billion. The company makes more than 300 food products, including Almost Home Cookies, Barnums Animal Crackers, Chips Ahoy!, Fig Newtons, Lorna Doone, Mallomars, Oreo, Graham Crackers, PREMIUM Saltines, RITZ crackers, Uneeda Biscuit, Mister Salty Pretzels, Cream of Wheat, Dromedary Dates, Milk-Bone Dog Biscuits, Shredded Wheat, Baby Ruth, Chuckles, Sugar Daddy, Life-Savers, Beech-Nut Gum, TUMS, Pine Brothers Cough Drops, Fleischmann's Margarine, and Planter's Peanuts. Reynolds will become the largest consumer products company and by far the top national advertiser in the US — spending more than $1 billion a year in advertising.

Mixed messages for women

A social history of cigarette smoking and advertising

VIRGINIA L. ERNSTER, PHD

> *They want to make you think that cigarettes will make you beautiful but really they just want to make money. Those ads are dumb because cigarettes make you die.*
>
> —7-year-old girl looking at mother's magazine

Messages promoting cigarette smoking are everywhere—in advertisements in the printed media, on billboards, on public transit, and in routine encounters with individuals who smoke. These appeals are countered by warnings of public health officials, health charities, school programs, and no-smoking signs. As the young girl's observation reveals, cigarette smoking has been fraught with mixed messages.

Over the years, the content and magnitude of the enticements and the warnings have changed, as has the social symbolism of cigarette smoking. American women began to smoke in large numbers two to three decades after American men, juxtaposing the sexes in different historical relation to emerging medical data on the health hazards of smoking. Cigarette smoking was initially a symbol of emancipation for women, and it has since become associated with self-destruction.

THE EARLY 1900s: SMOKING IS AVANT GARDE

At the turn of the century, cigarette smoking was socially unacceptable for women, but was gaining a foothold with American men, who still showed a preference for cigars. The cigarette had long been deemed a feminine object compared to the cigar. In the mid-19th century, it was considered poor taste for gentlemen to smoke in public during hours when women might be encountered, and at the end of the century, women could not join their male companions in the smoking room after dinner, even in private gatherings.[1] It was written of the period, "Between the lips of a woman [the cigarette] was generally regarded as no less than the badge of questionable character." [2]

Lucy Page Gaston, founder of the Chicago Anti-Cigarette League at the turn of the century, claimed that young boys who smoked developed "cigarette face," a condition that eventually led to "drink, crime and dreadful death." [3] She also decried smoking by women on the grounds that it undermined family values and the moral fabric of society. Gaston's efforts resulted in the enactment of local and regional laws prohibiting smoking.

Smoking by women in public places met considerable opposition. In 1904, a policeman in New York City arrested a woman for smoking a cigarette in an automobile, with the admonition, "You can't do that on Fifth Avenue." [4] Smoking by female schoolteachers was considered grounds for dismissal.[3] In 1906, in *Cigarettes in Fact and Fancy,* Bain observed that "American girl stenographers clandestinely smoked Egyptian cigarettes." [5] A headline in the New York *Herald* in 1908 read, "Women smoke on way to opera: are discovered puffing cigarettes when electric light beams into their carriage." In 1910, Alice Longworth, President Roosevelt's daughter, was scolded for smoking in the White House and retorted she would smoke on the roof.[6]* The potential for widespread adoption of cigarette smoking by women occasioned public alarm. New York's Sullivan Ordinance of 1908 made it unlawful for women to smoke in public, but the ban was largely ignored.[7]

Given the social climate of opposition to smoking by women, advertisers refrained from copy that suggested an appeal to women. Not until 1919 did a tobacco company (Lorillard) sponsor a series of advertisements for brands such as Murad and Helman in magazines and newspapers showing images of women.[3]

There are no reliable estimates of the breakdown of cigarette consumption figures by sex during the first two decades of the century. It is believed that the majority of cigarette smokers were men, and that women who smoked did so much less than men. World War I is credited with changing the social climate for cigarette smoking. American tobacco companies (often aided by charities) supplied cartons of cigarettes to soldiers abroad, converting many young men to the smoking habit, while at home women began venturing to smoke in public.

THE 1920s: "EMANCIPATION"

In 1929, Barnard commented on smoking prevalence:

> Women war workers took up the habit abroad and women at home in their men's jobs and new-found independence did likewise. Within the next three or four years cigarette smoking became the universal fashion, at least in cities, and children born since the war take smoking mothers for granted.[2]

In 1923, 5% of all cigarettes were consumed by women,

Address correspondence to Dr Ernster, Associate Professor, Department of Epidemiology and International Health, School of Medicine, University of California at San Francisco, San Francisco, CA 94143.

* *She would later appear in an advertisement for Lucky Strikes.* —*Editor*

increasing to 12% by 1929. (These figures may underestimate the proportion of women who smoked. Women on average smoked fewer cigarettes per capita than men; Moody reports daily consumption of 2.4 cigarettes by women compared to 7.2 by men who smoked in 1929.[8])

Once associated with indecent women or the ultrarich, cigarette smoking made inroads among social trendsetters in the 1920s. Fass[9] documents the symbolic importance of cigarette smoking to the crumbling of the double standard and emerging equality of female college students. In the early 1920s, smoking by women was banned on most campuses. Most college newspapers, reflecting the view of students but not of administrators, took positions in favor of smoking by women students. The issue became a cause célèbre in 1925, when the president of Bryn Mawr permitted smoking rooms on campus. But smoking by women students was still grounds for dismissal at many institutions. The University of Southern California refused registration to women students who smoked. In 1927, women at Stanford were permitted to smoke and the *Chronicle* of Duke University carried an advertisement for Old Gold, featuring two young female smokers. Fass states, "Smoking was perhaps the one most potent symbol of young woman's testing of the elbow room provided by her new sense of freedom and equality." [9]

Female students were the vanguard, and their behavior did not necessarily reflect smoking attitudes and patterns among the general population. Schudson[10] examined media reports of smoking-related activities in colleges and in public facilities such as railroads, restaurants, and art galleries. Views of female smoking ranged from condemnation to acceptance. In New York City, cigarette accessories could now be bought at jewelry stores, and one tobacco shop catered exclusively to women customers.

Despite the growing number of women smokers, cigarette manufacturers were concerned about a prohibitionist backlash, and they refrained from promoting their product directly to women. In a 1926 article, "Why cigarette makers don't advertise to women," Bonner stated, "The cigarette people are frankly afraid of stirring up the reformers and bringing down upon themselves a lot of nuisance legislation." [11] The article cited evidence of "indirect" appeals to women, including the slogan "Mild as May" for the recently introduced Marlboro brand, and the billboard advertising Chesterfield cigarettes that featured a young woman and her smoking male companion on a moonlit night. The woman in the Chesterfield advertisement appeared to be enjoying her escort's smoke; the caption said, "Blow some my way." The article predicted that public opinion would soon be on the side of the tobacco industry, and that within a year or two direct advertising appeals to women would be appearing on billboards, and in magazines and newspapers.

In 1927, Williamson discussed with obvious disdain "the firm-rooted belief in the reactionary mind that women—decent, respectable women—do not smoke." The author stated, "There can be but little doubt of the way the wind is beginning to blow, and with such a market awaiting the manufacturer we may expect almost any day to see him right after it." [12]

Two months later, an article titled "Marlboro makes a direct appeal" describes Marlboro's advertising campaign portraying a woman smoking on the back cover of *Le Bon Ton*, a women's fashion and travel magazine with a sophisticated readership. Shortly thereafter, a series of single column advertisements appeared in magazines and newspapers, showing a feminine hand in silhouette holding a lit cigarette with the "Mild as May" theme. By April of 1927, direct appeal insertions appeared in leading general and women's magazines. The copy suggested the social desirability of Marlboro: "Women—when they smoke at all—quickly develop discriminating taste. . . . That is why Marlboros now ride in so many limosines, attend so many bridge parties, repose in so many hand bags." In what may be the first promotional activity for cigarettes directed at women, these advertisements included an offer to receive free, upon request, the new Marlboro bridge score. A related development was the decision of *Pictorial Review* (which, like other mass circulation women's magazines of the day, had refused tobacco advertising) to accept such advertisements beginning with the May 1927 issue.[13]

Other signs of the changing times included opera star Ernestine Schumann-Heink's endorsement of cigarettes—although ultimately she came out against smoking after antitobacco crusaders succeeded in convincing some recital halls to cancel her appearances. Testimonials from film actresses and other female public personalities, including Amelia Earhart, appeared in cigarette advertisements. In the late 1920s, models, and later prominent debutantes, were hired by Edward Bernays, public relations manager for Lucky Strikes, to appear smoking in public in an effort to attract media attention. Bernays managed to commandeer the fashion industry to make green—the color of the Lucky Strike package—the color of fashion one year, hoping that coordinating Lucky Strikes with women's clothing would increase sales.[3]

The most renowned advertising campaign of the period directed at women was the association of cigarette smoking with staying slim, launched in 1928 with advertisements carrying the slogan "Reach for a Lucky Instead of a Sweet." The campaign brought on much hue and cry, especially from the candy industry, but to this day is considered one of the great successes in advertising history.[3]

Despite the many indications of a transition in cultural values, traditional views linking female smoking with immorality persisted in large segments of the population. Senator Reed Smoot, on June 10, 1929, introduced an unsuccessful bill in Congress to extend to tobacco the provisions of the Pure Food and Drugs Act of 1906. His remarks on the Senate floor reveal that the initial reluctance of the tobacco manufacturers to promote their product to women showed an awareness of the still prevalent sentiment:

> Not since the days when public opinion rose up in its might and smote the dangerous drug traffic, not since the days when the vendor of harmful nostrums was swept from our streets, has the country witnessed such an orgy of buncombe, quackery, and downright falsehood and fraud as now marks the current campaign promoted by certain cigaret [sic] manufacturers to create a vast woman and child market for the use of their product.[14]

THE 1930s: SMOKING IS IN VOGUE

Rudy Vallee composed his song "My Cigarette Lady" in 1931.[15] Within a few years, First Lady Eleanor Roosevelt was smoking in public.[16] An analysis of 40 motion pictures published in 1935 found that 30% of heroines smoked in the films studied compared to only 2.5% of villainesses.[17] (The comparable figures were 65% for heroes and 22.5% for villains.) That male and female "good guys" were portrayed as smokers suggests that cigarette smoking, by the 1930s, had achieved a high level of social acceptability. But the percentage of women in the general population who smoked was still less than that of the motion picture heroines. The Fortune Survey of 1935, quoted in the absence of national data, found that 52.5% of men and 18.1% of women reported themselves to be cigarette smokers; these figures varied with the respondent's age and place of residence.[18] Women under 40 years of age were more likely to say they smoked cigarettes than women over 40 (26.2% versus 9.3%). Women's smoking was most common in cities with populations between 100,000 and one million people (40.2%) and least common in rural areas (8.6%). An estimated minimum of 14% of all cigarettes was consumed by women in 1931.[8] An economic analysis of trends in tobacco production published in 1936 credited World War I and adoption of smoking by women with the "virtual doubling of the demand since 1920." [19] One article hinted that pipe manufacturers might try luring the female market, given the success of the cigarette in attracting female customers.[20]

Cigarette advertisements began to appear in major middle-class women's magazines such as *McCall's, Ladies Home Journal,* and *Better Homes and Gardens.* These advertisements were now directly pitched toward women. In general, advertisements of the period featured testimonials from American women socialites (a series for Chesterfields), celebrities (including opera star Helen Jepson for Lucky Strikes), elegant settings (during the Great Depression), athletic-looking women, and women whose smoking made them sexually attractive, judging from the number of male admirers depicted in the advertisements.

Observing that handbags and compacts were now designed for holding cigarettes and women were no longer concerned about smoke and ashes on their furnishings, Gottsegen commented in 1940 on the cultural context of unisex behavior:

> Cigarettes have become an item of consumption by women and men during the same era when women's dress is approximating that of men in type of cloth, color, design, and silhouette, and when many items, formerly restricted to men's use, are now being purchased for women's consumption.[21]

Cigarette brand preferences of male and female smokers who responded to the 1938 Fortune Survey were remarkably similar.[22] An attempt by Winston Cigarette Company to market a cigarette called Fems, unique for its red mouthpiece that would not show lipstick, was unsuccessful.[3] But a survey of 500 persons in four cities found that 81% of the women interviewed still did not approve of women smoking in public.[23]

1940s–1960s: SMOKING IS THE MIDDLE CLASS

By World War II, one third of American women smoked cigarettes.[24] During the war, the advertising campaigns of cigarette companies managed to link smoking with patriotism. Free packs were delivered to the armed forces, and in magazines women who smoked were depicted as role models hard at work in the national effort.[25-27]

The image of the female smoker as responsible and independent ended with the war. In advertisements during the second half of the 1940s, women were portrayed as wives and lovers, expecting or enjoying reunions with returning husbands and beaus. In an early morning scene from an advertisement of the period, an elegantly attired couple, arm-in-arm, look out the window. The text read, "It's spring again. It's two again. Just the way it used to be. Two to grab for the morning paper. Two places to set at the table. And two Chesterfields over two cups of coffee." [28] Another advertisement features a bride slipping a carton of cigarettes into her honeymoon suitcase.[29]

According to representative national surveys,[24] the prevalence of smoking among American women continued at relatively high levels through the mid-1960s (by then 33%), in contrast to American men, among whom the proportion of smokers began to decline in the 1950s. During this time, evidence of the serious disease risks associated with cigarette smoking—particularly the risk of lung cancer—was emerging from epidemiologic studies and was being broadcast to the public. Although most of the early health statistics were based on studies of men, it is probably fair to conclude that women who chose to smoke during this period did so with at least a suspicion that the incriminating medical data applied to them also.

Because of the availability of television for cigarette commercials, cigarette advertisements were not as prominent in the printed media during the 1950s and 1960s. Women's magazines of the period are therefore not a reliable index of the extent to which women were targeted in cigarette promotions. A review of three such publications (*Ladies Home Journal, McCall's,* and *Better Homes and Gardens*) for the years 1945, 1950, 1955, 1960, and 1965 revealed that the number of cigarette advertisements per issue ranged from zero to three.

1970s–1980s: WOMEN SMOKERS ARE EXPLOITED

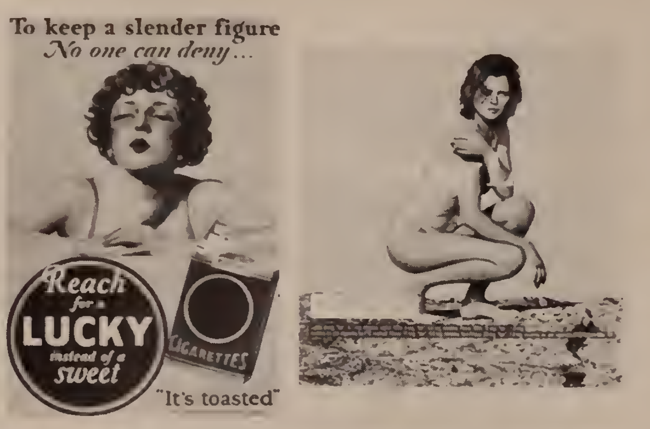
The tobacco industry publicly acknowledges that it is directing much of its contemporary advertising to the female market. A front-page article in *Advertising Age,* in 1981, headlined "Women top cig target" quotes the president and chief executive officer of RJ Reynolds describing the women's market as "probably the largest opportunity" for the company.[30] The article cites industry sources who viewed the working woman, under stress, as the ideal candidate for their product. In 1983, a major article in *Advertising Age* appeared under the headline "Marketers clamor to offer lady a cigarette." [31] Referring to the European market, a recent editorial in a tobacco trade publication entitled "Targeting women" noted the following:

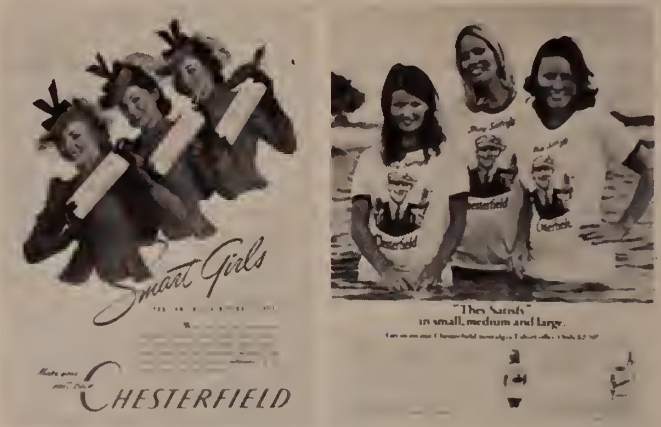
> Women are adopting more dominant roles in society; they have increased spending power; they live longer than men. . . . All in all, that makes women a prime target as far as any alert European marketing man is concerned.[32]

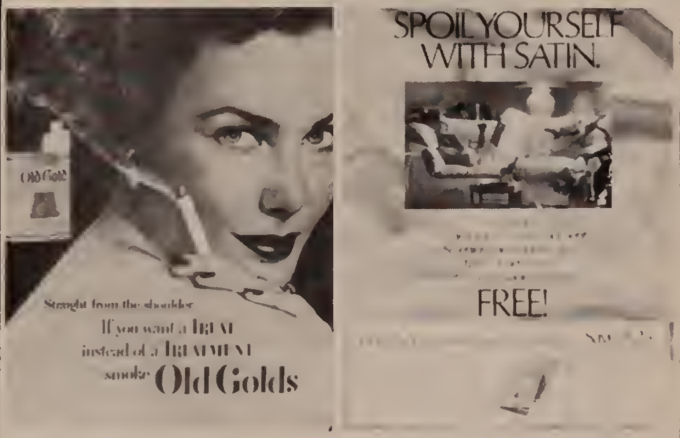
In recent years, a number of cigarette brands marketed specifically to women have been introduced, the most successful of which has been Virginia Slims (Philip Morris). Other brands that are marketed primarily to women include Eve (Grand Metropolitan), Satin (Loews), More (RJ Reynolds), and Ritz (RJ Reynolds).

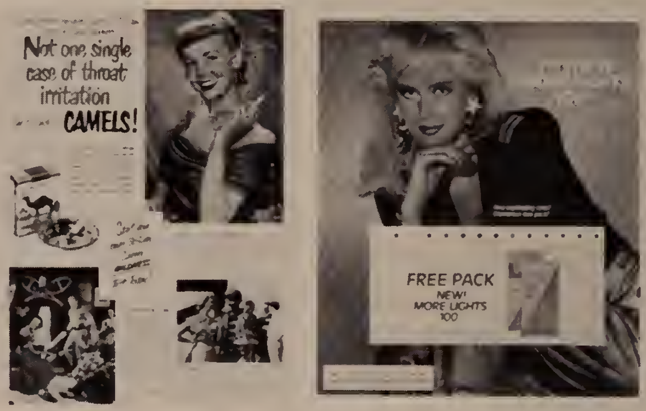
Following the ban on cigarette advertising on radio and

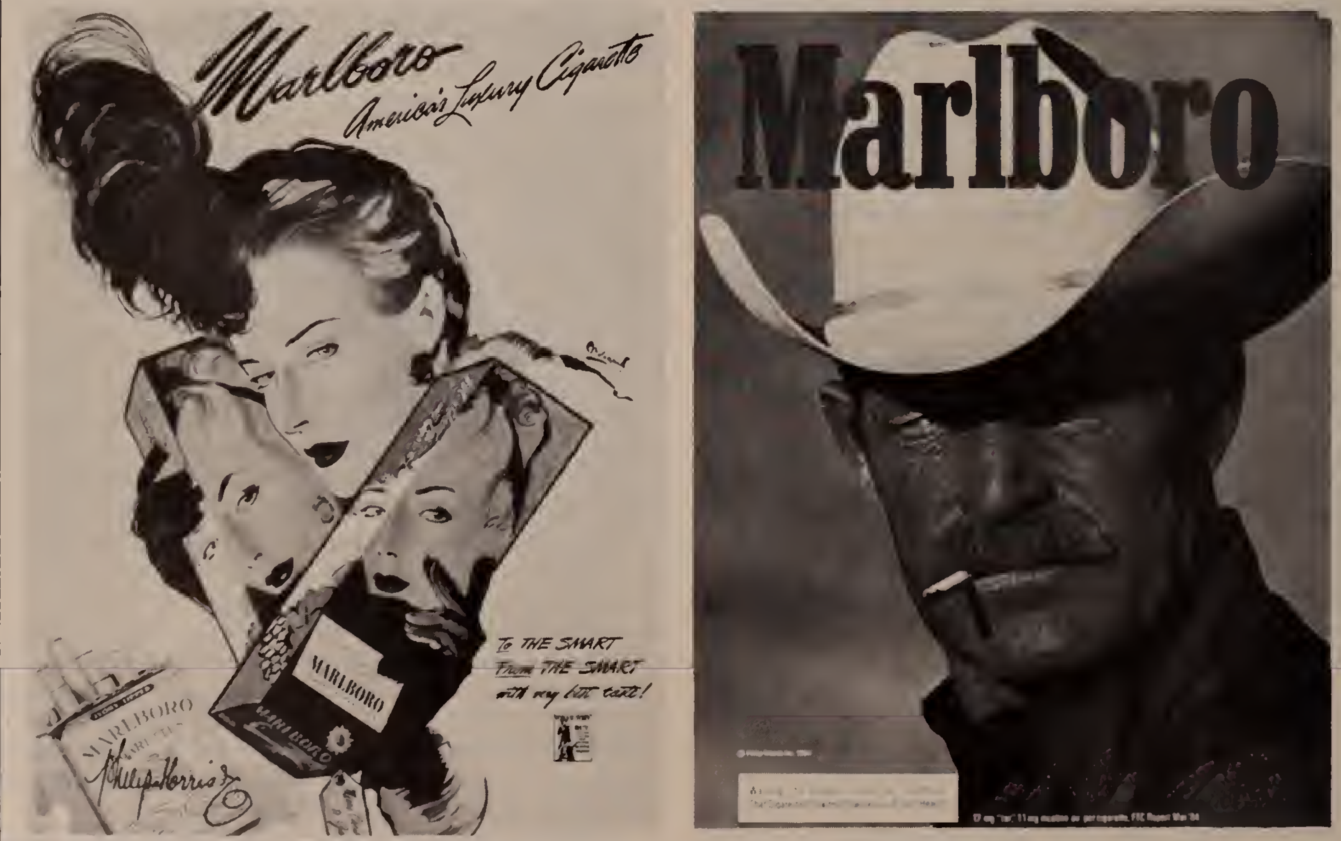
Side-by-side comparisons of old and new advertisements for cigarettes show how little has changed, except for Marlboro which overnight in 1954 went from being portrayed as the essence of femininity to the height of masculinity when a new advertising agency took over. (DOC Archive)


To keep a slender figure
No one can deny...
Reach for a LUCKY instead of a sweet
CIGARETTES
"It's toasted"









television, the number of cigarette advertisements in women's magazines increased dramatically (Fig 1). By 1979, cigarettes were the most advertised product in some magazines, with as many as 20 advertisements in a single issue.[33] Women's magazines are an important outlet for advertisers, since many of them rank among the best selling publications in the country. Of the 20 top-circulating magazines in the United States in 1980, eight were directed primarily at women, including six of the top 10 publications. Some of these magazines have estimated female readerships of more than 20 million. Except for *Good Housekeeping,* which has long refused to accept cigarette advertisements, the major women's magazines have become heavily dependent on such income. (*Seventeen* magazine, although not among the top sellers, also does not accept cigarette advertising, a significant stance given its target audience of young women.) Table I shows the average number of cigarette advertising pages per issue and the percentage cigarette advertisements represent of all advertising revenues for the major women's magazines in 1984.[34]

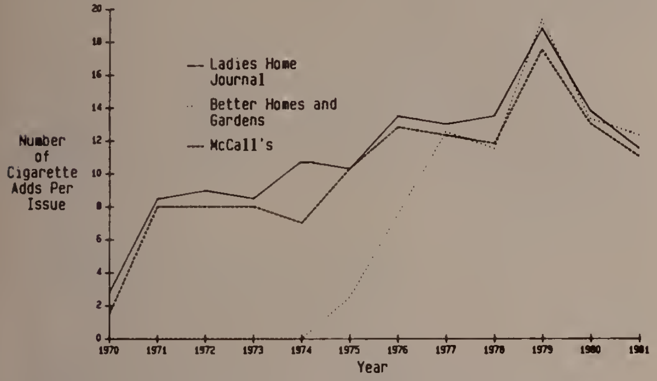


FIGURE 1. Average number of cigarette advertisements per issue in three women's magazines, 1971–1984.

TABLE 1. Cigarette Advertising in Major American Women's Magazines, 1984[1]

Magazine	Cigarette Advertising Revenues	Cigarette Advertising % of Total Revenues	Average Number of Cigarette Advertising Pages Per Issue
Better Homes & Gardens	14,970,751	11.9	14.8
Cosmopolitan	7,545,290	7.9	14.6
Essence	1,541,100	11.5	9.0
Family Circle	16,274,466	12.5	12.5
Glamour	5,753,343	8.2	12.2
Good Housekeeping	0	0.0	0.0
Harper's Bazaar	2,751,285	8.8	10.1
Ladies Home Journal	9,316,713	14.0	14.0
Mademoiselle	2,450,081	6.8	8.6
McCall's	10,706,748	14.0	13.3
Ms.	503,370	7.9	3.9
New Woman	1,404,935	20.1	9.6
Redbook	8,004,851	15.1	13.8
Vogue	3,622,795	5.4	12.2
Woman's Day	13,826,055	12.5	12.8
Working Mother	981,200	8.9	6.3
Working Woman	1,831,850	9.7	9.6

[1] Source: ref. 35

Work by Whelan and colleagues[35] suggests that the editorial policy of women's magazines that accept cigarette advertising is restrained in reporting the health hazards of smoking. Her group counted the number of articles about smoking that appeared in such major women's magazines during the period from 1967 to 1979; the number ranged from zero in some magazines to a maximum of two in others.

Cigarette promotions are not limited to suggestive copy. Many coupon offers for discounts or free packs appear in magazines and newspapers. In May 1983, an offer appeared in newspapers around the country for women to call a toll-free number and receive two free packs of Satin cigarettes and a pouch in which to hold them. The offer elicited an estimated 1.3 million calls within a 10-day period (Joanne Luoto, MD, director of the federal Office on Smoking and Health, personal communication). The More brand of cigarettes (RJ Reynolds) was promoted by sponsorship of fashion shows in 18 shopping centers throughout the United States. The publicity included a four-page advertisement in the March 1982 issue of *Harper's Bazaar*. Reynolds also sponsored a sweepstakes contest; the entry blank was included in an advertisement in *Woman's Day* magazine (April 6, 1982), and was accompanied by a discount coupon for More cigarettes. Philip Morris has offered clothing (including a rugby outfit and a T-shirt that reads "You've come a long way, baby") and calendars (The Virginia Slims Book of Days) in exchange for money and proof of cigarette purchase. Philip Morris sponsors the Virginia Slims professional women's tennis tour. Cigarette samples are given away at the entrance to the tennis matches. Leading professional women's tennis players have not taken public positions opposing cigarette promotions.

The industry has succeeded in being associated as a financial benefactor of the women's movement. *Ms.* magazine accepts a sizeable share of its advertising budget from cigarette companies and has yet to print a story on smoking, despite its inclusion of many health-related articles.[36] The National Organization for Women has had its meeting program partly underwritten by Philip Morris and recently refused to print in its national newsletter an adver-

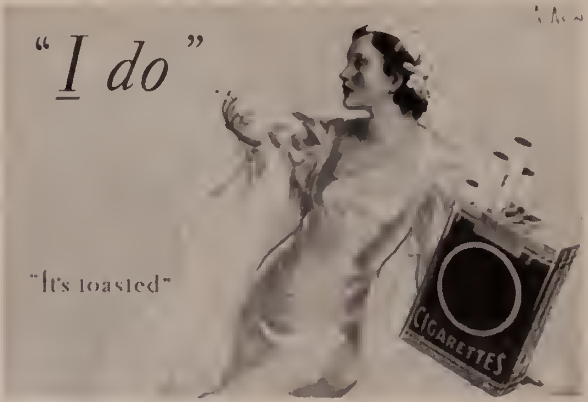
tisement taking *Ms.* to task on the cigarette advertising issue (Polly Strand, personal communication). By their silence on the issue of tobacco company exploitation of women, even when challenged, these representatives of the women's movement, as well as the publishers of women's magazines, must be viewed as accomplices in what has come to be called an "equal opportunity tragedy." The magnitude of current efforts to target women in cigarette advertisements and other promotions lends an aura of social legitimacy to a product whose users are often victims. British journalist and physician Bobbie Jacobson discusses social forces, including cigarette promotions, that contribute to smoking among women in her book *The Ladykillers: Why Smoking is a Feminist Issue.*[37]

CONCLUSION

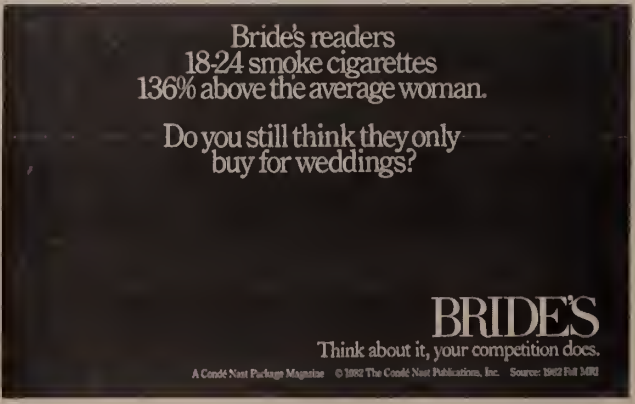
The negative moral connotations of smoking by women at the turn of the century gave way to the cigarette as a positive symbol of emancipation. Today, cigarettes once again have a negative image, but for health rather than moral reasons. What is the social meaning of cigarette smoking for the contemporary woman—or girl, since the decision to smoke is usually made before adulthood? The evidence suggests that women who are most "emancipated," if attained education is the measure, are no longer the most likely to smoke. Women with college educations and teenage girls who are college-bound are less likely to smoke than women with less education and girls who are not college-bound.[24,38,39] However, cigarette smoking continues at relatively high levels among women in the general population (25–30%). The symbolism of emancipation seems to persist and is deliberately fostered by the tobacco industry. Advertisements for cigarettes have always portrayed women favorably—as athletes, fun-loving, glamorous, sexually attractive, and as "in" socialites or flaunters of old-fashioned ways; they are rarely depicted in passive or traditional roles. For many young women, smoking still signifies defiance and independence. To this day, the messages about smoking for women continue to be mixed.

REFERENCES

1. Schlesinger AM, Fox DR: *The Rise of the City, 1878–1898,* New York, The MacMillan Company, 1933.
2. Barnard EF: "The cigarette has made its way up in society." *The New York Times* June 9, 1929, p 6.
3. Sobel R: *They Satisfy: The Cigarette in American Life,* New York, Anchor Press/Doubleday, 1978.
4. Sullivan M: *Our Times: The United States 1900–1925,* vol 2. New York, Charles Scribner's Sons, 1927.
5. Bain J Jr: *Cigarettes in Fact and Fancy.* Boston, HM Caldwell Company, 1906.
6. Sullivan M: *Our Times: The United States 1900–1925,* vol 4. New York, Charles Scribner's Sons, 1932.
7. Sullivan M: *Our Times: The United States 1900–1925,* vol 3. New York, Charles Scribner's Sons, 1930.
8. Women and cigarettes. *Printers' Ink* 1932; 158:25–27.
9. Fass PS: *The Damned and the Beautiful: American Youth in the 1920s.* New York, Oxford University Press, 1977.
10. Schudson M: Women, cigarettes, and advertising in the 1920s: A study in the sociology of consumption, in Covert C (ed): *Mass Media and the Unexpected.* Syracuse University Press, 1983.
11. Bonner L: Why cigarette makers don't advertise to women. *Advertising and Selling* 1926; 7:46.
12. Williamson O: An inhibition versus a market. *Advertising and Selling* January 26, 1927, p 56.
13. Marlboro makes a direct appeal. *Advertising and Selling* 1927; 8:25.
14. Fillmore CM: *Tobacco Taboo.* Indianapolis, Meigs Publishing Company, 1930.
15. Doyle NC: Smoking among women—an equal-opportunity tragedy. *American Lung Association Bulletin.* July–August 1980.
16. Hoover IH: Hail to the chief. *The Saturday Evening Post.* May 5, 1934.
17. Dale E: *The Content of Motion Pictures.* New York, The MacMillan Company, 1935.
18. The *Fortune* survey. III. Cigarettes. *Fortune Magazine* 1935; 12:111–116.
19. Cigarettes hit new high. *Printers' Ink.* July 9, 1936, pp 80–84.
20. "Blow some my way." *Printers' Ink.* 1932; 159:20.
21. Gottsegen JJ: *Tobacco: A Study of Its Consumption in the United States.* New York, Pitman Publishing Corporation, 1940.
22. The *Fortune* survey. VIII. Cigarette preferences. *Fortune Magazine* August 1938.
23. *Sales Management* 1937; 41:30.
24. *The Health Consequences of Smoking for Women—A Report of the Surgeon General.* Washington, DC, US Department of Health and Human Services, Public Health Service, Office on Smoking and Health, 1980.
25. *Better Homes and Gardens.* October 1942.
26. *Better Homes and Gardens.* October 1943.
27. *Better Homes and Gardens.* March 1943.
28. *Better Homes and Gardens.* May 1945.
29. *Ladies Home Journal.* June 1945.
30. O'Connor JJ: Women top cig target. *Advertising Age* 1981; 52:9,93.
31. Sobczynski A: Marketers clamor to offer lady a cigaret. *Advertising Age* January 31, 1983, pp M 14–16.
32. Rogers D: Targeting women. *Tobacco Reporter* 1982; 109:6.
33. Gardner F: Brand report 58: cigarettes. *Marketing and Media Decisions* October 1980, pp 175–190.
34. Publishers Advertising Reports, PIB Magazine Advertising Analysis: Magazine and Class Totals, December 1984. Publishers Information Bureau, 1985.
35. Whelan EM, Sheridan MJ, Meister KA, Mosher BA: Analysis of coverage of tobacco hazards in women's magazines. *Journal of Public Health Policy* 1981; 2:28–35.
36. Cromer MA: 'Precious baby.' *NY State J Med* 1983; 83:1292–1293.
37. Jacobson B: *The Ladykillers: Why Smoking is a Feminist Issue.* New York, Continuum, 1982.
38. *Smoking and Health—A Report of the Surgeon General.* Washington, DC, US Department of Health, Education, and Welfare, Public Health Service, Office on Smoking and Health, 1979.
39. Johnston LD, Bachman JG, O'Malley PM: *Student Drug Use, Attitudes, and Beliefs: National Trends, 1975–1982.* Rockville, MD, US Department of Health and Human Services, Public Health Service, National Institute on Drug Abuse, 1982.



1933



Bride's readers
18-24 smoke cigarettes
136% above the average woman.

Do you still think they only
buy for weddings?

BRIDE'S
Think about it, your competition does.
A Condé Nast Package Magazine © 1982 The Condé Nast Publications, Inc. Source: 1982 Fall MRI

1983

Getting women hooked: defending the indefensible

The following dialogue was written by Bobbi Jacobson, MB, BS, author of *The Ladykillers—Why Smoking is a Feminist Issue* (New York, Continuum, 1982). It is based on statements made by tobacco industry (TI) representatives in the mass media, in tobacco trade publications, and in interviews with the author.

Dr Jacobson: *The World Health Organization, the Royal College of Physicians, the British Medical Association, the Surgeon General's office of the United States, and countless other national and international medical bodies have all stated that smoking is the largest single avoidable cause of premature death in developed countries. These organizations also point to the high smoking rates among women and girls, who seem destined to be the next century's victims of lung cancer and heart disease. Is there any justification for the increase in cigarette promotion directed at young women?*

TI: First of all, we sell a product that is legal and for which there is enormous demand. We cater to the needs of tens of millions of smokers. Our advertising is directed only at current smokers and informs them of the choices available. As to the smoking and health controversy, we are not competent to make medical judgements, but we are aware that there is considerable disagreement within the medical profession itself concerning the smoking and health issue. Further research—to which we ourselves contribute heavily—will, we hope, lead to a fuller understanding of the complexities involved. Until then, we are happy—and are, indeed, obliged—to inform smokers and provide for their smoking needs.

BJ: *In March 1983, in an article entitled "Look to the ladies,"* Tobacco *magazine asked the question "Is there a manufacturer not setting out to win or consolidate a share of the woman smoker's habit?" And* Tobacco Reporter *noted in February 1982, "Women* [*are*] *a prime target as far as any alert European marketing man is concerned. So despite previous hesitancy, might we expect to see a more defined attack on the important segment represented by women smokers?" How much responsibility is the tobacco industry willing to accept for creating and reinforcing the women's market?*

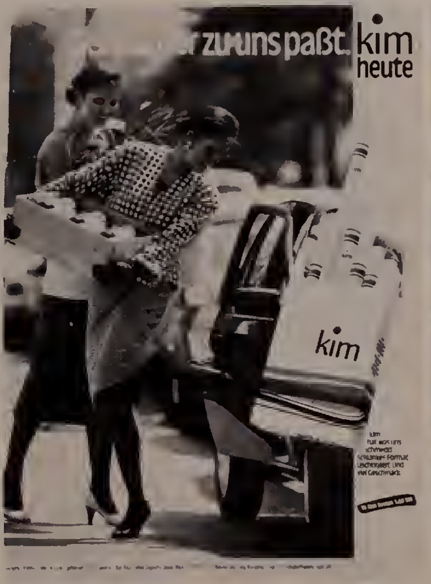


FIGURE 1. Cigarette brand heavily advertised to women in several countries.

FIGURE 2. Cigarette brand logo worn by tennis player at Wimbledon, televised worldwide.

TI: We simply cater to the demands of existing smokers, be they men or women. Obviously, we are aware of changing roles in society, and it is quite natural for us to market cigarette brands to women who are independent, self-confident, and who are looking for a cigarette that reflects their lifestyle.

BJ: *Are these brands aimed at girls?*

TI: No cigarette advertisement is ever specifically aimed at persons under 18 years of age. Our products are aimed at women smokers between 20 and 40 years who are looking for a distinctly female cigarette (Fig 1). The tobacco industry adheres to the letter of the stringent criteria of the voluntary self-enforcement agreements.

BJ: *Why do you think in England in recent years women tennis players at Wimbledon have worn dresses imprinted with cigarette brand name logos and designs(Fig 2)? Does not the exposure of these championship matches to 350 million viewers around the world suggest that tobacco companies are eager to associate smoking with athletes and to circumvent the ban on the advertising of cigarettes on television?*

TI: Tobacco companies had nothing whatsoever to do with the promotion of what was a line of leisurewear. Clothing does not constitute cigarette advertising, a fact that was accepted by the ruling tennis body, broadcasting authorities, and the Minister for Sport.

BJ: *In an article mentioned earlier it was reported that marketing to women involves the three 'L's': low tar, longer length, and lower price. Indeed, sales figures show that women are two to three times more likely than men to smoke low tar cigarettes—or "Lights" as you prefer to call them. These brands are favored by women over men in all age groups—including the young. Would you say that your lower tar promotions make it easier to get women and girls to smoke?*

TI: I reiterate that our advertising is designed to appeal to both men and women smokers. If more women prefer a light cigarette, we are happy to help them make an informed choice of milder brands. This is in keeping with government policy to reduce the tar content of cigarettes. Furthermore, our promotion is strictly not directed at the young.

BJ: *If tobacco companies do not direct promotions at young people, why do they advertise cigarettes in magazines like* Mademoiselle, *a sizeable proportion of whose readership is teenage girls?*

TI: If it is legal to sell cigarettes, it is legal to advertise them.

Superman and the Marlboro woman

The lungs of Lois Lane

PAUL MAGNUS, MB, BS

Since its release by Warner Communications in 1980, the film *Superman II* has been viewed by millions of children and teenagers around the world in theaters, on commercial television, and on video recorders. They have marveled at the high-tech adventures of the comic book hero who has fought for truth, justice, and the American way for nearly half a century. As expected Superman saves the day against assorted super-criminals. But in addition, Lois Lane, his romantic interest, plays a prominent role. On the one hand, she is a bright, independent, and daring reporter on *The Daily Planet*. On the other hand, she is sexy, romantic, and submissive to the muscular superhero.

Lois Lane, role model for millions of teenage girls and children, also smokes cigarettes, the product that kills more people than alcohol, other drugs, and automobiles combined. Moreover, in *Superman II* there are numerous images, both fleeting and lingering, of her brand of cigarettes—Marlboro—the world's number one seller, manufactured by Philip Morris, Inc, New York. Consider this scene: Lois Lane and Superman's alter ego, reporter Clark Kent, are hurrying through the offices of *The Daily Planet*. Lois has a cigarette in her mouth. They enter a room where she begins to squeeze some orange juice. There is a close-up of a cigarette dangling from her lips while she touts the benefits of vitamin C. As she stubs out her cigarette in a filthy ashtray, she talks of the "natural" quality of the orange juice.

This contrived scene—the camera zooms in on the ashtray just as Lois says "natural"—brings a healthy laugh from the adults in the audience. Without doubt, this was a deliberate, playful dig about the unnaturalness of cigarette smoking and Lois Lane's contradictory health priorities.

Yet, moments later, the camera focuses on Lois' Marlboro cigarette pack. What seemed to be a message that mocked smoking becomes confusing. How did a brand of cigarettes get in on the act? What comes to mind is the ironic imagery of the long-running advertising campaign for Virginia Slims cigarettes, also manufactured by Philip Morris. The advertisements feature two old-fashioned, sepia-tone photographs at the top of the page with a caption that pokes fun at the misfortunes of women who dared to smoke in the cruel male-dominated world at the turn of the century. The larger part of the advertisement, in full-color, features an attractive, smug, and inviting woman holding a cigarette, and the headline, "You've come a long way, baby." Could a bit of self-effacing humor by Lois Lane actually have been intended to accentuate the positive image of the woman who smokes?

Even those who do appreciate the cigarette-orange juice gibe may just as easily empathize with Lois. People seem to be giving smokers a hard time these days. It makes Lois Lane more vulnerable, endearing, and "womanly."

Questions also arise concerning other scenes in the film. In one scene Lois Lane drags on a cigarette; she is dressed in red and white, the colors of Marlboro. In another scene of a climactic battle with super-powered criminals (Figs), a truck with a bright red and white Marlboro logo is seen in the center of the screen. Superman is hurled into the truck, and he then fends off an attacker, with the cigarette brand logo in the background. The truck is then rammed by a bus hurled by one of the super-villains, burying Superman in the wreckage. After a worrisome pause, Superman emerges through the Marlboro logo. As an added touch, a taxi with a Marlboro advertisement appears in constant view in the background.

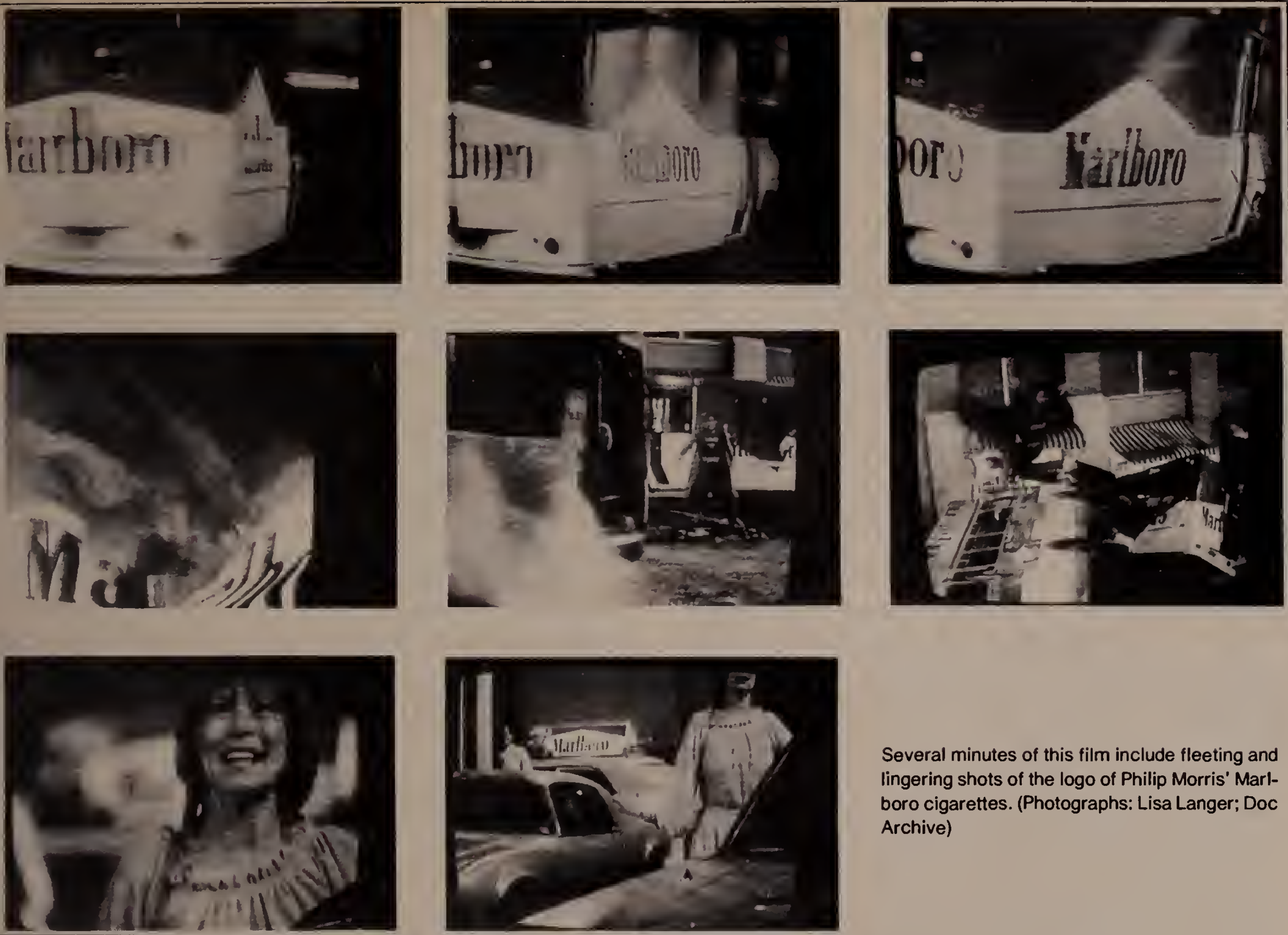
Marlboro is thus dramatically linked to both the superhero and the heroine. The pulling power of a clean-living universal legend is exploited by a commercial trick. Philip Morris has achieved an important recruiting message. For an undisclosed consideration, the moviemakers permitted repeated juxtapositions of a children's hero with a brand of cigarettes.

This hypothesis was confirmed by two sources associated with the film. According to one, the Lois Lane-orange juice scene was intended to be taken in the context of the first Superman movie, released in 1978. In a scene in that film, Superman used his x-ray vision to view Lois Lane's lungs, commenting that she really should not smoke. Taken in context, the message in the original scripts of both films was meant to discourage smoking, it was said. The Superman character has always shunned tobacco and alcohol. In England his picture has been lent to an anti-smoking campaign.

Then why have Lois Lane smoke in the first place, and why the repeated identification of Marlboro? Both sources acknowledged that the cigarette brand appearances were no accident. One source admitted that it was "most unfortunate."

It is possible that Philip Morris was not concerned with the message about smoking, as long as the viewer understood that when one smokes, one smokes Marlboro; and when there is a cigarette truck, it is a Marlboro truck. Yet not even tobacco wholesalers or Philip Morris representatives from several cities interviewed for this report could recall having seen similar Marlboro trucks. The lone exception was a public relations person for Philip Morris in New York, who insisted that such trucks exist in the United States. This person claimed that it was the moviemakers who approached Philip Morris for help with authentic background props, as is done in the movie business.

Address correspondence to Dr Magnus, Medical Associate, National Heart Foundation of Australia, PO Box 2, Woden, ACT 2606.



Several minutes of this film include fleeting and lingering shots of the logo of Philip Morris' Marlboro cigarettes. (Photographs: Lisa Langer; Doc Archive)

The fact remains that many impressionable young people on the threshold of lighting up would not understand the joke on Lois Lane. They would see that she smokes, however, and are unlikely to recall the comment by Superman in the earlier movie.

The two decisive factors are that the enviable Lois does smoke and that she smokes Marlboro. They ensure that one overriding message will be perceived: women who smoke (Marlboro) succeed. Far from suffering from this habit, Lois is rewarded for it. And with her is born a new myth: the Marlboro woman, an image reinforced by a recent upsurge of advertising for Marlboro in such women's magazines as *Vogue*, *Woman's Day*, *Ms.*, *Working Woman*, and *Family Circle.* If the movie's effect were truly anti-smoking, it is unlikely that any cigarette company would have permitted its brand name to be singled out.

Film critic Hollis Alpert has claimed that the tide is turning against the on-screen smoking habits of heroes and heroines, and that Hollywood has "all but kicked the tobacco habit." ("Smoking no longer sexy in films, TV." *Cancer News* [American Cancer Society], Spring/Summer, 1981). Most people would probably believe that if anyone still smokes on the screen these days it is most likely to be the bad guy. But *Superman II* laid the foundation for just the opposite circumstance. How many thousands of girls, primed for smoking, will the movie have given that last nudge into a cigarette habit or pushed a bit closer? How many who are already hooked will be encouraged to continue? The uncomfortable reality that most adults refuse to confront is that today's children face a higher risk of death due to cigarette smoking than with other more sensationalized drugs. *Superman II* will not help.

Logos of other products displayed in Superman II *include Cutty Sark whiskey, Coca Cola, Kentucky Fried Chicken (RJ Reynolds), and Omni and Rich Lights cigarettes (Brown and Williamson). Various cigarette brands have been shown in recent films, including* Animal House, The Goodbye Girl, Moonraker, *and* Educating Rita. *In a similar skirting of the law tobacco company sponsorship is increasing on cable television and public broadcasting. Rock videos featuring such stars as Barbra Streisand frequently show dancers and singers with a drink and a cigarette.* *—Editor*

Cigarette smoking and ill health among black Americans

RICHARD COOPER, MD, BRIAN E. SIMMONS, MD

Cigarette smoking might be equally regarded as a symptom of a society at odds with itself or as a cause of disease per se; at either level, it has earned the reputation of public health enemy number one. Smoking is also a crucial example of how the health of the black population has worsened under the guise of social advancement. Blacks now suffer the highest rates of coronary heart disease (CHD) and lung cancer of any population group in this country.[1] This fact has been obscured by the tendency in medicine to focus attention on "typical" black diseases such as hemoglobinopathies and hypertension.

In 1977, there were 80,000 excess deaths among blacks, compared to the mortality rate for the rest of the population.[1] Yet the only clear-cut genetic disease of blacks known to contribute to this differential is sickle cell disease, and, in 1977, only 277 deaths among blacks were recorded as hemoglobinopathies, or 0.3% of the excess.[1] Environmental and social causes must be defined to explain the remaining 99.7%. The structure of the educational system, a discriminatory job market, and residential segregation have created a series of social relationships that confine blacks to a fixed position in society. The sum of these social institutions, reinforced by common attitudes and prejudices, generates the invisible chains of racial oppression. Although the specific causes of black ill health may change over time, the fact of the disadvantage is a constant and will remain so until the intensity of racial discrimination is reduced.

The cigarette industry has exploited racial divisions in defining a profitable black market. Specific brands, notably Kool (Brown & Williamson) and, to a lesser extent, Winston, More, and Salem (RJ Reynolds), Newport (Loews), and Virginia Slims (Philip Morris) have been promoted for maximum consumption in the black community through the black-owned press and by means of sponsorship of black civic organizations by tobacco companies. At the same time, black-oriented educational campaigns to discourage smoking have been limited.

PATTERNS OF CIGARETTE-RELATED DISEASE

Sources of data. It is difficult to arrive at precise estimates of the disease burden imposed by smoking on blacks. Long-term prospective studies of a sufficiently large and representative cohort would be required for direct estimates. Unfortunately, no such studies exist, and there are none currently underway. The major prospective studies of cardiovascular disease have never enrolled adequate numbers of black participants. Of the original 5,000 persons in the Framingham cohort, only six were black,[2] and industry-based studies that were initiated at the same time did not include meaningful numbers of minority participants.[3] A prospective study of smoking by the American Cancer Society enrolled 1 million participants, only 22,000 of whom were black.[4] Two large randomized cardiovascular disease trials which were recently completed—the Multiple Risk Factor Intervention Trial (MRFIT) and the Hypertension Detection and Follow-Up Program (HDFP)—screened large numbers of blacks in the recruitment phase, and the follow-up data are now becoming available.[5,6] All participants in MRFIT were men, however, and recruitment was aimed primarily at employed members of the middle class, resulting in a sample that is not representative of the black population. The cohort being followed in a prospective study by the National Health and Nutrition Examination Survey (of the National Center for Health Statistics, Hyattsville, MD) includes a more representative black sample, but one that also is limited in size (only 528 black men and 635 black women over the age of 25).

In the absence of survey data, it is necessary to rely on vital statistics. There are important limitations of the vital records system. In recent years, deaths have been coded by race, as opposed to the color system of "white" and "nonwhite." Although this may improve present data sources, it introduces problems of comparability when looking at trends over time. In addition, coding of death certificates is subject to misclassification in categories such as sudden coronary death. Lastly, as in all mortality studies, comparisons between populations reflect survivor effects as well as selection by competing cause. The survivor effect is best seen in the black-white crossover in old age.[7] Thus, while fewer blacks live to the age of 80, those who do survive are healthier and suffer lower age-specific death rates than do whites. Since a larger proportion of the white population survives into old age, they will be more likely to die of the more common diseases of old age. The phenomenon of competing cause tends to eliminate potential candidates for a specific disease through premature death from another, related disease. Thus, if cigarettes contribute to both CHD and lung cancer, a population with a high rate of exposure to other risk factors (such as hypertension or elevated cholesterol level in the case of CHD, or industrial occupations in the case of lung cancer) might selectively lose high-risk individuals at a young age. A cigarette smoker dead of CHD at age 45 is no longer available to die of lung cancer at 50. Given the higher overall mortality of blacks compared to whites some distortions of cause-specific differentials will be observed. Unfortunately, there is no direct way to compensate for competing cause effects, and its true impact can only be a subject of speculation. One way to derive qualitative estimates of competing cause is to examine age-specific mortality patterns. While

---

From the Division of Cardiology, Department of Medicine, Cook County Hospital, Chicago.

Address correspondence to Dr Cooper, Associate Chairman, Division of Cardiology, Department of Medicine, Cook County Hospital, Chicago, IL 60612.

age-adjustment compensates for the differences in the age structure between the black and white populations, it still exaggerates the relative importance of the diseases of the very elderly, since a higher proportion of the white population dies in this age category. The practical significance of these problems will become apparent in the analysis of cigarette-related disease that follows.

Blacks have a six year shorter life expectancy and higher mortality rate from all but two of the 15 leading causes of death.[1] While violence and stroke are associated with higher relative rates among blacks, heart disease and cancer make up by far the largest contributions to excess black mortality, accounting for 30% of the increase in all causes of mortality for men and 40% for women. As the two major causes of death in this country and the major health consequences of smoking, heart disease and cancer will serve as the primary focus of this analysis.

Smoking patterns. Although surveys of cigarette use are subject to the bias of under-reporting, findings in numerous studies over a period of 40 years yield consistent conclusions.[8] Blacks generally took up the smoking habit 10 to 20 years later than whites, in large part as a consequence of the mass migration from the rural South to the urban North.[9] The first severely affected generation of blacks was that reaching young adulthood in the 1940s and 1950s. In the mid-1950s smoking rates for blacks were still lower than those for whites.[8] During the past 20 years the racial patterns of smoking have remained static, with parallel changes in both groups. The percentages of adult smokers have leveled off at about 45% for black men and 30% for women (Table I). Despite the higher prevalance of cigarette users among black men, numerous reports have shown that black smokers use fewer cigarettes per day. Thus, while 37% of white male smokers reported use of 25 or more cigarettes per day in 1980, only 11% of black men smoked that heavily. Among women, 26% of the white women smoked 25 or more cigarettes daily, compared to 10% of black women.[10] Age patterns of smoking are similar in women in the two races, yet among young black men there is a higher prevalence of cigarette use.[10] In sum, taking into account both the prevalence of smoking and the number of cigarettes consumed per smoker, it seems reasonable to conclude that as a population black women smoke 10% to 20% less than white women, while black and white men consume similar amounts of tobacco. These estimates do not take into consideration other factors such as differences in degree of inhalation, length to which the cigarette is smoked, the age at which smoking began, and brand preferences.

TABLE I. Smoking Rates by Age, Sex, and Race; US, 1965 and 1980

	Percentage Current Smokers			
	1965		1980	
	White	Black	White	Black
Men				
All ages ≥ 20	52.1	59.6	37.1	44.9
25–34	60.7	68.4	42.0	52.0
35–44	58.2	67.3	42.4	44.2
45–64	51.9	57.9	40.0	48.8
≥ 65	28.5	36.4	16.6	27.9
Women				
All ages ≥ 20	34.5	32.7	30.0	30.6
25–34	43.4	47.8	31.6	43.2
35–44	43.9	42.8	35.6	36.5
45–64	32.7	25.7	30.6	34.3
≥ 65	9.8	7.1	17.4	9.4

Source: National Health Interview Survey (18, p 365)

Coronary heart disease. There is a common misconception among physicians that blacks are relatively immune to CHD. The reasons for this belief are complex, based on such divergent causes as the reputation of CHD as a disease of affluence and the higher levels of high density lipoprotein cholesterol reported among blacks.[11] Although CHD rates appear to have been lower in blacks than whites for a decade or so after World War II,[12,13] they are slightly higher at the present time. Based on the 1981 vital statistics, age-adjusted mortality for CHD (both sexes combined) among blacks was 144 per 100,000, versus 133 for whites.[1] Two recent symposia have debated at length the epidemiology of CHD among blacks.[14] The data are inconsistent, particularly in relation to men, and additional population-based prospective studies are needed. The most recent age-sex-race specific data for CHD are presented in Table II. Age-adjusted rates are virtually the same for black men, compared to white, while they are 36% higher for black women. The phenomenon of an age crossover is apparent among men, with rates 50% higher in the age range 35 to 44 years for blacks, but higher for whites above 65. Only the much larger proportion of deaths among whites above age 80 make the age-adjusted rate similar in men. Among women, a crossover is seen at age 80, with rates over 85 being twice as high for white women.

TABLE II. Age-Specific Death Rates from Coronary Heart Disease, by Sex and Race, US, 1978 (per 100,000)

	Men		Women	
Age (yrs)	White	Black	White	Black
35–44	57.8	97.7	10.7	38.1
45–54	253.4	335.9	54.1	159.3
55–64	713.7	846.3	216.9	446.4
65–74	1,654.5	1,568.3	717.1	1,039.3
74–85	3,939.5	3,341.2	2,505.2	2,608.3
85+	7,597.7	4,024.6	6,246.0	3,463.1
All ages, age-adjusted	257.8	260.6	117.2	159.7

Source: *Health: United States, 1981*, pp 124–127.

Why should a population with high rates of disease in middle age become resistant as it grows older? No intrinsic biologic explanation for this phenomenon exists. In a period of rapid secular trends, it is possible that rates could be going down for whites overall, while moving in the opposite direction for blacks. The younger generation would thus represent a new high risk group; as this generation ages it would continue to experience higher rates, and, eventually, the differential would be consistent at all ages. In fact, the racial crossover is not a transient phenomenon but one that has existed in some form for many years. In addition, secular trends in CHD have paralleled each other in the two racial groups for the last 15 years, declining rapidly in both.[13] The most likely explanation of the crossover is a combination of the healthy survivor effect and competing cause. Blacks at high risk for CHD, namely those who smoke or have another, related disease, such as hypertension, are more likely to die before reaching the age of 80 than are whites.[7] Loss of these individuals from the cohort would falsely reduce the importance of the underlying process of coronary arteriosclerosis.

Direct evidence exists to support the argument that competing cause reduces apparent CHD rates. In the follow-up data from MRFIT, blacks had slightly lower rates of CHD, yet, as expected, they experienced a proportionately higher mortality from stroke.[5] CHD death rates were 4.6/1,000 for black men, and 5.2/1,000 for whites, while stroke mortality was 1.3/1,000 for blacks and 0.5/1,000 for whites. More important, given the prospective nature of these data, it could be demonstrated that the deficit of blacks dying from CHD occurred exclusively in the hypertensive group. It was this group that contributed to the excess number of deaths from stroke in blacks. It seems likely that many black men who are prone to coronary disease because of hypertension as well as cigarette use, are dying from stroke, renal disease, and heart failure before they have a chance to become a victim of CHD.

There has long been concern that death certification may be less reliable for blacks than for whites, and may have biased the vital statistics. A new longitudinal study of cardiovascular disease (Community and Cohort Surveillance Program) has been organized by the National Institutes of Health and will allow more precise study of mortality patterns.[15] Data from the pilot phase of the study indicated the following age-adjusted rates per 1,000 persons of fatal CHD: black men = 3.12, white men = 2.77; black women = 1.37, white women = 1.00.[15] Although both incidence and mortality appear to be higher at younger ages, the black disadvantage persists in the age-adjusted mortality data. However, these rates are based on relatively few events, and only after completion of this study will there be definitive evidence.

In summary, CHD remains the most common cause of death in the black population, accounting for more than 20% of all mortality. Despite the popular misconception, it occurs more frequently among blacks than whites. The increase in risk of CHD associated with cigarette smoking is the same in blacks and whites.[5] Cigarette smoking has been estimated to account for one third of all CHD deaths,[10] and doubles the relative risk of CHD among individuals with hypertension. Widespread smoking has accelerated the development of atherosclerosis in the black population.

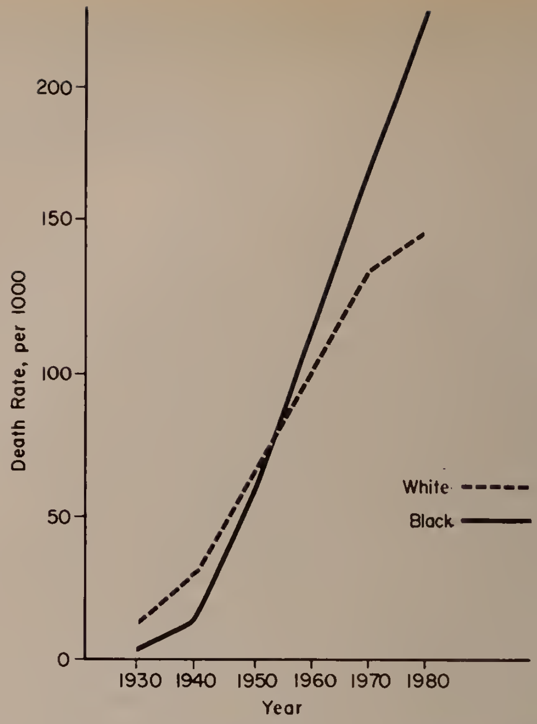


FIGURE 1. Death rates from lung cancer for men, black and white, ages 45 to 64, US, 1930–1980.

Lung Cancer. When the rise in the incidence of primary lung tumors was first recognized in the 1930s, the death rates among blacks were about half of those of whites. Since then there has been an enormous increase in lung cancer in the black population (Table III, Fig 1). Rates among both black and white women have accelerated since 1950. A racial age crossover in lung cancer is seen for both sexes (Table IV).

The rate of rise for men from 1950 has been three times greater for blacks than whites, and in the "maximally affected cohort"—the generation with the highest age-spe-

TABLE III. Respiratory Cancer Death Rates, Men, White and Non-White, US, 1930–1980 (per 100,000)

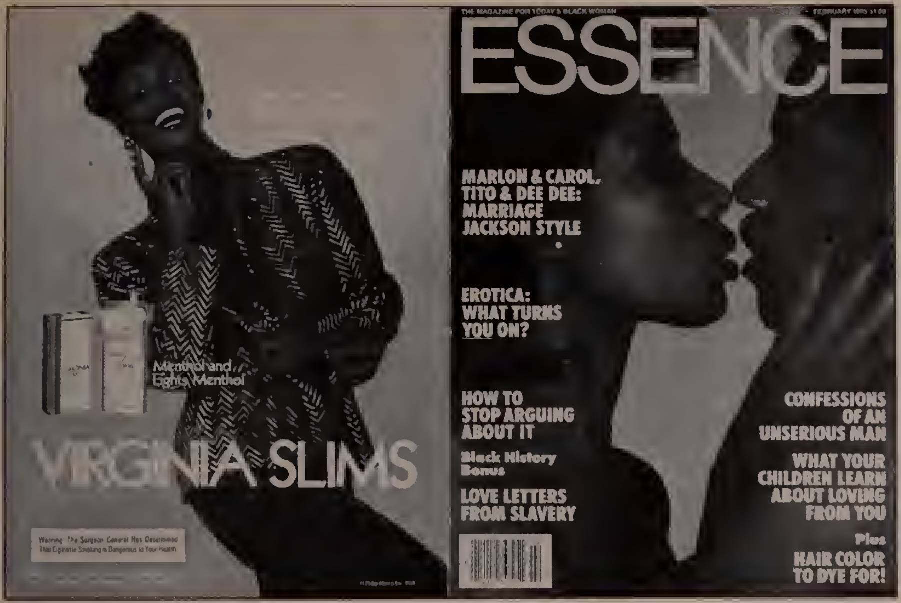
Age-Race Group	1930	1940	1950	1960	1970	1980	% Increase 1950–1980
White							
45–54	8	20	35	54	68	75	+133
55–64	13	41	85	152	199	214	+152
Non-White							
45–54	3	15	34	72	113	148	+335
55–64	6	20	69	159	232	340	+393

Source: US Vital Statistics Yearbooks, selected years; *Health: United States, 1981*, pp 132–133.

TABLE IV. Age-Specific Respiratory Cancer Death Rate by Sex and Race, US, 1978 (per 100,000)

	Men		Women	
Age (yr)	White	Black	White	Black
35–44	11.7	26.8	7.0	8.6
45–54	75.0	147.6	32.7	41.6
55–64	214.4	339.7	69.9	68.5
65–74	407.5	439.5	90.5	70.3
75–84	510.7	493.6	91.4	92.0
85+	359.3	215.4	77.6	50.8
All ages, age-adjusted	57.4	79.1	16.8	16.0

Source: *Health: United States, 1981*, pp 132–134.


Menthol and Lights Menthol
VIRGINIA SLIMS
THE MAGAZINE FOR TODAY'S BLACK WOMAN
ESSENCE
MARLON & CAROL, TITO & DEE DEE: MARRIAGE JACKSON STYLE
EROTICA: WHAT TURNS YOU ON?
HOW TO STOP ARGUING ABOUT IT
Black History Bonus
LOVE LETTERS FROM SLAVERY
CONFESSIONS OF AN UNSERIOUS MAN
WHAT YOUR CHILDREN LEARN ABOUT LOVING FROM YOU
Plus HAIR COLOR TO DYE FOR!

VIRGINIA SLIMS Lights
You've come a long way, baby.
A GUIDE TO BLACK ORGANIZATIONS

Come up to Kool.
Kool gives you extra coolness for the most refreshing sensation in smoking.
A sensation beyond the ordinary.

Alive with pleasure!
Newport
After all, if smoking isn't a pleasure, why bother?

Cigarette ads account for in excess of 12% of total advertising in *Essence*, "The magazine for today's black woman" (top left). Apart from alcohol advertising (20%), cigarettes are the leading product category in the magazine. Among advertisers tying in to Black History Month in February 1985 was RJ Reynolds, which marked the occasion in *Essence* with a discount coupon good for $1.50 off a carton of Salem (bottom left). The advertisement also featured the headline "A Salute to Black Scientists and Inventors" and a portrait of George W. Carver. Other advertisers in each issue of *Essence, Jet, Ebony* and other black-oriented publications include Brown & Williamson's Kool (bottom center) and Loews' Newport (bottom right). Since 1981 Philip Morris has published *A Guide to Black Organizations*, replete with cigarette advertisements featuring black models, which is distributed to black politicians and other leaders (top right). In the introduction to the 1983 edition, Philip Morris chairman Hugh Cullman wrote that the "directory's purpose is to illustrate for Blacks—and for all Americans—how much can be done when people work together in the name of progress and prosperity."

cific rates—mortality is more than twice as high. Age-adjusted rates for men are now almost 40% higher among blacks. Although the rate of increase has begun to slow for white men, the exponential increase continues among blacks, and the peak cannot be predicted.

The sharp upturn in lung cancer among blacks in the 1950s is consistent with the 20-year latency period expected from other population studies of cigarette use, given the increase in smoking among blacks during World War II. As noted above, however, blacks did not smoke more than whites overall at this time and certainly not in the 20-year period before 1960. The higher lung cancer rates cannot therefore be explained entirely by smoking. On the other hand, if racial susceptibility were important, a higher rate among black women (as compared to white women) would be expected.[16] It has also been noted that much of the black-white difference in lung cancer can be eliminated with control for social class.[17] It is possible that this class and race difference is mediated in part by other known risk factors for lung cancer, such as occupational exposures, low vitamin A intake, and inner-city residence. At the present time no direct data are available to assess the relative impact of these various causative factors. Whatever the mix of risk factors, it is clear that the introduction of smoking into this population has created a public health crisis.

Bronchitis and emphysema. Little has been written on the question of chronic obstructive pulmonary disease (COPD) in the black population. Based on vital statistics, blacks of both sexes have lower rates than whites.[1] Classification of deaths under this code began only recently, however, and adequate time trends are not available. Because COPD is a less frequent cause of death and is related to CHD and cancer by virtue of the common cause of smoking, interpretation of these data is further complicated by the problem of competing cause.

CAUSE AND CURE

In a disease-based analysis, the foregoing discussion has attempted to outline the impact of the cigarette on the health of the black population in the United States. Although the health consequences of smoking extend well beyond CHD and lung cancer, these disease categories subsume the majority of cigarette-related deaths and demonstrate the epidemiologic patterns. As noted at the outset, however, from the point of view of the etiology of this epidemic, smoking should be conceived of as a mediating factor—a proximate, not an ultimate, cause. Tobacco use is a social phenomenon and can be best understood only through an analysis of the relationships between people and institutions within that society. Approximately $30 billion per year is currently spent on tobacco products; about $6 billion in profits return to the cigarette industry, $6 billion to the government in taxes, and varying lesser amounts to a host of advertising agencies, retail stores, and farming interests. It is no secret that maintaining the flow of these profits has been the social purpose for which 360,000 persons a year die due to cigarette-related disease in this country. Cigarette smoking now displays a sharp class gradient, being concentrated among the working class and the poor of both races (Table V). Extracting profit by this means has proven to be an effective adjunct to the overall process of exploitation at work in contemporary American society. The basic causes of the cigarette-disease epidemic among blacks are, therefore, the same as the causes in the rest of the population.

TABLE V. Age-adjusted Percentages of Current Smokers by Education, White and Black Men, 1970-1975 and 1975-1981

Year	White: High School	White: College	Black: High School	Black: College
1970-75	48%	36	59	56
1976-81	37%	24	57	45

Source: Wynder EL, Goodman MT: Smoking and lung cancer: some unresolved issues. *Epidemiol Rev* 1984; 5:190.

There is no likelihood of effective cures for smoking-related diseases. Prevention requires the removal of tobacco, obstacles to which are too difficult to surmount in an economic system based on production for profit. The only realistic hope for prevention, under these circumstances, lies in broadening the gains made against smoking among the educated and privileged to include the working class and minority groups. Considering all the other health threats to black Americans, there is a most urgent need to sound the alarm about the compound ill effects of smoking (and the pernicious and pervasive influence of tobacco companies), especially by those who occupy positions of respect in the black community. Finally, there is ample evidence that doctors can change smoking habits of patients. Approximately 25% of patients will stop permanently at the urging of their physician, making this approach perhaps the single most potent means of influencing individual behavior.[18] Within the medical community, greater awareness of the problem of CHD and lung cancer among blacks is needed, and this must be translated into increased anti-smoking efforts.

The black-white health differential remains because little is done about it. The gains that followed on the heels of the civil rights movement and the urban rebellions of the 1960s and 1970s could be measured in improved health.[19] Despite a temporary reduction of racial inequality, the opposite trend has returned. Income among blacks was half that of whites until 1950; although the ratio rose to 0.61 in 1970, it has fallen back once again.[20] The widening gap in black-white infant mortality suggests a parallel outcome for health.[21] Institutional racism is an indispensible pillar of American society; without it current profit levels could not be maintained and capital would suffer enormous dislocation. At the same time, to take a serious view of this problem requires acceptance of the conclusion that inequality must no longer persist. Just as the ongoing power of the tobacco industry makes a mockery of any claim that the business community and government put the health of the nation above all other interests,[10] so the excess price in health demanded of the black population belies any claim that racism does not lie at the core of American society.

REFERENCES

1. National Center for Health Statistics: Advance report, final mortality statistics, 1981. *Monthly Vital Statistics Report*, Vol 33, No 3, suppl. DHHS Pub No (PHS) 84-1120, Public Health Service, Hyattsville, MD, June 22, 1984.

2. Wilson PW, Savage DD, Castelli WP, Garrison RJ, Donahue RP, Feinlab M: HDL-cholesterol in a sample of black adults: The Framingham Minority Study. *Metabolism* 1983; 32:328–332.

3. Pooling Project Research Group: final report of the Pooling Project. *J Chron Dis* 1981; 34:201–306.

4. Garfinkel L: Cigarette smoking and coronary heart disease in blacks: comparison to whites in a prospective study. *Am Heart J* 1984; 108:802–807.

5. Neaton JD, Kuller LH, Wentworth D, Borhani NO: Total and cardiovascular mortality in relation to cigarette smoking, serum cholesterol concentration, and diastolic blood pressure among black and white males followed up for five years. *Am Heart J* 1984; 108:759–769.

6. Langford HG, Oberman A, Borhani NO, Entwisle G, Tung B: Black-white comparison of indices of coronary heart disease and myocardial infarction in stepped-care cohort of the Hypertension Detection and Follow-Up Program. *Am Heart J* 1984; 108:797–801.

7. Manton KG: Temporal and age variation of United States black/white cause-specific mortality differentials: a study of the recent changes in the relative health status of the United States black population. *Gerontologist* 1982; 22:170–179.

8. Hammond EC, Garfinkel L: Changes in cigarette smoking, 1959–1965. *Amer J Public Health* 1968; 58:30–37.

9. Miller Jr WJ, Cooper R: Rising lung cancer death rates among black men: the importance of occupation and social class. *J Natl Med Assoc* 1982; 74:253–258.

10. *The Health Consequences of Smoking: Cardiovascular Disease.* A report of the Surgeon General. DHHS (PHS) Publ No 8-50204. Rockville, MD, Public Health Service, Office on Smoking and Health, 1983.

11. Gillum RF: Coronary heart disease in black populations. I. Mortality and morbidity. *Am Heart J* 1982; 104:839–851.

12. *Health: United States, 1981.* DHHS Publ No (PHS) 82-1232. Hyattsville, MD, National Center for Health Statistics, 1981.

13. Cooper R, Stamler J, Dyer A, Garside D: The decline in mortality from coronary heart disease, USA, 1968–1975. *J Chron Dis* 1978; 31:709–720.

14. Johnson KW, Payne GH (eds): Report of National Heart, Lung and Blood Institute, working conference on coronary heart disease in black populations, and Gillum RF (ed): Proceedings of a symposium on coronary heart disease in black populations. *Am Heart J* 1984; 108:633–862.

15. Community Cardiovascular Surveillance Program: Final report to the National Heart, Lung, and Blood Institute. Baltimore, MD, CCSP Coordinating Center, Department of Epidemiology and Preventive Medicine, University of Maryland, 1984.

16. Goldson A, Henschke U, Leffall LD, Schnieder RL: Is there a genetic basis for the differences in cancer incidence between Afro-Americans and Euro-Americans? *J Natl Med Assoc* 1981; 73:701–706.

17. Devesa SS, Diamond EL: Socioeconomic and racial differences in lung cancer incidence. *Am J Epidemiol* 1983; 118:818–831.

18. Cooper R, Soltero I, Stamler J: Smoking cessation in the Chicago Coronary Prevention Evaluation Program. *J Natl Med Assoc* 1982; 74:349–355.

19. Cooper R, Steinhauer M, Schatzkin A, Miller W: Improved mortality among US blacks, 1968–1978: the role of antiracist struggle. *Int J Health Serv* 1981; 11:511–522.

20. Cooper R: A note on the biologic concept of race and its application in epidemiologic research. *Am Heart J* 1984; 108:715–723.

21. *The Widening Gap: The Incidence and Distribution of Infant Mortality and Low Birth Weight in the United States, 1978–1982.* Washington, DC, Food Research and Action Center, 1984.

CIGARETTE COMPANIES GO TO COLLEGE

On July 25, 1984 the Fortieth Anniversary Gala of the United Negro College Fund (UNCF), representing 43 private, predominantly black colleges, was held in New York City at the world headquarters of Philip Morris, makers of Marlboro and other cigarettes, Miller beer, and 7-Up soda. Hugh Cullman, chairman and chief executive officer of Philip Morris, is a member of the UNCF Board of Directors and served as co-chairman of the 1984 corporate campaign for the UNCF along with the president and chief executive officer of Time, Inc.

On the afternoon of the reception, Christopher Edley, executive director of the UNCF, was asked by the editor of the *Journal,* Alan Blum, MD, if the organization had ever questioned the morality of the acceptance of money from a cigarette producer in light of the disproportionately high rate of lung cancer among blacks.

"This is the first time I've heard of it [the moral issue]. Philip Morris gave to black colleges long before there was a cancer scare. In the case of RJ Reynolds* and Philip Morris, they are located right in the heart of [regions with] black colleges. Philip Morris was originally a Richmond, Virginia company. The Cullman family is largely identified with black colleges. Let's face it. They have p.r. problems they did not have many years ago. Sure they know there's a double purpose to be painted as good guys. But take RJ Reynolds. They've diversified. I'm not even sure a majority of their profits are from tobacco. . . .

"I don't make those strong moral judgments. . . We have been more troubled by [funding from] alcohol [companies] because some of our schools are sponsored by religious denominations."

Asked if he were aware that Philip Morris holds nearly half of the South African cigarette maker, Rothmans, Edley responded, "I'm not surprised. Ford, IBM, and lots of companies that give to us have investments in South Africa. During the civil rights struggle and the rigid segregation against blacks in the South, you never heard anyone in the North saying don't buy from certain companies [that acquiesced in discriminatory policies]. So now we reach 6000 miles across the ocean and speak with impunity. . . .

"We get several hundred thousand dollars a year from cigarette companies. I am not about to lead a crusade to get that money removed. . . .

"It may be 50 years from now, or even 25 years from now when we will look closer to our contributors and question some of them, but for now we won't turn down a reception or a donation."

—**Editor**

* According to *The Charlotte Observer,* Nov 12, 1984, RJ Reynolds marked the 40th anniversary of the UNCF by donating a $250,000 ruby-and-diamond necklace from Tiffany & Co rather than money, as part of the tradition of giving rubies on 40th anniversaries. Actress Ruby Dee made the presentation in Chicago on November 10, 1984 during the intermission of the Ebony Fashion Fair. In 1983 Reynolds pledged $1 million to the UNCF over four years, and the necklace represented the second installment. Overall in 1984 UNCF sought $28 million, including $10 million from corporations.

Pharmacists who dispense cigarettes

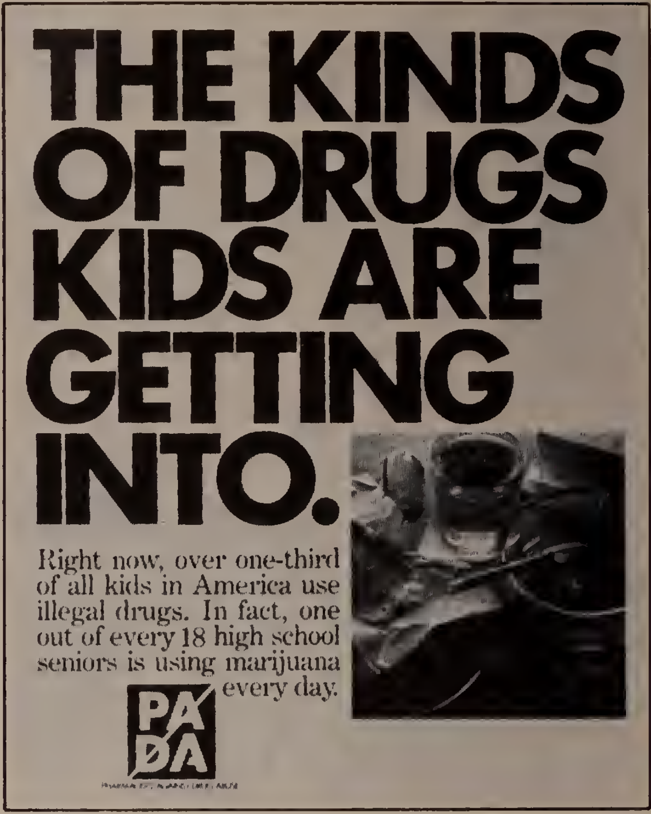
With reference to drug store chains and pharmaceutical companies

JOHN W. RICHARDS, JR, MD, ALAN BLUM, MD

In recent years pharmacists in the United States have attempted to move beyond the role of dispenser of medications and toward that of "gatekeeper of the community's health."[1] In many instances the pharmacy has become a site for screening of high blood pressure, reading of Hemoccult tests, and diabetic monitoring. The president of the California Public Health Association, a pharmacist, has predicted that pharmacists will be reimbursed by third-party payers for providing health care information.[1]

Organizations of retail pharmacies sponsor multiphasic health screening and health fairs, through which abnormal results of blood pressure measurements and laboratory blood and urine profiles stimulate increased utilization of health care services—including pharmacies. Aided by pharmaceutical manufacturers, pharmacies participate in publicity campaigns to curtail such problems as sexually transmitted diseases and accidental poisonings. McNeil Laboratories (makers of Tylenol with codeine) is sponsoring the program "Pharmacists Against Drug Abuse" (Fig 1), and Schering Laboratories has attempted to enhance the image of the pharmacist as a health educator and consumer advocate through pamphlets and mass media advertising that encourage the public to "Ask Your Pharmacist."

Such image-making coincides with increased advertising for numerous analgesics, antacids, vitamins, "diet pills," cough syrups, and "cold remedies," many of which have been cited by the Food and Drug Administration as ineffective and unnecessary. Pharmacy trade journals are replete with advertisements for these preparations reminding pharmacists who display and recommend the drugs to patients that substantial profits are assured.


THE KINDS OF DRUGS KIDS ARE GETTING INTO.
PA
DA

Right now, over one-third of all kids in America use illegal drugs. In fact, one out of every 18 high school seniors is using marijuana every day.

FIGURE 1. Pharmacists Against Drug Abuse: Pharmaceutical company-sponsored organization warns about widespread use of illicit drugs by teenagers.

INNOCENCE BY ASSOCIATION

As questionable as such promotions may be, they pale in comparison to advertisements in pharmacy publications aimed at increasing the sale of tobacco products. These advertisements do not even carry the health warning of the Surgeon General (Fig 2). In pharmacy publications, the role of the pharmacist as tobacconist is seldom if ever questioned, as is illustrated in the following statements from *Drug Store News*, the largest circulation publication of the pharmacy business:

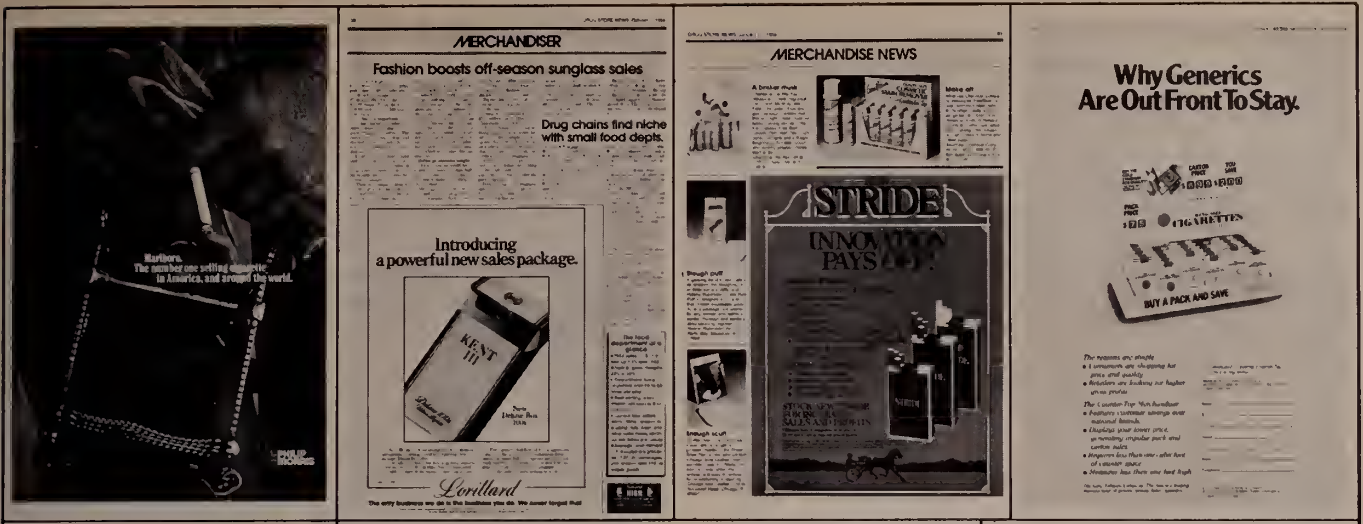
> Considering the increased health warnings about smoking, higher federal, state and local taxes and price hikes on several leading brands, chain drug stores continue to rack up strong cigarette sales.[2]
>
> Drug store retailers also say they'll promote [tobacco] harder and improve in-store displays to counter the decreased consumption due to increased health concerns. Attractive retail display allowances result in "very lucrative" agreements between [tobacco] vendors and retailers.[3]

Twenty-five percent of cigarette purchases are made in pharmacies. Eight percent of chain drugstore profits (and 6.2% of all drug store purchases) are derived from the sale of cigarettes, which ranks fourth among all products sold in pharmacies.[3-5] Moreover, at a time when overall tobacco sales increases have slowed, chain drug store sales of tobacco products increased 9% in 1984 to nearly $2 billion.[5] According to the *United States Tobacco and Candy Journal*, the major trade newspaper of tobacco distributors, cigarettes have become a "staple and stable" product category for the chain drug stores. Cigarettes represent more of an "automatic sale," "volume-builder," and "traffic-builder" than any other single product sold in drug stores. Although cigarette sales are not rising in the United States, cigarettes and drug store chains retain a "top-of-mind" association among consumers: in other words, one of the major reasons for going to the drug store is to buy ciga-

---

From DOC (Doctors Ought to Care).

Address correspondence to Dr Richards, Assistant Professor, Department of Family Medicine, Medical College of Georgia, Augusta, GA 30912.


Marlboro. The number one selling cigarette in America, and around the world.
PHILIP MORRIS
MERCHANDISER
Fashion boosts off-season sunglass sales
Drug chains find niche with small food depts.
Introducing a powerful new sales package.
KENT III
Lorillard
MERCHANDISE NEWS
STRIDE
INNOVATION PAYS OFF
Why Generics Are Out Front To Stay.
CIGARETTES
BUY A PACK AND SAVE

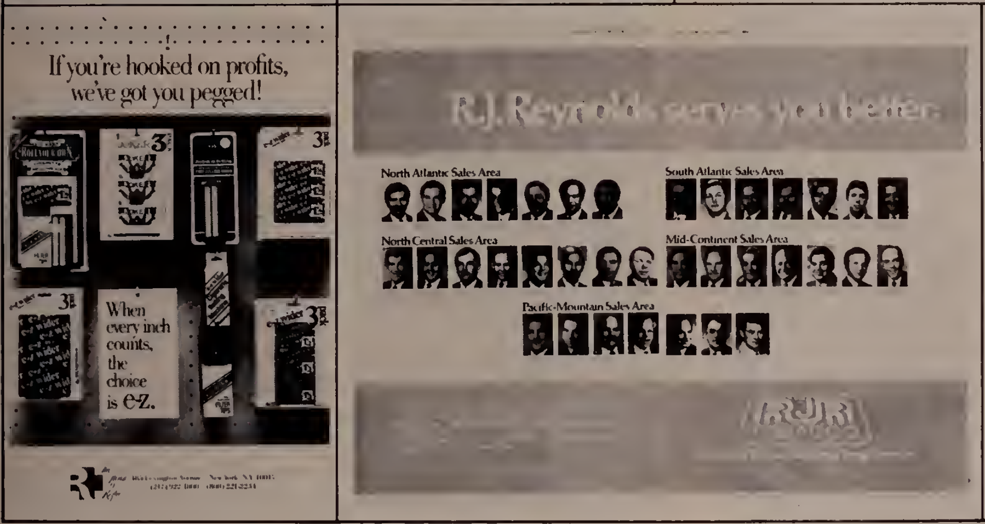

If you're hooked on profits, we've got you pegged!
When every inch counts, the choice is e-z.
RJ Reynolds serves you better
North Atlantic Sales Area
South Atlantic Sales Area
North Central Sales Area
Mid-Continent Sales Area
Pacific-Mountain Sales Area
RJR

FIGURE 2. Examples of pro-smoking advertisements in *Drug Store News*. Top: Ads, minus the Surgeon General's warning, for Marlboro (Philip Morris), Kent III (Loews), and Stride (Grand Metropolitan) suggest greater profits for pharmacists who sell these cigarettes. A generic cigarette manufacturer pushes value and visibility. Bottom: "If you're hooked on profits" slogan promotes rolling papers. RJ Reynolds touts its sales force.

rettes. The most recent survey by the Point-of-Purchase Advertising Institute and Dupont Laboratories found that 70% of cigarette purchases in drug stores were specifically planned.[5]

Marketing strategies of tobacco companies for pharmacy sales are meticulously planned and implemented. Cigarette salesmen pay premiums of $120–$140 per year to pharmacists to place cigarette displays at the cash register or check-out counter.[4] Maximum visibility is reinforced by applying stickers ("PUSH Winston," "It's KOOL Inside," "PULL Marlboro," for example) at child-eye level on doors and shopping carts. Such "point-of-purchase"* messages, changed frequently, encourage customers to believe there are new and improved (and hence possibly more healthful) attributes of one brand or another. Whatever the brand, the point is to buy cigarettes. A major purpose of such cigarette advertisements in pharmacies is to introduce those who do not smoke or who have not yet started to smoke to the acceptability of selling tobacco products in an ostensible health care setting. Who can doubt that patients who smoke rationalize their adverse health behavior by pointing to the sale of cigarettes at the place where they are directed by physicians to obtain medications?

COMMON OBJECTIVES

Many pharmacists doubtless have misgivings about aiding the perpetuation of tobacco use. But in one survey of 100 retail pharmacies in San Francisco, only 11 did not sell cigarettes.[6] And sales of cigarettes in pharmacies appear likely to rise as the trend toward concentration of ownership of pharmacies by large conglomerates increases. In 1983, tobacco sales rose 6% to $2.56 billion.[3] In contrast to their staunch support of various public health measures, no retail pharmacy chain has led a campaign to curtail smoking and its promotion. (Similarly, many retail pharmacy chains have become major discount liquor outlets.) Thus, the definition of drug abuse that many of the pharmacy chains and pharmaceutical manufacturers are supporting refers only to abuse of illicit drugs. Publicity surrounding the widespread abuse of a legal drug—methaqualone or dextroamphetamine, for example—has led to voluntary if belated withdrawal of various products. But the fact remains that the connections between the tobacco and pharmaceutical industries run deep and remain strong. One has only to consider the role of BAT Industries (formerly British American Tobacco), the British parent corporation of Brown and Williamson Tobacco Company (USA), manufacturer of such brands as Kool, Raleigh, and Barclay. In Canada, BAT owns 45% of Imasco (formerly Imperial Tobacco Company), the country's largest cigarette manufacturer. In 1983, Imasco purchased Shoppers' Drug Mart, the largest retail pharmacy chain in Canada, with more than 450 stores. In 1984, Imasco added Peoples Drug Stores, which operates more than 750 outlets in the United States under such names as Sav-A-Lot, Lane, Reed, Lee, Haag, Rae and Derrick, Mill, Health Mart, and Peoples. The company is expected to become the largest retail pharmacy chain in North America.

---

* In October 1984 RJ Reynolds (maker of Camel, Winston, Salem, More, Now, Sterling, Bright, and Vantage cigarettes) received the first Chief Award of the Point-of-Purchase Advertising Institute for its "outstanding and innovative" point-of-purchase program. Pierre Salinger, chief foreign correspondent for ABC News, presented the award to Gerald Long, president and chief executive officer of RJ Reynolds, at a banquet attended by 1,000 business leaders. Long told the gathering, "If a company desires to be a premier consumer products firm, then it must be a leader in point of purchase." (Sources: *Advertising Age*, November 5, 1984, p 28; *United States Tobacco and Candy Journal*, November 29–December 19, 1984, p 40.)

In developing nations, certain pharmaceutical companies are launching major advertising campaigns for drugs not named on the World Health Organization's list of essential drugs; yet these companies have done nothing to discourage cigarette smoking, which has shown a rapid increase due to major advertising campaigns and free sampling by tobacco companies. Certain advertising agencies develop campaigns for both cigarettes and pharmaceutical products, a clear conflict of interest where human health is concerned. A cynical viewpoint would hold that the tobacco companies are paving the way for greater use of medications through the anticipated increase in cigarette-related disease. Cynicism aside, there is evidence of direct ties between the two industries. At least two pharmaceutical companies (Sterling; Johnson & Johnson) advertise products (Bayer aspirin; Tylenol) in tobacco trade journals. The chairman and chief executive officer of CIBA-Geigy (makers of Transderm-nitroglycerin, Esidrix, and Serpasil) serves on the International Advisory Board of RJ Reynolds, one of the world's cigarette manufacturing giants. Merrell Dow (makers of Nicorette) is a major supplier of pesticides for tobacco growing as well as chemical additives used in cigarette manufacture. Pfizer (makers of Minipress, Procardia, and Vibramycin) owns a cigarette flavorings factory. Hoechst (makers of Lasix) manufactures cellophane for cigarette packaging. More than half the revenue of the billboard subsidiary of Minnesota Mining and Manufacturing (3-M), makers of Theolair, surgical masks and the Littman stethoscope, is derived from tobacco company advertising (Fig 3).

In spite of the clear conflict of interest in aiding the sale of the leading preventable risk factor for cancer and a major factor for heart disease, duodenal ulcers, bronchitis, and a host of other ailments, only a handful of American retail pharmacies have refused to sell cigarettes. Indeed, pharmacy journals have only once addressed the ethics of dispensing tobacco products.[7]

PROGRESS IN CANADA

There are hopeful signs, however. In 1984, the Canadian Pharmaceutical Association, the leading pharmacists' society in Canada, launched a health campaign, "Stand Up and Be Counted," aimed at ending tobacco sales in the country's 5,400 drugstores, as well as distributing educational materials on smoking prepared by the Canadian Health Department. Jean-Guy Cyr, editor of the *Canadian Pharmaceutical Journal*, condemned the "harmful and irresponsible act" of selling tobacco products and pointed to the mockery it makes of the health role of the pharmacist.[8] Doubtless aware of the failure of its own policy that pharmacists should not dispense cigarettes, the American Pharmaceutical Association (APA) in 1984 proposed a "guilt-free" solution for pharmacists, whereby

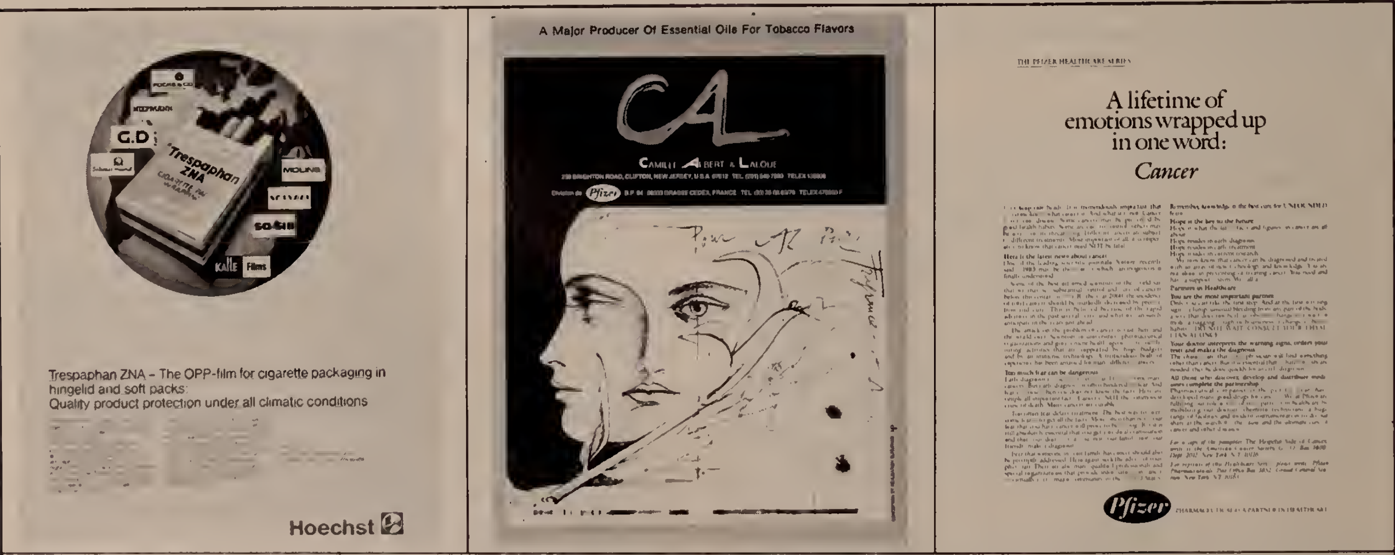

Trespaphan ZNA
Trespaphan ZNA - The OPP-film for cigarette packaging in hingelid and soft packs
Quality product protection under all climatic conditions
Hoechst
A Major Producer Of Essential Oils For Tobacco Flavors
CA
Camilli Albert & Laloue
Pfizer
A lifetime of emotions wrapped up in one word:
Cancer
Pfizer

FIGURE 3. Examples of pharmaceutical company links to the tobacco industry: Hoechst, manufacturer of Lasix and a leader in cigarette packaging; Pfizer owns a tobacco flavoring products company, advertises in tobacco trade publications, and publishes an editorial-style advertisement in major daily newspapers to allay public anxiety about cancer ("Too much fear can be dangerous . . . Hope is the key to the future"). The advertisement implies that substantial control and cure of cancers will be achieved by the year 2000. "Scientists in universities, pharmaceutical organizations and governmental health agencies are coordinating activities that are supported by huge budgets and by an immense technology." No mention of smoking is made—or the number of deaths from cancer that could be prevented simply by not smoking.

Sausalito pharmacist Fred Mayer decided five years ago that ethics outbalanced economics, and opted to drop tobacco and tobacco-related products from his inventory.

"I realized that as a health professional, I was being hypocritical by selling tobacco products," said Mayer. "So I dumped the whole line—pipes, rolling papers, pipe cleaners, the entire thing."

That decision would have cost Mayer $50,000 a year in gross income, had he not hit on a remedy: remerchandising. Mayer redesigned his smoking products section, substituting a line of cameras and films. He had his salesclerks trained in the subject, and what might have been a deficit based on sound principle is instead a better profit margin based on sound economics. . . .

"I don't think I've lost money or customers, and I noticed that when we announced we were throwing tobacco products out, people began to perceive us differently. If a customer comes in and doesn't see the cigarettes and rolling papers, and he or she knows you've gotten rid of them because you felt it's your obligation as a health professional, then the customer begins to perceive you as more of a health professional."

MICHAEL DOLAN
American Pharmacy 1978; 18(9):29

profits from the sale of tobacco products could be donated to the American Cancer Society. Apart from the fact that heart disease and chronic obstructive pulmonary disease account for an even greater overall smoking-related mortality and morbidity than cancer, the APA is shirking responsibility in lieu of playing an active role to curtail smoking and its promotion.

Whereas hospital-based pharmacists have earned a role as respected members of the health care team, retail pharmacists who continue to sell cigarettes do not have a credible claim to the role of health educator or consumer advocate. Unlike their American counterparts (with few exceptions), many Canadian pharmacists have recognized their proper role and are foregoing tobacco sales. Regrettably, efforts by both American and Canadian pharmacists are being undermined by executives of both the tobacco and retail pharmacy industries.

Physicians should support those pharmacists who make a commitment to health promotion and should aid pharmacists in effecting a policy change in their stores. A register of pharmacies that refuse to sell tobacco products should be set up by local medical societies and publicized among their membership. A national registry is being compiled by Paul Fischer, MD, Director of Research, Department of Family Medicine, Medical College of Georgia, Augusta, GA 30912. Physicians should submit resolutions to their local and state medical societies condemning the sale of cigarettes in pharmacies and praising pharmacists (perhaps by giving a plaque or certificate to display) who refuse to sell cigarettes. Pharmacies are the last place in which cigarettes should be sold.

REFERENCES

1. The pharmacist: gatekeeper of the community's health. *Drug Topics,* May 16, 1983:38.

2. Tobacco sales still strong despite warnings, pricing. *Drug Store News,* September 17, 1984:13.

3. *Drug Store News,* March 19, 1984.

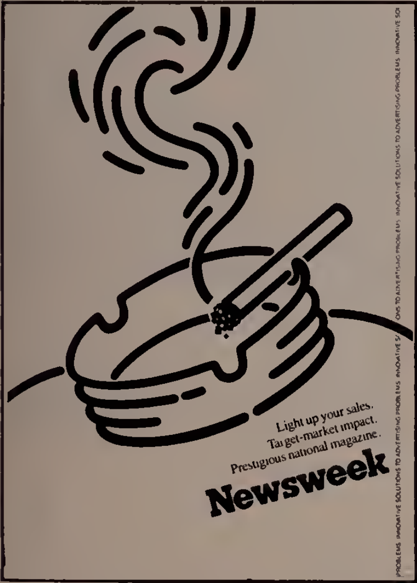
4. "Automatic" cigarette sales a boon to drug chain. *US Tobacco and Candy Journal,* Feb. 11–Mar. 6, 1985:13.

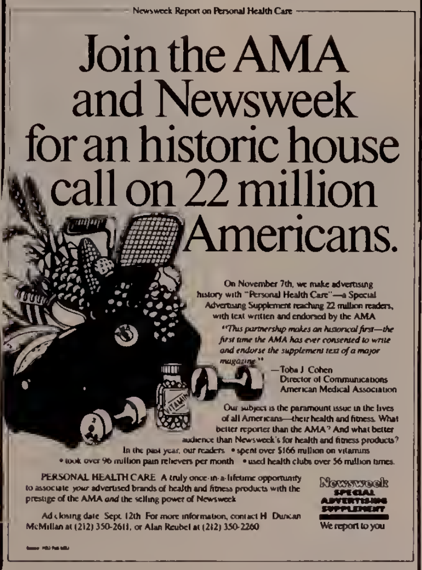
5. 1985 annual report of the chain drug industry: cigarettes garner high sales per sq. ft. *Drug Store News* April 29, 1985:124, 159.

6. Schroeder SA, Showstack JA: Merchandising cigarettes in pharmacies: a San Francisco survey. *Am J Pub Health* 1978; 68:494–495.

7. Dolan M: Caught between ethics and economics: pharmacists and tobacco. *American Pharmacy,* September 1978:28–29.

8. Smith D: Canadian groups target drugstores and print ads in new anti-smoking campaigns. *Advertising Age,* July 9, 1984:38.


Light up your sales.
Target-market impact.
Prestigious national magazine.
Newsweek
PROBLEMS INNOVATIVE SOLUTIONS TO ADVERTISING PROBLEMS INNOVATIVE SOLUTIONS TO ADVERTISING PROBLEMS INNOVATIVE SOLUTIONS TO ADVERTISING PROBLEMS INNOVATIVE SO


Newsweek Report on Personal Health Care

Join the AMA and Newsweek for an historic house call on 22 million Americans.

On November 7th, we make advertising history with "Personal Health Care"—a Special Advertising Supplement reaching 22 million readers, with text written and endorsed by the AMA.

"This partnership makes an historical first—the first time the AMA has ever consented to write and endorse the supplement text of a major magazine."

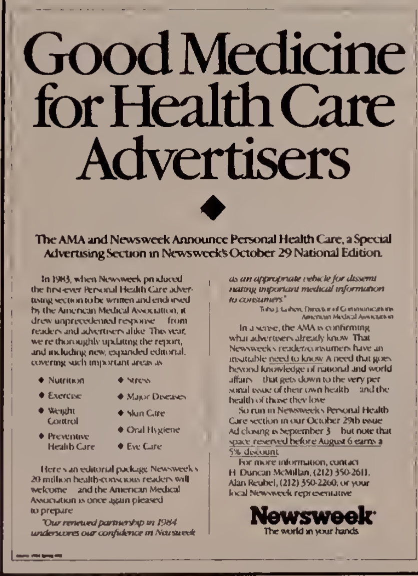
—Toba J. Cohen
Director of Communications
American Medical Association

Our subject is the paramount issue in the lives of all Americans—their health and fitness. What better reporter than the AMA? And what better audience than Newsweek's for health and fitness products? In the past year, our readers • spent over $166 million on vitamins • took over 96 million pain relievers per month • used health clubs over 56 million times.

PERSONAL HEALTH CARE. A truly once-in-a-lifetime opportunity to associate *your* advertised brands of health and fitness products with the prestige of the AMA *and* the selling power of Newsweek.

Ad closing date: Sept. 12th. For more information, contact H. Duncan McMillan at (212) 350-2611, or Alan Reubel at (212) 350-2260.

Newsweek
SPECIAL ADVERTISING SUPPLEMENT
We report to you


Good Medicine for Health Care Advertisers

◆

The AMA and Newsweek Announce Personal Health Care, a Special Advertising Section in Newsweek's October 29 National Edition.

In 1983, when Newsweek produced the first-ever Personal Health Care advertising section to be written and endorsed by the American Medical Association, it drew unprecedented response — from readers and advertisers alike. This year, we're thoroughly updating the report, and including new, expanded editorial, covering such important areas as:

- Nutrition
- Exercise
- Weight Control
- Preventive Health Care
- Stress
- Major Diseases
- Skin Care
- Oral Hygiene
- Eye Care

Here's an editorial package Newsweek's 20 million health-conscious readers will welcome — and the American Medical Association is once again pleased to prepare.

"Our renewed partnership in 1984 underscores our confidence in Newsweek as an appropriate vehicle for disseminating important medical information to consumers."

Toba J. Cohen, Director of Communications
American Medical Association

In a sense, the AMA is confirming what advertisers already know. That Newsweek's reader/consumers have an insatiable need to know. A need that goes beyond knowledge of national and world affairs — that gets down to the very personal issue of their own health — and the health of those they love.

So run in Newsweek's Personal Health Care section in our October 29th issue. Ad closing is September 3 — but note that space reserved before August 6 earns a 5% discount.

For more information, contact H. Duncan McMillan, (212) 350-2611, Alan Reubel, (212) 350-2260, or your local Newsweek representative.

Newsweek
The world in your hands.

Newsweek (owned by the Washington Post Co) has advertised itself in *The US Tobacco and Candy Journal* and other tobacco trade publications as a leading vehicle for lighting up worldwide sales of cigarettes (left). At the same time, the magazine has attempted to portray itself as a reliable source of information on health. In special issues in 1983 and 1984, *Newsweek* used the prestige of the American Medical Association to lend credibility to advertisers. Both issues contained numerous cigarette advertisements. There was scant mention of smoking in otherwise detailed advice on personal health care.

Smoking and the military

GREGORY H. BLAKE, MD

Many significant medical advances can be attributed to the efforts of military medical personnel. The Continental Army was the first entire army to be immunized against contagious disease. Military physicians demonstrated that mosquitos were carriers of yellow fever, and developed anhydrous chlorine to purify drinking water. More recently, military physicians developed a polysaccharide vaccine against group C meningococcus.

With an impressive history of research and development in preventive medicine, the American military should be a leader in the area of improved health care and fitness. Recent efforts have been made to enhance the health and well-being of American military personnel, attesting to the strong commitment of the armed forces to improve health care. A program has been instituted that requires service personnel 25 years of age or older with risk factors for cardiovascular disease to participate in patient education programs. "Happy Hours" have been abolished at military clubs, and breath alcohol analyzers have been installed in an effort to curb alcohol consumption and alcohol-related accidents and injuries. Structured physical exercise programs and weight guidelines have been established. These programs all point to a commitment to preventive medicine by the military, but there is much more to be done.

One area in which the military could have a profound impact on the general wellness of its personnel is that of cigarette smoking. Unfortunately, many health care providers and the population in general still believe that chronic obstructive pulmonary disease and lung cancer are the sole adverse effects of cigarette smoking, and that morbidity and mortality occur only over many years.

During the past decade, considerable evidence has come to light to alter this thesis. Small airway dysfunction has been reported in male smokers aged 18 to 25 years.[1] Pulmonary function tests have shown a decreased forced expiratory volume in one second, and maximal expiratory flow rates at 50% and 25% of vital capacity in smokers aged 15 to 24 years.[2] Smoking causes inhibition of the cleansing ciliary motion in the lungs and depression of immune response, which result in increased risk of respiratory infection.

Research in the military population supports these findings. In a study of 3,049 white male naval recruits aged 18 to 29 years, peak expiratory flow rates were higher in nonsmokers than in smokers.[3] Preliminary unpublished studies (Blake GH, Stanley WG, Shy C) of recruits undergoing basic combat training suggest a statistically significant relationship between smoking and upper respiratory infection. Further, the results showed that more convalescent time for respiratory ailments was required by smokers than by nonsmokers. This study involved 887 basic combat training graduates with an average age of 21 years, 50.1% of whom smoked cigarettes. Of the smokers in the group, 76% began smoking before age 16. In another unpublished study of recruits admitted to an acute respiratory disease ward, 72% said they smoked at least a half a pack of cigarettes per day; bronchitis and lower respiratory tract infection were diagnosed three times more often in smokers than in nonsmokers.[4]

Chesterfield
CIGARETTES
"Gee! I'm in luck"
A cigarette of the finest TURKISH and DOMESTIC tobaccos-*blended*.
And the blend can't be copied.
they "Satisfy"
Liggett & Myers Tobacco Co.

LIFE 1919

These findings have widespread implications for military training programs and combat readiness, as well as for the general health and well-being of military personnel.

The smoking problem is magnified by the prevailing attitude in the military toward tobacco. Tobacco products are prominently displayed in military commissaries; ciga-

---

Dr Blake, Major, Medical Corps, served on active duty from 1977 to 1984.

Address correspondence to Dr Blake, Assistant Professor, Department of Family Medicine, University of Oklahoma College of Medicine, PO Box 26901, Oklahoma City OK 73190.

The opinions or assertions contained herein are the private views of the author and are not to be considered as reflecting the views of the Department of the Army or the Department of Defense.

rettes sell for 26% to 42% less than in civilian stores, so there is scant financial incentive to stop smoking or curtail usage.[5] Service personnel may even have a more difficult time not smoking than civilians, because smoking is built in to many aspects of military life. Smokers may be allowed extra breaks from work assignments, while the nonsmoker continues working. Smoking habits are reinforced by the number of enlisted personnel who smoke and supervise training and work details. Role models play an important part in military life. Enlisted personnel and officers frequently emulate superiors they admire. If a respected superior smokes, the aspiring serviceman or servicewoman will likely continue to smoke, or even begin smoking.

The link between the tobacco industry and the military is old and enduring. Cigarettes have been a part of the K-rations and C-rations provided to soldiers and sailors. Campaigns led by the tobacco industry sought to send cigarettes as morale boosters to the troops fighting overseas in both world wars.

During World War II, cigarette advertisements praising America's servicemen and servicewomen were heard daily on the leading radio programs and appeared in most periodicals. Today there continues to be an attempt by the tobacco industry to link patriotism, toughness, and military service with smoking. *Army Times, Air Force Times,* and *Navy Times*—privately owned publications which report topics of interest to military personnel—publish numerous cigarette advertisements and have seldom addressed the subject of smoking. The Park Avenue Tobacco Company has announced plans to market a "1776" brand cigarette, advertising for which is targeted to military personnel.[6] The pack features an embossed American flag.

Because peer pressure in a military setting influences the initiation and perpetuation of tobacco use, it is imperative that the military's attitude toward smoking be changed. The "macho" image of the hard-charging, hard-drinking, cigarette-smoking soldier should be changed to one of the hard-charging, physically fit, non-smoking soldier. This can be accomplished in several ways.

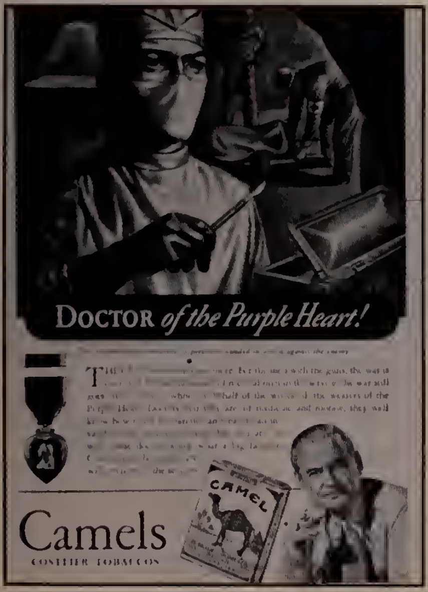
First, smoking cessation techniques can be implemented in basic combat training while soldiers are being acclimated to military service. Second, health promotion topics, including the hazards of tobacco use, should be presented regularly to officers and enlisted personnel participating in formal military education classes. Third, military hospitals should enforce smoking regulations and sponsor smoking cessation classes for military personnel and their dependents. Similar recommendations were made in an editorial in *Military Medicine* in 1977.[4]

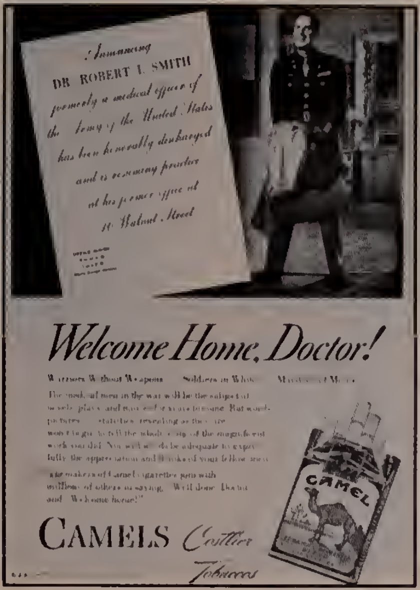
The message is being heard. In June 1983 a conference on health promotion convened by the Department of Defense addressed the challenge of reducing smoking in the military. Among other proposals, the conferees called for an increase in the cost of cigarettes sold in every commissary and the removal of all cigarette vending machines from military bases. Assistant Defense Secretary for Health Affairs William E. Mayer, an outspoken opponent of cigarette smoking, should be encouraged and supported in his efforts to implement stronger measures.

Lastly, the military should modify its current health care manpower allocation system. At present, the greater the number of patient contacts related to illness per department, the greater the allocation of resources to that department. No credit is given for preventive medicine activities. By providing increased resources for those departments that stress preventive medicine, the military medical care establishment can begin to shift its focus from morbidity to wellness and the promotion of healthy lifestyles among American military personnel.

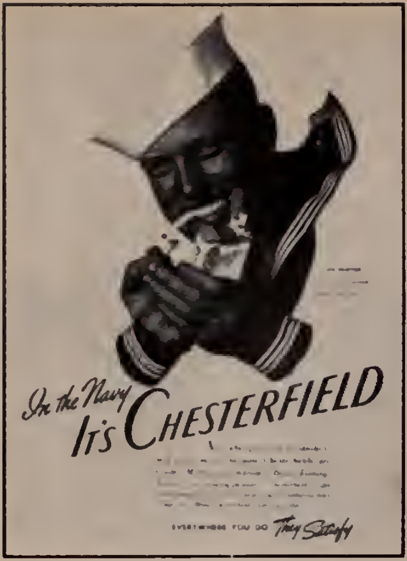
REFERENCES

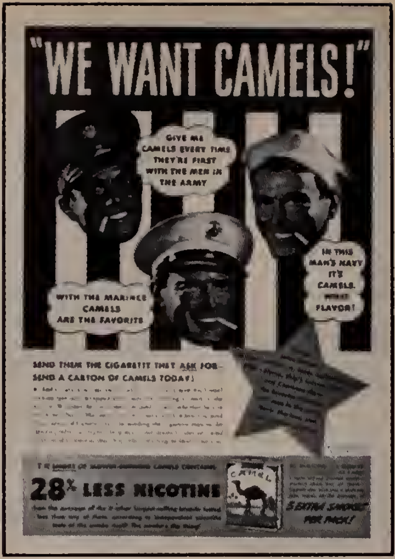
1. Enjeti S, Hazelwood B, Permutt S, Menkes H, Terry P: Pulmonary function in young smokers: male-female differences. *Am Rev Respir Dis* 1978; 118:667–676.
2. Beck GJ, Doyle CA, Schachter EN: Smoking and lung function. *Am Rev Respir Dis* 1981; 123:149–155.
3. Comstock GW, Rust PF: Residence and peak expiratory flow rates among Navy recruits. *Am J Epidemiol* 1973; 98:348–354.
4. John JF Jr: Smoking, the soldier, and the army. (editorial) *Milit Med* 1977; 142:397–398.
5. Smith P: "Services must stop aiding military smoking," major says. *Navy Times*, 1984.
6. Hall T: Smokers in military are firm's market for 1776 cigarette. *Wall Street Journal*, February 12, 1985, p 1.

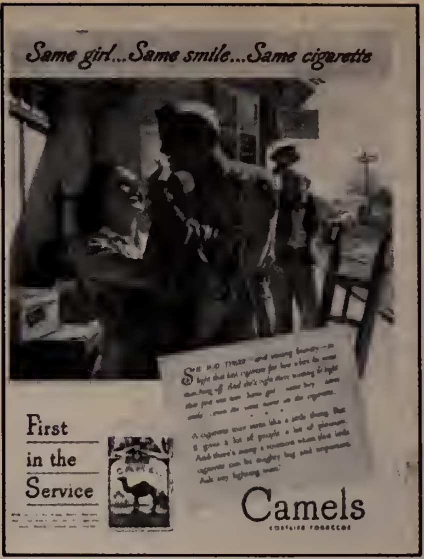


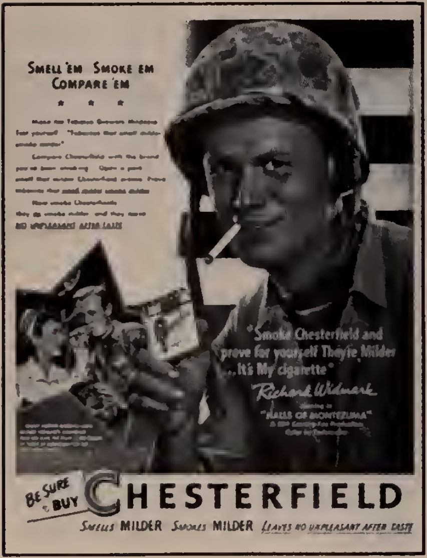


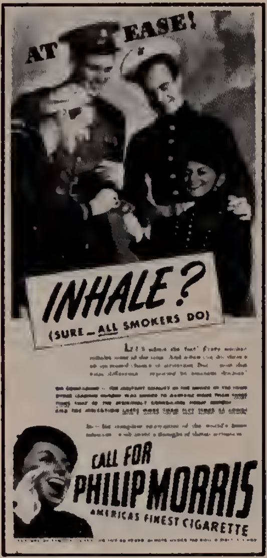
Post-war cigarette advertisements by RJ Reynolds in the *New York State Journal of Medicine* in 1945 praised "doctors of medicine and morale." (See also *NY State J Med* 1983; 83:1347–1352.)


In the Navy
It's CHESTERFIELD
EVERYWHERE YOU GO They Satisfy

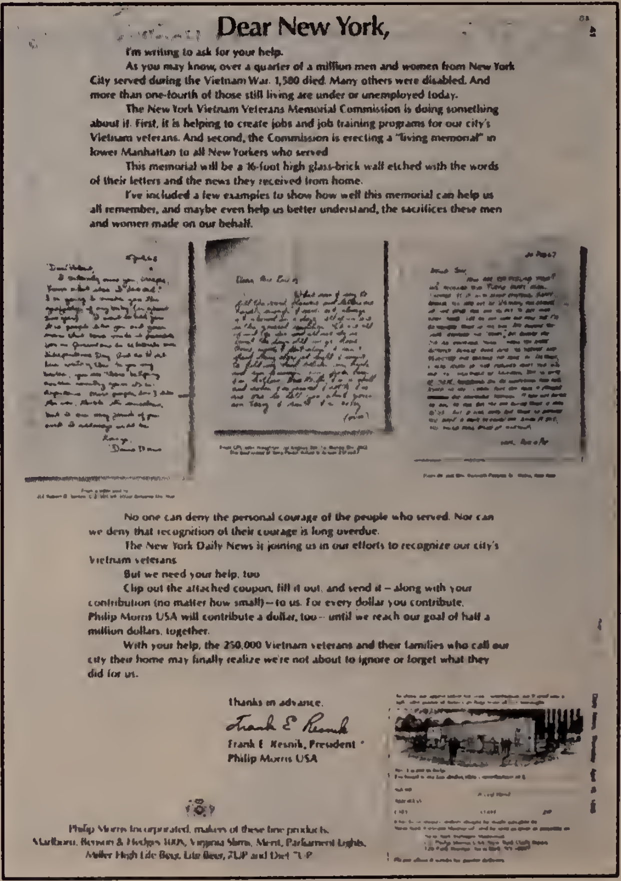

"WE WANT CAMELS!"
GIVE ME CAMELS EVERY TIME THEY'RE FIRST WITH THE MEN IN THE ARMY
WITH THE MARINES CAMELS ARE THE FAVORITE
IN THIS MAN'S NAVY IT'S CAMELS. FLAVOR!
SEND THEM THE CIGARETTE THEY ASK FOR—SEND A CARTON OF CAMELS TODAY!
28% LESS NICOTINE
5 EXTRA SMOKES PER PACK!




SMELL 'EM SMOKE 'EM COMPARE 'EM
"Smoke Chesterfield and prove for yourself They're Milder ...It's My cigarette"
Richard Widmark
"HALLS OF MONTEZUMA"
BE SURE TO BUY CHESTERFIELD
SMELLS MILDER SMOKES MILDER LEAVES NO UNPLEASANT AFTER TASTE



Throughout the 20th century, whenever American soldiers have gone to war, cigarette manufacturers have remained behind to promote the image of smoking as patriotic and manly in magazines, movies, and on radio and television. In 1941 RJ Reynolds advertised that men in all branches of the service clamored, "We want Camels!" a claim that could be verified over the past 25 years at any Veterans Administration hospital. One of the most recent campaigns by a tobacco company to associate its cigarette brands with the military was Philip Morris' fund-raising drive for a New York Vietnam War Memorial (below). Advertisements placed in the *Daily News* for several weeks prior to a large Vietnam veterans parade named the *News* as co-sponsor of the drive and listed the various cigarette brands made by Philip Morris.



Dear New York,

I'm writing to ask for your help.

As you may know, over a quarter of a million men and women from New York City served during the Vietnam War. 1,500 died. Many others were disabled. And more than one-fourth of those still living are under or unemployed today.

The New York Vietnam Veterans Memorial Commission is doing something about it. First, it is helping to create jobs and job training programs for our city's Vietnam veterans. And second, the Commission is erecting a "living memorial" in lower Manhattan to all New Yorkers who served.

This memorial will be a 16-foot high glass-brick wall etched with the words of their letters and the news they received from home.

I've included a few examples to show how well this memorial can help us all remember, and maybe even help us better understand, the sacrifices these men and women made on our behalf.

No one can deny the personal courage of the people who served. Nor can we deny that recognition of their courage is long overdue.

The New York Daily News is joining us in our efforts to recognize our city's Vietnam veterans.

But we need your help, too.

Clip out the attached coupon, fill it out, and send it—along with your contribution (no matter how small)—to us. For every dollar you contribute, Philip Morris USA will contribute a dollar, too—until we reach our goal of half a million dollars, together.

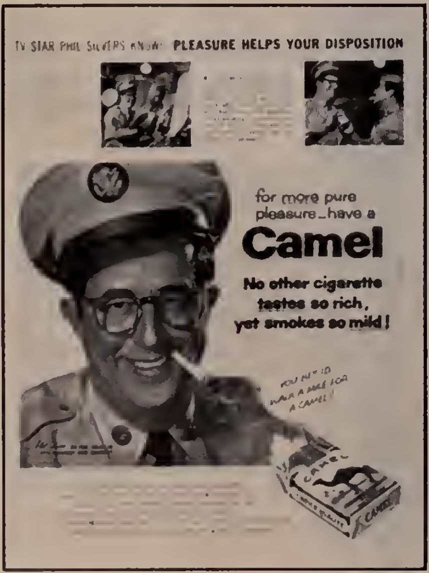
With your help, the 250,000 Vietnam veterans and their families who call our city their home may finally realize we're not about to ignore or forget what they did for us.

Thanks in advance.

Frank E. Resnik

Frank E. Resnik, President
Philip Morris USA

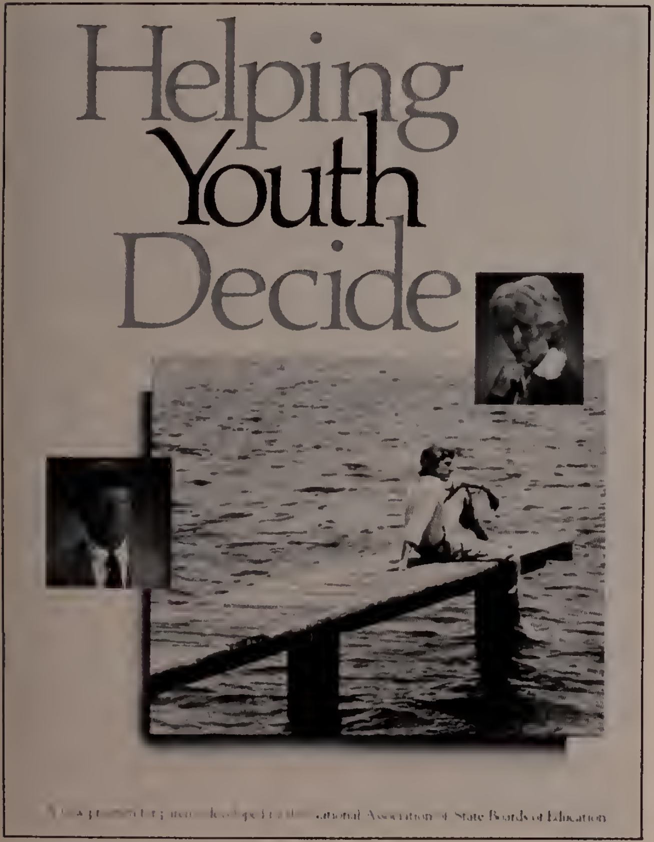
Philip Morris Incorporated, makers of these fine products:
Marlboro, Benson & Hedges 100's, Virginia Slims, Merit, Parliament Lights,
Miller High Life Beer, Lite Beer, 7UP and Diet 7UP


TV STAR PHIL SILVERS KNOWS PLEASURE HELPS YOUR DISPOSITION
for more pure pleasure—have a Camel
No other cigarette tastes so rich, yet smokes so mild!
YOU NEED WALK A MILE FOR A CAMEL!

Helping Youth Decide:
"When the fox preaches, beware the geese"

W. R. COULSON, PHD, EDD

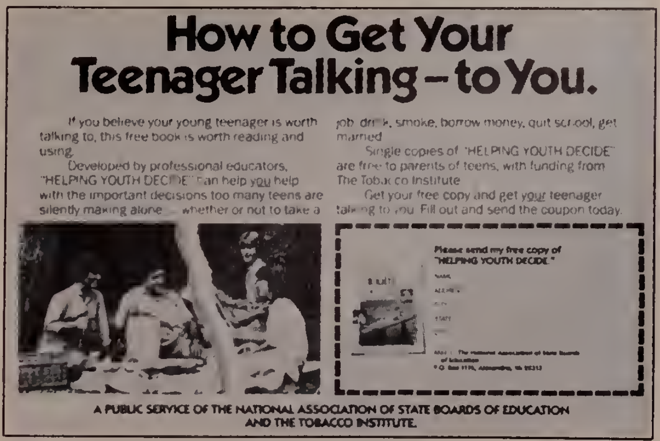
Helping Youth Decide is a 20-page booklet with color illustrations, made available free to parents with funds provided by the Tobacco Institute. It purports to teach good parent-teenager communication. Released in the fall of 1984, its mode of distribution was unusual for a booklet published by a non-profit educational group (the National Association of State Boards of Education). On its behalf advertisements were placed in such national magazines as *Better Homes & Gardens, Ladies Home Journal, People, Sports Illustrated, Time,* and *TV Guide* and in such major newspapers as *The Wall Street Journal* and *The New York Times.* At a press conference in Washington, DC, Tobacco Institute Vice-President Walker Merryman told of his organization's enthusiasm for the project: "To date we have avoided *encouraging* youngsters to smoke. This effort should actively *discourage* youth smoking." [1] He spoke of his industry's "open-ended commitment" to *Helping Youth Decide,* a commitment confirmed when additional materials were scheduled for 1985 release, including discussion leader guides and translation into Spanish. By May, 350,000 copies of the booklet were in print.



If Merryman's judgment is correct—that the campaign should discourage youth smoking—major changes in industry demographics and profits will ensue. Studies of age of initiation of smoking indicate that most adults who smoke began in their teens. For example, a 1975 survey by the National Clearinghouse for Smoking and Health found that 76.22% of Americans who smoke or formerly smoked took up smoking by age 20. Only 11.28% began smoking after age 24.[2] (These percentages are conservative, since they do not include the four million Americans under 21 who smoked at the time [personal communication, Shopland DR, Technical Information Officer, Office on Smoking and Health, US Public Health Service, Feb 19, 1985].)

Merryman pointed out that it has been industry policy "for many years" to hold that "smoking is an adult custom." Indeed, the by-now-familiar slogan is featured in a brief preface to *Helping Youth Decide.* Otherwise smoking goes unmentioned except in this account of the "many new decisions" teenagers face: "Some decisions will be small, others important. Consider the following examples: whether to drive the car, to drink, to smoke, to borrow money, to quit school, to take or quit a job, to marry." [3] So, while the booklet may be sincere in its intention to discourage smoking, it is not about smoking. Rather, it is about what mothers and fathers can do to help their teenagers develop "decision making skills." To this end *Helping Youth Decide* teaches parents to set aside their instincts—"threatening . . . preaching . . . blaming . . . analyzing . . . pacifying . . . probing . . . avoiding"—in favor of the methods of non-directive psychotherapy.

It was psychologist Thomas Gordon who first tested the



How to Get Your Teenager Talking – to You.

If you believe your young teenager is worth talking to, this free book is worth reading and using.

Developed by professional educators, "HELPING YOUTH DECIDE" can help you help with the important decisions too many teens are silently making alone — whether or not to take a job, drink, smoke, borrow money, quit school, get married.

Single copies of "HELPING YOUTH DECIDE" are free to parents of teens, with funding from The Tobacco Institute.

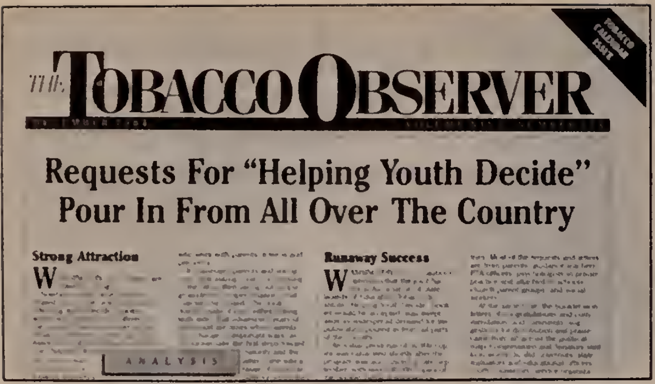
Get your free copy and get your teenager talking to you. Fill out and send the coupon today.

Please send my free copy of "HELPING YOUTH DECIDE."

A PUBLIC SERVICE OF THE NATIONAL ASSOCIATION OF STATE BOARDS OF EDUCATION AND THE TOBACCO INSTITUTE.

Dr Coulson is a professor of psychology and education at United States International University, San Diego. In 1978 he founded Project Mozart, a non-profit organization that attempts to restore authoritative parenting to families, particularly on matters of health.

Address correspondence to Dr Coulson, Center for Enterprising Families, PO Box 8337, La Jolla, CA 92038.



THE TOBACCO OBSERVER

Requests For "Helping Youth Decide" Pour In From All Over The Country

Strong Attraction

Runaway Success

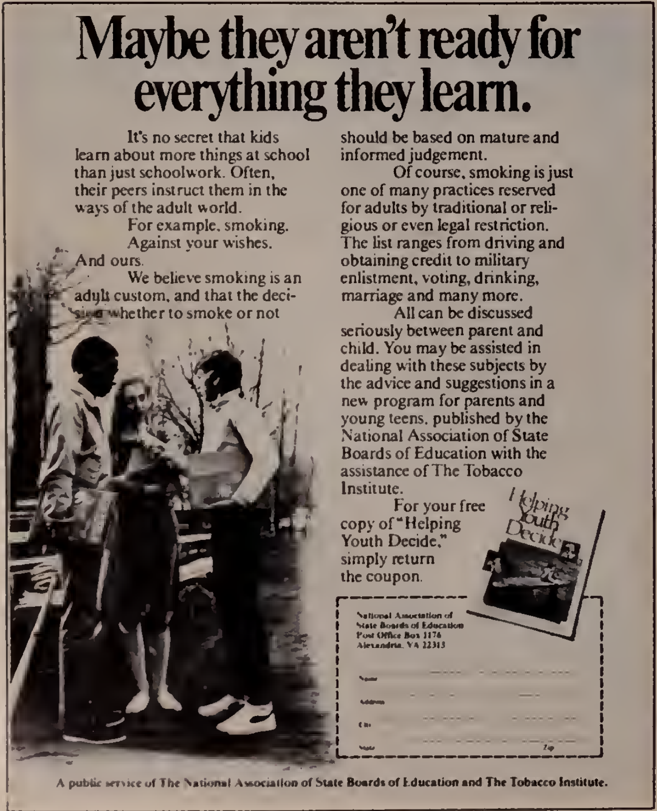
ANALYSIS

The Tobacco Institute's *Tobacco Observer* reported the enthusiastic support of a spokesman for New York Governor Mario Cuomo, as well as two other governors, for *Helping Youth Decide*. In January 1984 Governor Cuomo announced the appointment of George Weissman, chairman of the board and chief executive officer of cigarette maker Philip Morris Inc, as special advisor to the governor on New York's efforts to attract new business.

In June 1983, the head of the White House Office on Drug Abuse Policy, Carlton Turner, was quoted in the *Tobacco Observer* as praising the Tobacco Institute's efforts to curtail smoking among teenagers.

idea that the "acceptance, understanding, and permissiveness . . . of the clinic" [4]—techniques developed by Carl Rogers and his associates in the late 40's and early 50's at the University of Chicago [5]—could be extended profitably to extra-clinical settings. Gordon's numerous books and articles are the major influence on the burgeoning "parenting" movement, including *Helping Youth Decide.* (Gordon's work is uncredited—no sources are referenced—but staff of the National Association of State Boards of Education, the corporate author, privately acknowledge their debt to him.)

Gordon found, however, that an important class of problem behaviors would not yield to his recommended methods, and it may cause embarrassment at the Tobacco Institute to learn that he offers smoking as the foremost example. Besides smoking, his list includes premarital sex, marijuana use, and neglecting homework. He writes, "It is understandable that many parents feel so strongly about certain behaviors that they do not want to give up trying to influence their children, but a more objective view usually convinces them that they have no other feasible alternative except to give up—to accept what they cannot change." He adds, "I often tell parents, 'Don't want your child to become something in particular; just want him to become.'" [6] One can see the appeal of this system to any marketer interested in young people's becoming customers, and it is unfortunate that the Tobacco Institute, concerned about discouraging youth smoking, has stumbled into lending support to an inapposite method.



Maybe they aren't ready for everything they learn.

It's no secret that kids learn about more things at school than just schoolwork. Often, their peers instruct them in the ways of the adult world.

For example, smoking.

Against your wishes.

And ours.

We believe smoking is an adult custom, and that the decision whether to smoke or not should be based on mature and informed judgement.

Of course, smoking is just one of many practices reserved for adults by traditional or religious or even legal restriction. The list ranges from driving and obtaining credit to military enlistment, voting, drinking, marriage and many more.

All can be discussed seriously between parent and child. You may be assisted in dealing with these subjects by the advice and suggestions in a new program for parents and young teens, published by the National Association of State Boards of Education with the assistance of The Tobacco Institute.

For your free copy of "Helping Youth Decide," simply return the coupon.

National Association of
State Boards of Education
Post Office Box 1176
Alexandria, VA 22313

Name
Address
City
State Zip

A public service of The National Association of State Boards of Education and The Tobacco Institute.

On the other hand, it is possible that the English proverb applies: "When the fox preaches, beware the geese."

What would be an effective anti-smoking campaign involving the parents of teenagers? There are clues in recent studies headed by Judith Brook and by Douglas Wilson, MD. Brook's team studied the qualities and strategies of the relationship between fathers and sons, comparing youngsters who smoked to those who did not.[7] Whether the father smoked was not significant, either as a main effect or an interaction. What was significant was that fathers of boys who abstained were "intolerant of tobacco and marijuana use." They had "high expectations for their sons." Neither characteristic is consistent with passive parenting. In the study by Wilson et al, 211 patients were counseled to stop smoking; 106 received follow-up sessions, the balance did not. "From the patients' reports 6 to 12 months after entry into the trial we found that, overall, only 12% of the patients in the control group but 23% of those in the experimental group had quit smoking. . . . [T]he proportion of patients who quit smoking was about five times as great in the experimental group as in the control group among those who smoked regular-tar cigarettes . . ." [8]

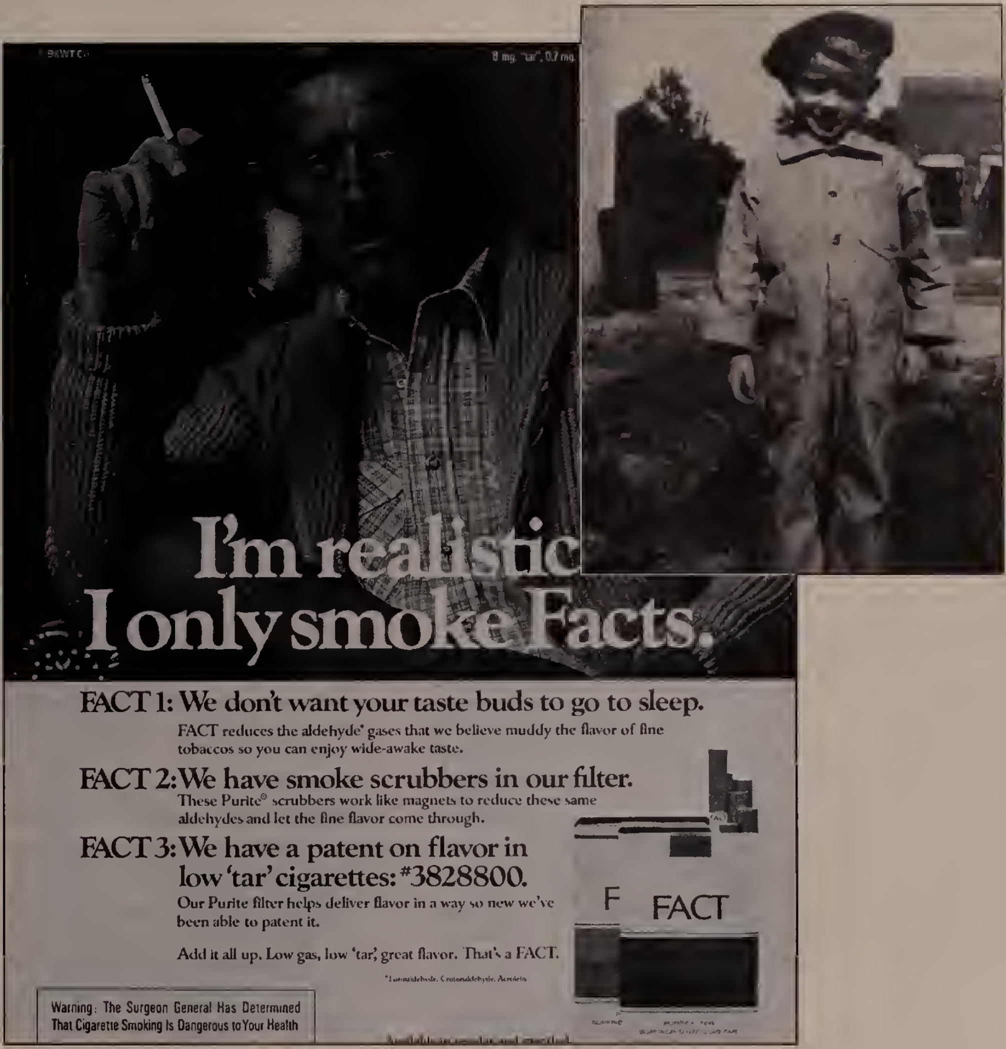
Follow-up, which differentiated the successful from the unsuccessful cases, is nothing other than a form of repetition—preaching, if you will. Condemned for parents in *Helping Youth Decide,* it is nonetheless essential to successful tobacco advertising—and, it seems, health education. If they are to retain their self-respect, parents might look to advertising practices and patient education for parenting tips—rather than to psychotherapy.

REFERENCES

1. Educators and Tobacco Institute cooperate to launch joint "Helping Youth Decide" program. *Tobacco Observer.* 1984; 9:1.
2. *Adult Use of Tobacco,* 1975. Bureau of Health Education, Center for Disease Control, US Department of Health, Education, and Welfare, 1975. Table IV-1.
3. *Helping Youth Decide.* The booklet is available free from the National Association of State Boards of Education, PO Box 1176, Alexandria, VA 22313.
4. Gordon T: Group-centered leadership and administration, in Rogers, CR: *Client-Centered Therapy.* Boston, Houghton Mifflin, 1951, p 320.
5. Gordon T: Crippling our children with discipline. *J Education* 1981:163.
6. Gordon T: *Parent Effectiveness Training.* New York, Wyden, 1970, pp 278, 288.
7. Brook JS, Whiteman M, Gordon AS, Brook DW: Fathers and sons: their relationship and personality characteristics associated with the son's smoking behavior. *J Genetic Psychol* 1983; 142:271-281.
8. Wilson D, Wood G, Johnston N, Sicurella J: Randomized clinical trial of supportive follow-up for cigarette smokers in a family practice. *Can Med Assoc J* 1982; 126:127-129.

IN MEMORY

The last cigarette advertisement



FACT 1: We don't want your taste buds to go to sleep.
FACT reduces the aldehyde* gases that we believe muddy the flavor of fine tobaccos so you can enjoy wide-awake taste.

FACT 2: We have smoke scrubbers in our filter.
These Purite® scrubbers work like magnets to reduce these same aldehydes and let the fine flavor come through.

FACT 3: We have a patent on flavor in low 'tar' cigarettes: #3828800.
Our Purite filter helps deliver flavor in a way so new we've been able to patent it.

Add it all up. Low gas, low 'tar', great flavor. That's a FACT.

*Formaldehyde, Crotonaldehyde, Acrolein

Warning: The Surgeon General Has Determined That Cigarette Smoking Is Dangerous to Your Health

Two images of Dave Morris.

What better man than Dave Morris as the model in a national advertising campaign for a new brand of cigarettes—Fact? His commanding good looks and ever-present cigarette made the perfect image on thousands of billboards, in hundreds of newspapers like *The New York Times* and *The Washington Post*, and in dozens of magazines like *Sports Illustrated* and *Playboy*.

Early in 1984, less than eight years after Brown & Williamson Tobacco Company launched this $30 million educational effort for its brand of facts, Dave Morris' legs became paralyzed. The diagnosis was lung cancer that had spread to his brain and spine. He never left the hospital, and in 40 days he was dead.

All the geniuses who think up images for cigarette advertisements could never recreate those final 40 days—a lifetime to the victim and the family: sharing the pain, loneliness, and isolation; watching the rapid deterioration, the loss of functions, the loss of dignity.

The survivor tries to remember only the wonderful times and the images of the sweet-faced boy and the vigorous man in the cigarette advertisement. But this is overwhelmed by the last picture from real life: an emerging skeleton.

To the physician who is familiar with this scenario but has not taken a stand, think about the last picture a family remembers, and how it might have been prevented.

Then be realistic. Tell all your patients and their families the facts about smoking and the people who still push it to sweet-faced boys and girls.

JOYCE H. PORTER
6625 N. 14th St
Phoenix, AZ 85014

Obstacles to the prevention of teenage smoking

Joseph A. Califano, Jr served as Secretary of Health Education and Welfare in the Carter administration during its first 30 months. Regarded as one of the most innovative leaders ever appointed to the highest health post, he earned the enmity of the tobacco industry because of the smoking and health programs he sought to implement. The tobacco industry went so far as to finance bumper stickers that read "Califano Is Dangerous To My Health." President Carter asked for his resignation on July 18, 1979. Mr. Califano tells the story of his years with presidents Kennedy, Johnson, and Carter in Governing America—An Insider's Report from the White House and the Cabinet *(New York, Simon and Schuster). In response to a request from* Journal *editor Alan Blum, MD, Mr Califano agreed to discuss the subject of smoking and its prevention. The following dialogue occurred on November 20, 1984 in Mr Califano's law office in Washington, DC.*

Alan Blum, MD: Did President Carter cut your legs out from under you on the cigarette issue or is that too strong a term?

Joseph A. Califano, Jr: He never directly told me not to go forward with the cigarette program but he clearly regarded it as an enormous political liability. Dr Peter Bourne, who was his health advisor, clearly wanted to temper the program. I think in a funny way President Carter got hoist by his own petard. He wanted a health promotion/disease prevention program. He told me that when we first talked about the job. Every doctor I interviewed for the top medical posts at HEW—over 100—said without exception we had to move on cigarette smoking, the number one killer and the number one cause of disease and disability.

AB: Did you ask the AMA for nominees?

JC: No, but they also agreed on this point. I then decided to move against cigarettes, but I didn't know how. This action, incidentally, had nothing to do with the fact I had quit smoking in 1975. I never even thought of the smoking campaign when I went to HEW. We looked at two things. First 90% of smokers had tried to quit in the past year. They may have gotten up in the morning and said, "I want to quit smoking," but only got as far as their first cup of coffee. Or they may have given it up for a day or a week. Or maybe they actually quit. But the point is they had tried in the previous 12 months. Second, 75% of the adults hooked on cigarettes were addicted before they were 21. This information came from surveys done by the Office on Smoking and Health.

AB: In your book you talk about constantly fighting the label "ex-smoker."

JC: Yes, because it became an issue. Tobacco interests kept charging that I was a zealous ex-smoker.

AB: In 1985, Jimmy Carter is going to make smoking one of his major priorities in his Carter Center. Did you know that?

JC: No, I did not, but that certainly is fascinating information in view of his administration's record on the tobacco industry. It has been a cold political issue from his point of view.

AB: You are the only senior government official and the only HEW secretary not only to propose anti-smoking campaigns but also to condemn cigarette advertising. What led you to do that?

JC: The problem of cigarette advertising is that it is not directed at 35-year-old men and women who decide to give up at least five years of their lives and greatly increase the danger they will get cancer, heart disease, and emphysema. Cigarette advertising is largely directed at making smoking a glamorous activity for teenagers. That came out in the Federal Trade Commission Report in 1981 spearheaded by Mike Pertschuk, but the tremendous fight of the cigarette companies prevented the FTC from disclosing any of their advertising strategy. Their fight demonstrated just how important their strategies are. Tobacco advertising is a billion and a half dollar a year industry, but I had less than 10 million dollars in the HEW budget to deal with this. Let me tell you this, if you have any doubts about how important advertising is: the tobacco industry didn't care about how much money we devoted for research, but they fought against all the money I tried to get for educating kids about the dangers of smoking.

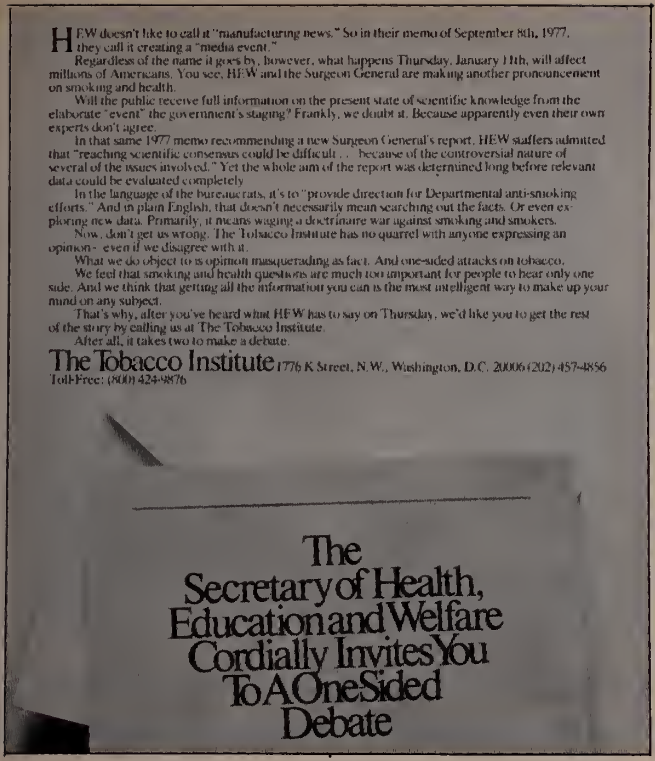
AB: In 1979 you said that the companies each should give 10% of their advertising budget for a mass media program to discourage teenage smoking. Now Reynolds has placed ads in teen magazines saying "We don't want kids to smoke." They've even collaborated with the National Association of State Boards of Education on a book called *Helping Youth Decide*. They've done what you've asked them—or have they?

JC: I don't think they have. We need a concerted effort that lays out the dangers of smoking for kids. Tobacco companies should not have anything to do with the copy that is written. They should simply provide 10% of the money they spend on advertising and give it to the state public health organizations and the school systems—organizations that will look at this from the health point of view and from the children's welfare.

AB: But while you were secretary, New Jersey was given $95,000 for a year-long anti-smoking program. That's less than one hour's worth of cigarette advertising.

JC: It was not advertising in the usual sense, but rather educational materials that could be used by a teacher in the classroom. We know those programs work. Schools that have those programs graduate a far lower proportion of teenagers who smoke. That is where the effort ought to go.

AB: Are you excluding contexts other than the class-



HEW doesn't like to call it "manufacturing news." So in their memo of September 8th, 1977, they call it creating a "media event."

Regardless of the name it goes by, however, what happens Thursday, January 11th, will affect millions of Americans. You see, HEW and the Surgeon General are making another pronouncement on smoking and health.

Will the public receive full information on the present state of scientific knowledge from the elaborate "event" the government's staging? Frankly, we doubt it. Because apparently even their own experts don't agree.

In that same 1977 memo recommending a new Surgeon General's report, HEW staffers admitted that "reaching scientific consensus could be difficult . . . because of the controversial nature of several of the issues involved." Yet the whole aim of the report was determined long before relevant data could be evaluated completely.

In the language of the bureaucrats, it's to "provide direction for Departmental anti-smoking efforts." And in plain English, that doesn't necessarily mean searching out the facts. Or even exploring new data. Primarily, it means waging a doctrinaire war against smoking and smokers.

Now, don't get us wrong. The Tobacco Institute has no quarrel with anyone expressing an opinion - even if we disagree with it.

What we do object to is opinion masquerading as fact. And one-sided attacks on tobacco.

We feel that smoking and health questions are much too important for people to hear only one side. And we think that getting all the information you can is the most intelligent way to make up your mind on any subject.

That's why, after you've heard what HEW has to say on Thursday, we'd like you to get the rest of the story by calling us at The Tobacco Institute.

After all, it takes two to make a debate.

The Tobacco Institute 1776 K Street, N.W., Washington, D.C. 20006 (202) 457-4856
Toll-Free: (800) 424-9876

The
Secretary of Health,
Education and Welfare
Cordially Invites You
To A One Sided
Debate

room?

JC: No, but the classroom is the one place where you have a captive audience. With a tradition of teaching about health and hygiene, why not deal with real health problems? With these kids, we are talking about cigarettes, drugs, and alcohol in urban areas.

AB: Major League Baseball advertises itself as a family sport, but if you go to a game your children are a captive audience for Winston or Marlboro billboards at nearly every stadium. That makes a mockery of the law concerning cigarette advertising on TV and radio. Did you ever consider combating smoking by using the same sort of ploys?

JC: Not exactly in that context. I did try to get teenage idols and celebrities to combat smoking by using posters, for example, Brooke Shields and Tony Randall. Actually, when I was fired we were on the verge of trying to do that with some rock stars using a TV campaign. That stuff works. An example: immunization. Between 60% and 70% of children were immunized before an ad campaign, and then we moved up to 90%. One of the elements in that campaign was the Star Wars stuff, which was very popular at that time. I got football, hockey, basketball, and baseball people to run those ads, and they had a tremendous impact on the kids and their parents.

AB: You've said that more research must be done on why kids smoke. Don't the advertising agencies already have all the data?

JC: Research is needed to find out the most effective ways to get people to quit smoking. We have all this publicity which says that many people are quitting, but the reality is this continual increase in the number of teenage and young adult women who are smoking. They are not quitting.

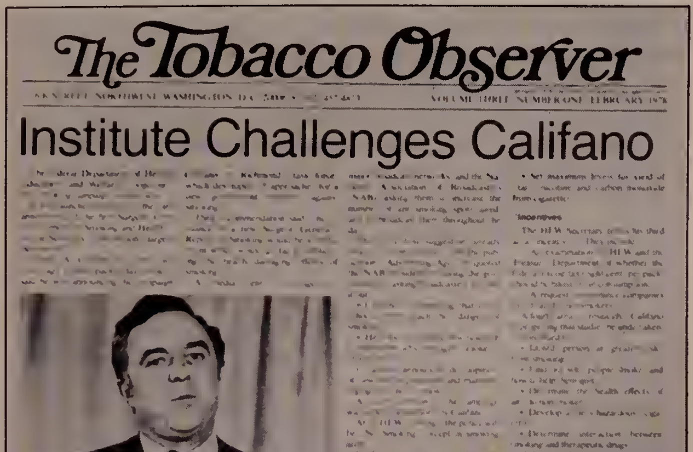
AB: Is there any mystery to that? If the Berlin wall were torn down tomorrow a stream of people would leave, but not everybody. If cigarette advertising were ended tomorrow, not everybody would stop smoking, but an enormous number of people would. The industry does not aim at subsets of people, they aim at everybody. Have you ever sat down with tobacco advertising people and said, "Come on, tell me what is going on?"

JC: No. I knew what was going on with cigarette advertising because I had represented companies. Coca Cola is an example. In the course of working with them I studied all of Coca Cola's advertising and realized the enormous effort that went into identifying why people would drink this and to whom it would be appealing—all of those things. I knew that the cigarette guys were immersed in the same marketing strategies; what do you do to hook a 16 year old and get them interested in smoking?

AB: A company will spend $30 million a year selling weed killer, but you had only $30 million for that whole budget on smoking. Did you ever consider blowing it on two week's worth of prime time ads and then hope the momentum against smoking would perpetuate?

JC: I didn't have $30 million, but I would have done that absolutely. If most smokers tried once in the past year to quit, no matter how feeble the attempt, then we could really start reminding them over and over. That would have an impact. But not having the money, I tried to do the same thing in a different way: by using newspeople and the media. And we really did that. With the Surgeon General's report on smoking we had a week of media coverage. We identified media markets all around the country. In an attempt to keep banging away publicly our theory was "this is our advertising; we don't have the money for our own ad campaign but every time we remind 2,000 smokers about quitting, somebody out there would quit." Let me tell you something: despite the controversy I had at HEW, people still come up to me and say "I quit smoking," or "You got my wife to quit." Nothing makes me happier. That feedback makes my time at HEW worth the effort.

AB: There is a lot of concern in your book about North Carolina. A lot of the health and smoking issue revolves around North Carolina these days. They almost had a chairman of the House Subcommittee on Health, they've



The Tobacco Observer

Institute Challenges Califano

President Discusses Califano

had Helms as head of the Senate Agriculture Committee, and they've been able to sabotage any kind of counter-cigarette bill. But, ironically, in the past year the North Carolina Council of Churches and the Southern Baptist Convention—tobacco-state based religious groups—have questioned the very continuation of tobacco-growing, and *The Greensboro News and Record*, and *The Louisville Courier-Journal* have covered the problems caused by the continued cultivation and selling of tobacco. What about New York? Little of this questioning has gone on in New York, which is headquarters for three of the six major cigarette companies. Is New York our leading tobacco center?

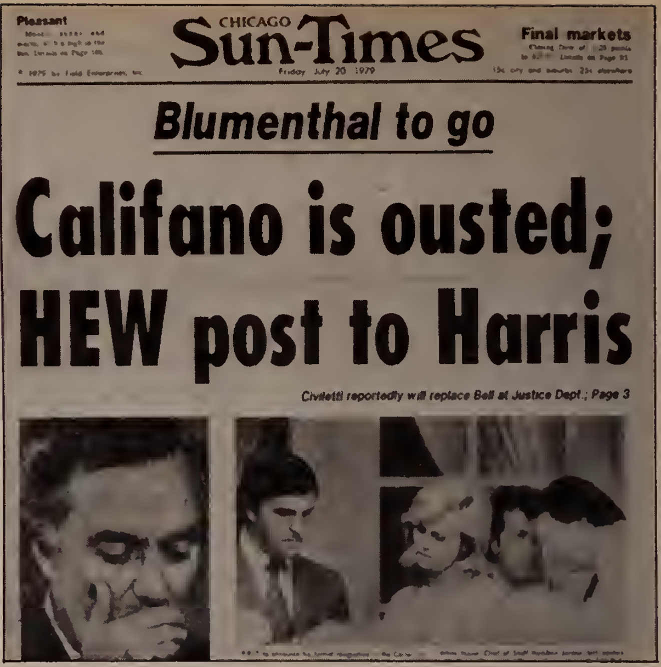
JC: I don't think so, but the papers in New York—*The Times, The Daily News*—are supportive of efforts against smoking.

AB: But if you were running for the Senate from New York, and you learned that in Albany one of the leading lobbies is Philip Morris, that the Governor has appointed George Weissman (chairman of the board of Philip Morris) as a special advisor and head of a business development campaign for New York State, and that most members of the black and Hispanic caucus voted against the Clean Indoor Act in 1984, the political pressure in favor of tobacco would be profound. The legislative record of New York probably is worse than that of any state, including North Carolina.

JC: That's true and equally unfortunate. I would be interested to see Governor Cuomo trying to take some leadership with the legislature, for example, banning of smoking in public places or requiring that people have a smoke-free work place. Legal action works, and we know it works. It works in California. I just read a story about Minnesota, which has the objective to be a smoke-free state by the year 2000. We must have laws, because then people can sit in offices and indoor public places and not have to breathe that stuff.

AB: Are you willing to take a closer look at *The New York Times?* You were very concerned about the reaction of the *Times* to you, to your policies, and to your beliefs. Yet, what is the *Times* if it will not even address its largest single source of revenue? Is this a newspaper that can be condemned? Can it be taken as an organ that is subverting the health belief system?

JC: Anyone who runs a newspaper is going to tell you first of all right on the editorial page about the need for products that are legal advertising. *The New York Times* and *The Washington Post* gave plenty of coverage to the issues I raised, as distinguished from *TIME* or *Newsweek*. The second question is the ethics of whether or not newspapers have an obligation not to permit themselves to be used to promote something that is unhealthy. That is a tough question because it gets into a whole host of areas of how and where to draw the line. I spent years representing *The Washington Post*. We faced awfully tough problems in ethical advertising: what we should allow and what we should stop. Ultimately the paper came to the conclusion that it would let advertising run that was found to be honest and socially acceptable. That still may not answer the cigarette question, but my hunch is that most newspaper publishers will say, "I'll run the ad with the health warning that the US Congress says to put on. That's the law of the land and I'm following that." Some people would say that is a cop-out.



Pleasant

CHICAGO Sun-Times

Friday July 20 1979

Final markets

Blumenthal to go

Califano is ousted; HEW post to Harris

Civiletti reportedly will replace Bell at Justice Dept.; Page 3

AB: There seems to be subversion of the issue when you compare the enormous number of articles on EDB and asbestos with the handful of those on cigarettes or cigarette advertising. Can *The New York Times* or *The Washington Post* be exposed for having failed to address the ethical dilemma?

JC: The way to approach this is to have the readership constantly raise the issue and let the papers ponder their response.

AB: Incidentally, you sound as if you would have worked very well with Surgeon General Koop.

JC: Koop has been first class—gutsy and strong. I wish he would be more vocal. He's got charisma, but he has to keep talking. He has a good image with that great beard and uniform.

AB: Are there any Congressmen who will help carry the day on this issue?

JC: Congressmen are interested in this issue. It has gained a little bit of support every year, but it's very tough. Look at the big vote two years ago on the agriculture subsidy for tobacco farmers which came the same year as the extension of the Civil Rights Act. My recollection is that the entire black caucus and a large number of liberals voted for the tobacco subsidy. They voted for it as a tradeoff because the North Carolina delegation and some other southern delegations agreed to vote for an extension of the Civil Rights Act. That kind of deal will exist in every democratic legislative body in the world, and it certainly exists here. While I don't like that kind of deal, I understand it. What I don't like and don't understand is the tremendous power of Congress. You ask me what I worry about in this country. I'm worried about Congress. I'm worried about a Congressman who earns $70,000 and has to keep up two houses. The day after he's elected, he has to start raising half a million dollars to pay off campaign debts and another million for the next campaign. I'm worried about the environment where the committees and subcommittees he sits on are more important to the congressman because of the money he can raise, than are the constituents in his district. That is what is happening.

The Comprehensive Smoking Education Act

HON. HENRY A. WAXMAN

> "WARNING: The Surgeon General has determined that cigarette smoking is dangerous to your health."

For more than a decade this statement has been the familiar health warning label that appears on cigarette packs and advertisements. In 1981, the Congress began considering legislation to change the label to reflect more specific knowledge about the health effects of smoking. In the closing days of the 98th Congress, public health advocates were victorious.

Effective October 12, 1985 the current 14-year-old health warning will be replaced by four new labels:

> SURGEON GENERAL'S WARNING: Smoking Causes Lung Cancer, Heart Disease, Emphysema, and May Complicate Pregnancy.
>
> SURGEON GENERAL'S WARNING: Quitting Smoking Now Greatly Reduces Serious Risks to Your Health.
>
> SURGEON GENERAL'S WARNING: Smoking By Pregnant Women May Result in Fetal Injury, Premature Birth, and Low Birth Weight.
>
> SURGEON GENERAL'S WARNING: Cigarette Smoke Contains Carbon Monoxide.

The labels will be rotated every three months, so that each will appear approximately the same number of times on the packaging and advertising of each cigarette brand. The warnings will be abbreviated for use on outdoor billboards, where they will be enclosed in a black border. In magazines and newspapers, the statements will continue to be enclosed in a box.

The old warning's usefulness in disseminating information has diminished with time, and the substance of its message has been outdated by scientific advances. In 1981 the Federal Trade Commission determined that fewer than 3% of those who see a cigarette advertisement notice the warning, let alone read or consider its meaning.[1] Further, thousands of additional scientific studies not only confirmed smoking's causal relationship to lung cancer, emphysema, and cardiovascular disease, but have added a variety of other disorders, including injury to the fetus. The risks are specific, predictable, and thoroughly documented. During consideration of the labelling legislation, the Surgeon General issued three reports describing the relationship between smoking and cardiovascular disease, cancer, and chronic obstructive lung disease.[2-4]

JUST THE FACTS

The wording of the new warnings was the product of negotiation and compromise. They were not designed to win literary prizes. Rather, they were intended to be simple vehicles for conveying factual, scientific information. With the exception of the carbon monoxide label, the warnings were based on earlier legislative proposals and suggestions contained in the Federal Trade Commission's 1981 *Staff Report on the Cigarette Advertising Investigation.* The carbon monoxide label was a compromise proposed by the tobacco industry. The industry preferred the imposition of a fourth warning rather than an earlier proposal that the actual carbon monoxide content of cigarettes be disclosed on packages and in advertising.

To enhance the visibility of the warnings, the size of each label and the letters of the warning will be increased by 50%. Authority to enforce the new law has been given to the Federal Trade Commission.

In addition, the legislation requires that cigarette manufacturers disclose, for the first time, the chemical ingredients added to cigarettes. This information must be reported annually to the Secretary of Health and Human Services. The regulation supercedes the informal, voluntary arrangement negotiated in 1980 between cigarette manufacturers and the Department of Health and Human Services. Under the earlier agreement, only those ingredients used by at least three manufacturers were disclosed.[5] Mandating the disclosure of a complete list of cigarette ingredients will permit the federal government to initiate the toxicologic research necessary to measure any additional health risk posed by the use of additives.

Finally, the legislation established a permanent statutory requirement that the Secretary of Health and Human Services carry out a program to inform the public of the adverse health effects of smoking. The mandate is backed up by a federally chartered Interagency Committee on Smoking and Health. The committee is composed of representatives from federal research agencies such as the National Cancer Institute and the National Institute on Drug Abuse as well as agencies like the Federal Trade Commission and the Department of Education. In addition, the committee includes five members from the public sector representing major voluntary health organizations

---

Representative Waxman was first elected to Congress in 1974 and represents California's 24th Congressional District. He is chairman of the Subcommittee on Health and the Environment and the author of the Comprehensive Smoking Education Act.

Address correspondence to Representative Waxman, Chairman, Subcommittee on Health and the Environment, US House of Representatives, 2415 Rayburn House Office Building, Washington, DC 20515.

active in the prevention of cigarette smoking. The committee will enable the federal government to expand its commitment of educational and research resources and to coordinate the use of these resources with the private sector.

The new cigarette labelling law emerged after extended discussions and much compromise. Negotiations between members of Congress, the Coalition on Smoking OR Health and representatives of the Tobacco Institute spanned eight months. While the proposal fell short of what many believed would be ideal, it represents progress in helping the public become more knowledgeable about the adverse health effects of smoking.

The Coalition on Smoking OR Health was the inspiration of the American Heart Association, the American Lung Association, and the American Cancer Society. By combining resources and establishing a daily presence in Washington, DC, the coalition became a source of bitter frustration to their better financed opponents in the tobacco industry. The coalition's success was due largely to its ability to transform an issue many viewed as a parochial concern of anti-smoking zealots into an issue of national interest and political importance.

What brought the cigarette industry to the bargaining table related more to politics than enlightened recognition of the health effects of smoking. It was the realization of the coalition's growing political strength and the fear on the part of the industry's legal advisors that stronger legislation, potentially more damaging to the industry, might emerge from Congress if the labelling issue were not resolved in 1984.

INDUSTRY FEARS

The proponents of stronger labelling were willing to negotiate with the industry for two reasons. First, it was the only way to obtain passage of the bill in the United States Senate. Senate rules would permit a single senator from a tobacco-growing state to delay—perhaps indefinitely—any legislation reported by the House. Second, resolution of the three-year labelling controversy would permit energies to be directed to other important concerns, such as using the federal cigarette excise tax to assist in helping solve Medicare's financial problems, eliminating the tobacco subsidy in the federal agriculture program, and promoting more effective anti-smoking messages through the use of television and radio.

Does the industry's willingness to compromise indicate any shift in their historical unwillingness to admit the health effects of smoking? Absolutely not. In fact, after House passage of the negotiated compromise, the industry attempted a last minute, but unsuccessful, effort to scuttle the legislation in the Senate. Some Senators from tobacco-growing states in the South threatened to delay Senate consideration of the compromise unless amendments were made to address concerns raised by home state farming interests and cigarette distributors. (The final legislation incorporated a Senate amendment deleting the bill's congressional findings and adding a technical clarification that the warning label requirements did not apply to tobacco distributors who were not also cigarette manufacturers or importers. No substantive changes in the labelling requirements were made as a result of the Senate action.) Through the work of the coalition and concerned members of the House and Senate, final Senate passage was achieved despite the industry's change of heart.

There are lessons from this experience which can help in efforts to reduce the prevalence of smoking. The cigarette industry continues to argue that there is no evidence that smoking poses any form or degree of health threat, as one tobacco industry executive testified before the subcommittee:

> After three decades of investigation and millions of dollars invested, the smoking and health controversy remains unresolved.... we are firmly opposed to this legislation because ... the medical and scientific assumptions or findings underlying it are incorrect and unsubstantiated.[6]

For this reason the industry insisted in the negotiations that any warning label be prefaced with an attribution to the Surgeon General. Industry lawyers claimed their concern was motivated by fear that an unattributed warning might broaden the industry's product liability exposure. Industry representatives also insisted that any references in the legislation to the addictive nature of smoking be deleted, notwithstanding the position of the National Institute on Drug Abuse (NIDA) that "The addictive nature of cigarette smoking is why cigarette sales continue year after year in spite of the health hazards of smoking."[7]

There is evidence of an integral relationship between early cigarette use and illicit drug use by young people. In testimony before the Congress, William Pollin, director of NIDA, sounded a warning every parent should heed:

> ... cigarette smoking by youth is of concern to us both for the predictable health consequences this generation will face in the future and for the implication increased cigarette use trends have for trends of illegal drug use. Historically cigarette use has indicated the direction of illicit drug use trends by youth and longitudinal studies have identified tobacco use as an integral and crucial step in the sequences of developing drug use patterns.[8]

It is no wonder the industry is sensitive to the addiction issue and disputes these contentions. In its defense, a psychologist-consultant for the tobacco industry compared the addictive effects of smoking to the "... equally strong attachments [people have] to tennis, jogging, candy, rock music, Coca-Cola, members of the opposite sex and hamburgers ... "[9]

Although references to addiction were deleted from the text of the new law, public health experts should move to make addiction a major focus of educational activities. Efforts to discourage young people from beginning what can become a life-threatening addiction deserve no less of a commitment than that to remove the threat of other dangerous drugs from the schools.

CLEVER CIRCUMVENTIONS

Another important issue public health advocates must consider is the role of advertising in promoting cigarette sales and cigarette consumption, particularly among the young. It is no secret that during the past 14 years cigarette advertisers have found clever and effective methods of working with (perhaps more accurately working around) the health warning label. In the hearings it was ironic to find the tobacco industry and its advertising and marketing consultants among the most impassioned advocates for leaving the current warning unchanged. One tobacco industry executive testified, "We think your Surgeon General's warning is working very effectively." [10] A marketing consultant to the industry criticized the legislation for, in his words, "[replacing] a highly successful program of informing consumers .... " [11]

Industry representatives expressed concern over the impact of new warning labels on the effectiveness of their advertising. But it is the effectiveness of cigarette advertising that most alarms public health experts. The very design of many cigarette promotions seem intended to undercut or overwhelm public concern about the adverse health effects of smoking with positive, attractive imagery. In time, the industry will find the most effective way to mask these new warnings and to help smokers rationalize what Joseph Califano, former secretary of the federal Department of Health and Human Services, characterized as "slow motion suicide." This is why cigarette companies and their advertising agencies hire legions of psychologists to develop sophisticated marketing strategies. Successful promotion of a product regarded by the scientific and medical communities as the nation's most preventable cause of death and illness is hard work.

It is also no accident that cigarette companies now promote cigarette sales through nontraditional forms of advertising, such as sponsorship of sporting events and rock music concerts. Such "lifestyle advertising" is not suitable to display of health warnings and oftentimes provides the company with indirect access to free television and radio coverage. Associating cigarette products with sporting events can have a profound impact on children and teenagers and can blunt the effectiveness of smoking education efforts. Cigarette sponsorship of such events should be discouraged by working with television networks, stadium owners, and sports governing bodies.

Passage of the Comprehensive Smoking Education Act is a victory for those concerned about public health. New warning labels will provide the public with a greater array of information on the adverse health effects of smoking. Yet this progress can only be considered a first step toward the ultimate goal of reducing and one day eliminating cigarette smoking as a major cause of death, disease, and disability.

COMPLACENCY CAN KILL

Those concerned about the health effects of tobacco must not be complacent. Public complacency is what the tobacco industry was counting on when they withdrew their public opposition to the labelling bill. Reducing the incidence of smoking requires a comprehensive approach. The new health warnings are only one step. To be successful, the nation must also address and reform the agricultural and taxing policies that have shielded smokers from bearing the actual costs of their dangerous practice.

Further federal subsidy of tobacco should end and tobacco farmers should be encouraged to plant other less toxic and potentially more profitable crops. The federal tobacco excise tax must be increased. The current $0.16 levy is scheduled to fall to $0.08 in October. Although a deficit-conscious Congress is not likely to allow this to happen, the tax should be raised further. Legislation has been introduced in the 99th Congress that would increase the cigarette excise tax to help finance the Medicare health program. (HR 1594 was introduced on March 19, 1985 by myself and the Honorable Tom Tauke, R-Iowa.) The legislation would increase the federal cigarette excise tax from $0.16 to $0.32 a pack.

With the enactment of Public Law 98-474, the tobacco lobby hoped to minimize their losses and avoid more stringent legislation in the future. Yet the new law demonstrates the industry's vulnerability to public health pressures. The law is a charge to redouble smoking prevention activities—particularly at the grassroots level where restrictions on smoking in public facilities and in the workplace have met with success.

Renewed commitment to educating the public about the adverse effects of smoking—particularly the addictive nature of tobacco—combined with the new warning labels will contribute to achieving the goal of a nonsmoking generation by the year 2000. The Surgeon General's goal is achievable if public health advocates can build on the progress achieved by the labelling bill.

REFERENCES

1. *Staff Report on the Cigarette Advertising Investigation.* Washington, DC, Federal Trade Commission. May 1981, p 20.
2. *The Health Consequences of Smoking: Cancer.* A report of the Surgeon General. Washington, DC, US Department of Health and Human Services, 1982.
3. *The Health Consequences of Smoking: Cardiovascular Disease.* A report of the Surgeon General. Washington, DC, US Department of Health and Human Services, November 1983.
4. *The Health Consequences of Smoking: Chronic Obstructive Lung Disease.* A report of the Surgeon General. Washington, DC, US Department of Health and Human Services, May 1984.
5. Testimony of Edward N. Brandt, MD, before the Subcommitee on Health and the Environment, Energy and Commerce Committee, House of Representatives. Smoking Prevention Act. Hearings serial no 98-8, March 9, 1984, pp 84–85.
6. Testimony of Edward A. Horrigan, Jr, chairman and chief executive officer, RJ Reynolds Tobacco Co before the Subcommittee on Health and the Environment, Energy and Commerce Committee, House of Representatives. Comprehensive Smoking Prevention Education Act. Hearings serial no 97-106, March 12, 1982, pp 353, 355.
7. "Why People Smoke Cigarettes." PHS publication no (PHS) 83-50195, Washington, DC, US Department of Health and Human Services, 1983.
8. Testimony before the Subcommittee on Health and the Environment, Energy, and Commerce Committee, House of Representatives. Smoking Prevention Education Act. Hearings serial no 98-8, March 9, 1984, p 42.
9. Testimony of Theodore H. Blau, PHD, before the Subcommittee on Health and the Environment, Energy and Commerce Committee, House of Representatives. Comprehensive Smoking Prevention Education Act. Hearings serial no 97-106, March 12, 1982, p 488.
10. Testimony of Edward R. Horrigan before the Subcommittee on Health and Environment, Energy and Commerce Committee, House of Representatives. Comprehensive Smoking Prevention Education Act. Hearings serial no 97-106, March 12, 1982, p 393.
11. Testimony of Roger D. Blackwell, PHD, Professor of Marketing, Ohio State University before Subcommittee on Health and the Environment, Energy and Commerce Committee, House of Representatives. Comprehensive Smoking Prevention Education Act. Hearings serial no 97-106, March 12, 1982, p 474.

New Jersey's hard-fought battle for clean indoor air legislation

An insider's view

HON. MARTIN A. HERMAN

On December 3, 1981 then-New Jersey governor Brendon Byrne signed into law three bills that prohibited smoking in elevators and schools and limited smoking in hospitals and physicians' offices. Left behind were many other measures that would have limited smoking in the work place, restaurants, and most places of public access and accommodation.

The governor was not reluctant to sign such measures. Rather, the legislature, responding to pressures from the tobacco industry, unions, and the business community, abstained en masse from voting. Although the state constitution requires just 41 affirmative votes out of 80 for passage of a bill in the General Assembly, the legislation drowned in a sea of abstentions in both the 1980–1981 and 1982–1983 sessions.

Having waged several tough but ultimately successful battles on controversial issues such as the raising of the drinking age to 21 and the enactment of an effective Generic Drug Law, I wondered why I was not making headway on an issue that had overwhelming public support. All surveys, other than those commissioned by tobacco companies, have found that three quarters of the population does not smoke, that 9 out of 10 persons who smoke want to stop, and that nearly everyone who does not smoke—and most of those who do—support clean indoor air legislation.

After much soul-searching I realized that a good part of the blame rested with me. First, I had not engaged in the "one-to-one" colleague discussions so necessary to pass hotly contested legislation. And unlike the generic drug and drinking age fights, which both took several years to succeed, I had not kept up a continuous exchange of information and one-to-one dialogue with my colleagues. By default, I had left the legislative inside track wide open to the opposition from the tobacco industry. In 1984 I began personally seeking the vote of each legislator and providing well-researched responses to the arguments of the opposition.

In my 11 years in the legislature I have learned that having the good guys on one's side and being right will not necessarily carry the day in the face of well-organized, well-financed opposition. Strength must be met with strength. The strength of the rich, be they the tobacco industry, pharmaceutical manufacturers, or brewers and distillers, lies in their ability to employ high-priced lobbyists to try to prevent their profits from being diminished. Profits have their own set of social blinders which shade out human misery and suffering no matter how right the cause one espouses.

COALITION BUILDING

Fortunately, a legislator has a potent counterpunch to big bucks: big votes. Thus, in the generic drug bill battle, not even the huge pharmaceutical industry could overcome the lobbying efforts of one million older persons who made known to legislators their strong support of the bill. However, unlike the senior citizen activists who carried the day for the generic drug bill, and unlike the coalition of parents, teachers, police, and health professionals who defeated the liquor lobby to raise the drinking age, there was no broad-based group willing to devote the time and effort to oppose the tobacco industry. A number of individual organizations such as GASP (Group Against Smoking Pollution), the New Jersey divisions of the American Cancer Society and the American Lung Association, and the New Jersey Medical Society had called for the passage of legislation, but they were acting as individuals and did not coordinate their efforts. The president of the New Jersey Lung Association, Lee Reichman, MD, director of the pulmonary division at the College of Medicine and Dentistry of New Jersey, Rutgers Medical School, stepped forward to help lead the battle and promised to meet with each legislator if need be to help insure the passage of the various measures. There were five primary opponents to "no smoking" legislation: the tobacco industry, restaurants, the Business and Industry Association representing 11,000 of New Jersey's larger firms, the Communication Workers of America (CWA, the state's largest employee union), and police groups who felt the legislation was unenforceable.

There has been no way to achieve any agreement with the tobacco industry, which is sensitive to any threat to its enormous profits. Since many people will smoke fewer cigarettes or give up smoking if there are disincentives to smoking in public, sales of cigarettes could be expected to decline. Small wonder that the tobacco industry has sent in lobbyists to even the smallest communities to defeat clean indoor air laws. Both the tobacco industry and the New Jersey Restaurant Association retained the same lobbying firm. This left little doubt of the association's continued opposition to legislation calling for smoke-free sections, even when the only penalty for non-compliance would be the posting of a notice on the door advising patrons that the restaurant did not provide such a section. However, in winning over allies, it is a mistake to assume

---

Mr Herman is Assistant Majority Leader of the New Jersey General Assembly.
Address correspondence to Mr Herman, 59 North Broad St, PO Box 377, Woodbury, NJ 08096.

that all opponents wear black hats and do not have a legitimate reason for opposing a piece of legislation.

In the 1984–1985 session, then, it was necessary to gain the support of all these organizations. Securing the release of a bill from the Assembly Judiciary Committee (which I chair) for a floor vote did not pose a problem. But a strategy other than "release, post, and pray" was in order.

To that end, a strong effort was made to broaden natural support constituencies to the New Jersey Federation of Senior Citizens (the premier lobbyists for the state's one million older persons) and the state Parent Teachers Association (PTA) representing over 400,000 parents.

WINNING OVER THE OPPOSITION

In late summer, a planning meeting was held to expand public support and legislative lobbying. In the legislative process timing is essential, and the building of momentum is the key ingredient to adding important constituent and colleague support. Among the contingency strategies agreed on was for physicians to be assigned to specific legislators to get the point across. Profiles of each legislator were developed.

A public hearing on September 25 was highlighted by a letter of support from the Surgeon General of the United States (generated by GASP) and medical testimony from several physicians and other health professionals who took a day off to travel to Trenton to speak on the dangers of second-hand smoke. The hearing received coverage in the mass media.

A major breakthrough occurred when the CWA and the New Jersey Business and Industry Association, although re-affirming their long-standing opposition, expressed sympathy for the legislation's goals and left the door open for further discussion. It appeared that given fair accommodation and a voice in the legislation's final structure, the CWA and the Business and Industry Association could be won over.

Following the public hearing, a meeting was arranged by leaders of the New Jersey Business and Industry Association, at which time I pledged to the membership that if I could not prove to them that this legislation was good for business as well as the health of their employees, I would withdraw the bills. In return, I asked for their support for the legislation. I made the same request to the president of the CWA.

Within two weeks, agreements for bill amendments were hammered out and pledges of active support from both the CWA and the Business and Industry Association were made. We had succeeded in communicating the seriousness of the smoking problem and in impressing upon both labor and industry that this was not just a "bad habit" to be dealt with by a "nuisance ordinance."

The tobacco lobby could no longer hide behind the skirts of two of New Jersey's influential lobbying groups. For the first time the cigarette interests stood naked except for their allies in the New Jersey Restaurant Association. Yet the restaurant industry opposition itself began to divide, as the 450-member Garden State Restaurant Association stepped forward at the public hearing to register support for the bills as being in the best interest of the industry and the public. The New Jersey Fraternal Order of Police and chiefs of police added a most welcome pledge of support. Endorsements also were received by the New Jersey Nurses Association, the New Jersey Pharmaceutical Association, and the New Jersey Education Association.

The press conference to announce the support of the CWA and the New Jersey Business and Industry Association, a major news event in itself, became a statewide front-page story when it coincided with the day of the Great American Smoke-out of the American Cancer Society.

On the following Monday, the bills were released from the Judiciary Committee for a floor vote. Although there were more than enough "yes promises" to produce sufficient votes to pass all six bills, letters were sent to all legislators prior to the vote thanking them for their support.

WHAT THE MEASURES WOULD DO

Assemblyman Herman's six amended measures included this statement: "The purpose of the bill, as well as the companion bills, is to protect and preserve in a reasonable manner the health and comfort of the people of this state by establishing areas that are free from the toxic and annoying effects of tobacco smoke. This bill is intended to establish a social policy without providing complex or onerous administrative or law-enforcement procedures." The bills are as follows:

- Smoking is restricted in work places of 50 or more employees. Although employers establish the specific policy, persons who do not smoke cannot be forced to sit near persons who do.
- Smoking is prohibited in retail food stores with selling areas in excess of 4,000 square feet.
- All restaurants must establish and post a smoking policy.
- Smoking is banned on all public transit buses, except charters.
- Smoking is restricted in government buildings.
- Smoking is banned in pharmacies and in any other areas where health supplies are sold; owners of concert halls and department stores can set up non-smoking areas.

AMATEURS BEAT THE PROS

The General Assembly vote on December 13, 1984 went exactly as planned. On the day of the vote, a Clean Indoor Air Coalition of physicians, pharmacists, nurses, members of GASP, the American Cancer Society, the American Lung Association, the New Jersey State PTA, and other organizations marshalled a strong lobbying presence at the State House. They distributed "No Smoking" buttons in a friendly manner; and they engaged in no-pressure, one-to-one chats with fence-sitting Assembly members and thanked other members who had promised to vote "yes."

In the debate, key members of both parties spoke in support of the bills. Watching the proceedings from the balcony, sitting side by side with the tobacco industry lobbyists, were the unpaid amateur lobbyists who had given of their time to come to Trenton. Green light after green light on the Assembly's electronic tallyboard added up to a resounding victory for the six-bill smoking control package over an industry that had counted on old ways and old prejudices to prevail. Every legislator who had promised to vote "yes" did so.

Obtaining 21 of 40 Senate votes would not be as easy as getting 41 out of 80 in the Assembly. By New Year's Day, two weeks following the Assembly vote, I had talked to a majority of Senators by phone or in person and believed that more than the 21 votes needed for passage were at hand. Coalition members also had been busy reinforcing the message.

A breakthrough occurred when I called officials at the New Jersey Restaurant Association to ask how they continued to allow their lobbying firm to create the appearance that they stood aligned with the tobacco industry on such a measure, especially one that was destined for defeat. The officials said that I should not be so hasty to judge, and if I would clarify certain ambiguities in the bill, they would consider endorsing it.

After a constructive hour-long meeting, the language changes were agreed upon—with one bonus: at the association's suggestion, the restaurant bill was to be amended to make it applicable to all establishments, not just those with more than 50 seats.

On January 28 the bills were released for a floor vote from the Senate Law and Public Safety Committee by its chairman, Senator Frank Graves, who had been a sponsor of similar legislation. One remaining hurdle was getting the Senate president to list the bills for a vote. Notwithstanding his friendship with a lobbyist for Philip Morris, I knew him to be fair and believed he would eventually agree to post the bills. My impression proved accurate, and the bills were listed for a vote on March 7.

ABSTENTION STRATEGY

The New Jersey Senate has 40 members; 21 votes are needed to pass a bill. It was clear the tobacco industry needed a breather to regroup and to slow down the runaway "no smoking" train the bills were travelling. The longer the bills could be kept off the Senate floor, the more time they would have to peck away at the 31 "yes" votes I believed I had. Holding the bills until March 7 would prove to be very helpful to their cause. But they needed a new strategy, for with the defection of the New Jersey Restaurant Association, the tobacco industry stood alone.

The new tactics were based on a keen understanding of the legislative psyche: "be nice to all at all costs . . . ." To implement this strategy required a lobbying firm that could ply both sides of the aisle. During the present administration such a firm was created by Raymond Bateman, a former Republican State Senator and gubernatorial candidate, and Harold Hodes, former chief counsel to Democratic Governor Byrne.

Their clever strategy was to lobby all senators, both Democratic and Republican, with a last-minute message I called the "four for him two for us" gambit. For Republicans, the message had a "political kicker" mixed with it: "Herman is a candidate for re-election in a swing district. If you give him a six-bill, complete victory it will make him look really good and hurt our chances to defeat him." To Democrats the message was a seemingly conciliatory one: "Herman's getting four bills, so don't worry, he'll still look good. We would really appreciate it though if you could abstain on the two bills with which we have real problems." The strategy worked, and the two bills failed by four votes.

The tobacco lobbyists knew that senators could not be made to look like they were going back on their word; they also knew that four of the bills were supported by those businesses to whom the bill applied (restaurants, the workplace, food stores, buses). Therefore, by focusing on the remaining two bills (government buildings and public places) the tobacco lobby could avoid the appearance of a total defeat in the public eye.

But, as editorials criticizing the Senate vote have suggested, support is growing for legislation to curb smoking in public places. I have no doubt whatsoever that in the near future the two remaining bills—and others—will pass with little opposition.

Acknowledgment. This paper is dedicated to my wife Rose, who after 17 years was able to stop smoking, and to our children Elliott, Robbie, and Nicki—and to all children. It was written in memory of my mother, who died of lung cancer at age 49.

Governor Thomas Kean signed the four bills into law on June 12, 1985. On June 17, after a minor amendment was attached, the New Jersey Senate passed both of the remaining bills. On June 24, the Assembly overwhelmingly concurred. —*Editor*

SUFFOLK'S SUCCESSFUL CLEAN INDOOR AIR ACT

On July 12, 1984, Suffolk County (NY) enacted one of the most stringent clean indoor air bills in the United States. Under the law, which is enforced by the Department of Health Services, restaurants with a capacity of 50 or more have the option of setting aside one fifth of their seats for no smoking or installing smoke filtering devices. Workplaces with 50 or more employees must adopt a written policy on smoking such that a worker has the right to designate his immediate area smoke free. A proposal by county legislator Ira Hariton that prohibited smoking in nearly all public and work places had been rejected a year earlier by the Suffolk County Legislature in an 8 to 7 vote.

According to county officials, employers, and employees, the year-old law has been successful and businesses are cooperating. Smoking has been banned entirely in the common work areas of many of the county's 350 companies with more than 50 employees. The law inspired the 20,000 employee Grumman Corp, Long Island's largest employer, to restrict smoking at all of its facilities in the US. The Department of Health of neighboring Nassau County has been inspired to press for an even more stringent measure than Suffolk's.

Source: DAVID HARRIS, MD
Health Commissioner
Suffolk County, NY

Proposition P: anatomy of a nonsmokers' rights ordinance

PETER HANAUER, ESQ

On November 8, 1983, voters in San Francisco passed "Proposition P," a referendum on the city's workplace smoking ordinance which had been enacted by the Board of Supervisors (the equivalent of a city council) six months earlier. The vote marked the first time that the tobacco industry, which has consistently opposed all laws regulating public smoking, had been defeated in such an election contest, and it meant that San Franciscans had approved what was then the strongest workplace smoking law in the country. The ordinance requires that all public and private office workplaces have policies on smoking that seek to accommodate the needs of smokers and nonsmokers. It requires that notice of the policies be given to employees and that appropriate signs be posted. The law is enforced by the city's health department, and civil penalties are imposed on employers who fail to establish reasonable policies.

WINNING OVER BUSINESS LEADERS

To understand why the ordinance reached the ballot in the form of a referendum, it is helpful to review the process by which it became law. When first introduced before a three-member committee of the Board of Supervisors, in January 1983, the ordinance was strongly supported by Californians for Nonsmokers' Rights (a non-profit organization developing legislative, legal, and educational approaches to curtail smoking in public places and the worksite) and by the health community in general. It drew virtually no opposition. But by the end of the second hearing, the sympathetic committee had reservations about the specific language, partly due to a letter from Robert Beck, a Bank of America vice-president and a leader in the American Cancer Society. Although he expressed strong support for the concept of the ordinance, Beck was concerned that the rigid conditions of certain provisions would adversely affect businesses. The committee asked for a one-month continuance to see if the problems could be ironed out. On the day before the next scheduled hearing, the Chamber of Commerce asked for a meeting with Californians for Nonsmokers' Rights to discuss an alternative proposal by the Bank of America. The bank presented the idea that became the cornerstone of the ordinance and the center of controversy: that every employer be required to establish a smoking policy to satisfy the needs of both smoking and nonsmoking office workers; but if an accommodation satisfactory to the nonsmokers, whatever their number, could not be made, then smoking would be prohibited in that work area. This proposal would give businesses flexibility in solving a problem, but would give employees a statutory right to a smoke-free environment.

In exchange for accepting the proposed compromise language, the Chamber of Commerce agreed not to oppose the ordinance. The precise language was hammered out at one further meeting, attended by Supervisor Wendy Nelder, the author of the ordinance, and representatives of Californians for Nonsmokers' Rights, the Chamber of Commerce, the Small Businessmen's Association, Bank of America, and two other banks. The ordinance was passed unanimously by the supervisors' committee in early May. In two required votes by the full Board, the measure passed by 9 to 2 and 10 to 1. It was signed into law by Mayor Dianne Feinstein on June 3. This happened despite intensive lobbying against the law by the Del Monte Corporation, a subsidiary of RJ Reynolds since 1979, the local attorney for the Tobacco Institute, and even some members of the national Democratic Party who tried to persuade the Mayor to veto the law. In support of the measure were hundreds of telephone calls and letters from San Franciscans. The Mayor later reported that she had received more than 100 letters from people around the country who urged her to sign the ordinance so that their local governing bodies would be encouraged to enact similar legislation.

Shortly before the first vote by the full Board, the Chamber of Commerce broke the agreement not to oppose the ordinance. Since its promised neutral stance had never been publicized, the Chamber of Commerce did not appear to have changed its position. On June 15, six people held a press conference to announce a campaign for a referendum to repeal the ordinance. Having confronted the tobacco industry in two statewide initiative campaigns in 1978 and 1980, proponents of the ordinance knew the real significance of the announcement and were prepared for what followed. Each tobacco industry campaign has certain predictable elements, and the Proposition P campaign followed the usual pattern:

- The major cigarette companies, through their public relations arm, the Tobacco Institute, hire a campaign manager, whose first task is to organize a local organization of "concerned citizens," which then becomes the nominal campaign organization. In a normal political campaign, a group of people forms a steering committee to act as a policy-making body and it, in turn, hires a campaign manager. The tobacco industry does exactly the opposite, because no grassroots or locally financed organizations opposed to nonsmokers' rights legislation have ever been formed. Moreover, the industry seeks total control over policy matters, and the way to accomplish that is to hire its own campaign manager.
- The tobacco companies contribute to the campaign in direct proportion to their respective market shares. This is a key indication that the campaign is actually being run by the industry itself—as a single entity—and not by the local organization.
- The tobacco companies go to great lengths to downplay both the extent and nature of their involvement. They deny that they are doing anything other than making financial contributions to a local campaign organization and they grossly underestimate the amounts of money they are contributing. They also delay in making the largest contributions until the end of the

---

Mr. Hanauer is an editor of law books and a past-president of the Board of Directors of Californians for Nonsmokers' Rights.

Address correspondence to Mr. Hanauer, Californians for Nonsmokers' Rights, 2054 University Ave, Suite 500, Berkeley, CA 94704 campaign so as to minimize any adverse publicity.

- The tobacco companies studiously avoid the subject of the health hazards of second-hand smoke and attempt to steer the debate to such issues as government regulation and the costs to taxpayers of enacting the law. They do this in a manner designed to convince the voters that extremely harmful consequences will necessarily flow from the law. For example, it is usually predicted that smokers will be arrested while rapists and robbers go undetected, that people will be driven out of business, and that vital social services will be shortchanged in order to pay for the law.
- Two important secondary themes are always present to back up the primary arguments: (1) the law represents an attack on civil liberties and denies smokers the freedom of choice; (2) the industry will admit there is a problem but claim the proposed law is not the right solution. Thus the slogan that appeared at the end of TV advertisements against Proposition P: "Proposition P is simply not the answer." The industry position is that to the extent second-hand smoke might be a minor annoyance to a few particularly sensitive people, "common sense" and "common courtesy"—not government regulation—will suffice. This has the double advantage of seeming to be understanding of the problem and at the same time depicting proponents of the law as well-meaning but sadly misguided.
- When the subject of the effects of second-hand smoke cannot be avoided, such as during live debates and press interviews, the tobacco industry representatives will deny there are any health hazards, often misquoting and quoting out of context medical authorities in the process. They will claim that even if some studies show second-hand smoke to be harmful, others do not, and thus the jury is still out. In other words, they imply, virtual unanimity in the medical community is needed before any laws should be passed that would limit smoking.
- The industry uses its economic and political power to secure endorsements from both individuals and organizations.
- On a general level, the industry's best weapon is confusion. As any political analyst will attest, when voting on ballot issues, a confused voter will invariably vote "No." Thus, a common thread running through all the industry's campaign advertising and debating is the attempt to confuse the public.

All of these factors came into play during the Proposition P campaign. The first public move was the press conference to announce that "Citizens Against Government Intrusion" (later changed to "San Franciscans Against Government Intrusion" doubtless when it was realized that the acronym for the name would have been CAGI) would place a referendum on the ballot to repeal the workplace smoking ordinance. The group announced that Jim Foster, the founder of one of the city's gay Democratic clubs (San Francisco has the largest gay community in the United States), would manage the campaign and that, at the group's request, the tobacco industry would contribute $40,000 to fund their petition drive because of "insufficient time to raise funds locally."

SLEIGHT OF HAND

None of the people at the news conference had ever testified against the ordinance, and except for Foster, none of them was heard from again during the campaign. A few days later Foster stated in a newspaper interview that he had been recruited for the campaign by the Tobacco Institute's attorney. Another person who lost out on the job later revealed that he had been interviewed by a vice-president of the Tobacco Institute. The $40,000 figure, which was repeated in every interview on the subject over the next two weeks, was untrue. The industry had pledged more than $100,000, and, in fact, $97,000 was spent to put the measure on the ballot. The tobacco industry, however, denied any involvement in the campaign other than as a financial supporter and at one point stated that subsequent funding by the industry would be contingent on a demonstration of local support. The industry ended up putting $1,250,000 into the campaign; the local contributions amounted to $3,300.

The money spent by the "No on P" campaign set a new national record for a local ballot measure (surpassing the $1.1 million the tobacco industry spent in Miami in 1979 to defeat a similar ordinance). To find out firsthand why the tobacco companies were contributing so much money to overturn a local health regulation, Supervisor Nelder held a press conference early in the campaign to announce that she had sent telegrams to the presidents of four cigarette companies challenging them to debate her on the merits of the law. None replied.

The petition drive itself was a travesty of the democratic process. Since the tobacco industry was not likely to find volunteer signature gatherers, it hired a professional petitioning company from Los Angeles and paid petition circulators as much as $0.70 per signature. One method used to collect signatures was for the paid circulators to explain that they were merely trying to put the measure on the ballot for a public vote—rather than to repeal a law that had already been enacted. At least two of the paid circulators (who had obtained several thousand signatures between them) were not bona fide registered voters of San Francisco, as required by the election laws. Ironically, when the deceptive practices of the tobacco industry were brought to the attention of the press, they were so confused by the referendum procedure (they could not understand that the tobacco industry put the measure on the ballot in order to repeal an existing law), that they made it appear that the supporters of the ordinance had committed the improper practices.

OBJECTIVE: HOME RULE

The campaign organization in favor of Proposition P was formed in August under the same "San Franciscans for Local Control." The decision was to make the central issue of the campaign the attempt by a large out-of-state special interest group to overturn a local health ordinance. The organization included representatives from Californians for Nonsmokers' Rights, the San Francisco Chapter of the American Cancer Society, the San Francisco Lung Association, the Sierra Club, and Common Cause, as well as several political activists. The proposition was soon endorsed and supported by a number of prominent individuals and organizations, including the San Francisco Police Officers Association.

Early in the campaign, a local attorney appeared on a television debate with Supervisor Nelder and claimed that imposing restrictions on smoking in the workplace would set a precedent for denying employment to gays on the grounds that they might have AIDS. This was perceived as a crass attempt by the tobacco industry to scare the large gay community, and several gay leaders denounced the tactic. The argument was not raised again, and the attorney never appeared again on behalf of the "No on P" campaign.

The tobacco industry's efforts settled on the following allegations: that the law constitutes government intrusion into the private workplace; that the law is unnecessary and the matter of smoking should be worked out privately; and that the law fosters "one-man rule" by giving a single nonsmoker the power to dictate personal behavior by all the other people in the office. One of the first pieces of literature by the tobacco industry told citizens that they should be angry that they were being forced to vote in an unnecessary election, thereby heaping scorn on proponents of the ordinance for what the tobacco industry itself had engineered. They claimed that the law is discriminatory because it applies to all private employees but only 20% of public employees—in other words, city as opposed to state and federal employees. Actually, the City Attorney had determined that the city did not have the power to enforce the law in state and federal offices and, in any event, all state employees were already protected by a state law. The most outlandish allegation came in response to the growing public awareness that the "No on P" campaign was receiving 99.7% of its money from out-of-state tobacco companies. One "No on P" representative charged during a debate that proponents were getting 94% of their money from outside San Francisco, and this absurd claim was continued throughout the campaign.

Probably the most important endorsement would be that of the San Francisco Democratic Central Committee. The Committee had solidly supported the two statewide initiatives for nonsmokers' rights in 1978 and 1980, and there seemed to be no reason why they would not endorse the ordinance, particularly since it had the backing of the Mayor. But no one reckoned on just how much pressure would be brought to bear, and the committee endorsed "No on P" by a vote of 15 to 14. Just how much that endorsement meant was brought home the day after the election, when the contact person for the "Yes on P" campaign in the Mayor's office said that her own husband was so confused upon receiving the voting recommendations from the Democratic Central Committee that he almost voted against the proposition.

Perhaps the saddest moment in the campaign came when *The Bay Guardian*, a liberal biweekly newspaper, which had been the only newspaper in the state to support the 1978 statewide initiative and one of the few to support the 1980 initiative, announced its opposition to Proposition P. Neither the publisher nor his support for nonsmokers' rights had changed, but the perceived threat of the loss of cigarette advertising evidently became overwhelming.

UNDERSTANDING MASS MEDIA

Proponents of the ordinance developed three key components of their campaign. First, Edgar Spizel, a media consultant and producer of thousands of radio and television commercials, volunteered his services. The "Yes on P" television commercial he created featured a tough-looking cowboy riding a horse on a San Francisco street and decrying the fact that the tobacco companies were spending 99% of the money to overturn the new smoking ordinance. At the end he repeats the campaign slogan: "Tell the tobacco companies to butt out!" (The advertising community named it the best television commercial in Northern California for 1983.)

AUDIO (western music up and under)
You know, we San Franciscan's are a
proud bunch. We like to do things
our own way.
So when some folks, like the four
leading tobacco companies from out
of state, put up 99.5% of the dollars
to overturn our new smoking ordinance,
well, that just makes me mad.
If you're as mad as I am, why not join
me in telling these tobacco companies
to butt out.
Vote Yes on P
(music up and out)

The second key element was to force the tobacco industry to change the taglines at the end of the "No on P" radio and TV advertisements to say "paid for by the tobacco industry" rather than "paid for by San Franciscans Against Government Intrusion." The Federal Communications Act of 1934 requires broadcast stations to identify the true sponsor of all political advertisements. Using all volunteer attorneys, headed by Paul Loveday, who had led the two statewide initiative campaigns, proponents put together a 30-page memorandum (and more than 100 pages of supporting documents), which was then presented to all the stations broadcasting "No on P" commercials. Within two days of receipt of this material, KRON-TV, the NBC affiliate and the station with the largest amount of "No on P" campaign advertising, informed San Franciscans Against Government Intrusion that if they did not modify the tagline, the advertisements would be taken off the air. KNBR (NBC radio) followed suit. The tobacco industry then changed the tagline for KRON to read, "paid for by San Franciscans Against Government Intrusion, which is funded by companies in the tobacco industry." They reluctantly agreed to this because the wording was visual only and in relatively small type, but they refused to do it for radio and thereby lost their largest radio buy for the last three weeks of the election. This was the first time that any radio or television station in the country had acted to require such a tagline change without having been ordered to do so by the Federal Communications Commission. However, none of the other stations acceded to the request.

The third major component of the campaign, a voter contact mailing program, was where much of the money was spent. While free radio and television time was obtained under the Fairness Doctrine, $50,000 was put into a mailing program. Three hundred thousand pieces of mail in seven versions were targeted to particular groups within a larger population of probable voters. The two most important mailings were one featuring pictures of six celebrities who had died of cancer and suggesting that they would all vote for Proposition P if they were alive, and one entitled, "The Big Tobacco Companies Have Lied To You 10 Times," in which 10 lies of the tobacco industry were documented.

As the campaign drew to a close one humorous and telling incident stood out. A week before the election the local public broadcasting station telecast a program focusing on the campaigns for two ballot measures—Proposition P and one aimed at imposing a moratorium on high-rise development. One of the staff people whom the tobacco industry hired for its "No on P" effort was also the campaign manager of the anti-high-rise proposition. It had been assumed that he took the tobacco industry job so he could defray the management expenses for the other proposition, which had a very low budget. Indeed, he spent very little time campaigning against Proposition P, and it is doubtful the tobacco industry was getting its money's worth. As the television show focused on the anti-high-rise proposition, it showed this individual sitting behind a battered old desk, in a spartan room, bemoaning how his barebones campaign

The basics of beating the tobacco industry

ISSUES CERTAIN TO ARISE

Health. The deleterious effects of secondhand tobacco smoke are the raison d'être for any law limiting smoking. Proponents of the law should have a firm grasp of all the relevant evidence, particularly the most reliable studies showing a link between secondhand smoke and serious diseases, as well as other studies that may be less conclusive. Even though the health issue is the one in which the proponents stand on the most solid ground, the tobacco industry can confuse voters (as it has even on the issue of smoking's role in lung cancer and heart disease) by claiming that "proof" is lacking and that more "research" is needed.[1-3]

Costs. The tobacco industry will claim that any law that restricts smoking will be too expensive both to government and private industry. The purported cost factors will include the posting of signs, enforcement, and disruption of business operations. The industry will claim that the law will force some businesses to close or relocate. Proponents can safely rely on the experience in communities in which such laws have already been in effect to demonstrate that any costs are negligible.

Enforcement. Whether the law is to be enforced by the police or a health authority, the industry will raise the spectre of important criminal or health matters being ignored while smokers are being hauled off to jail. The fact is that the law is intended solely as a recourse in the event of a dispute over the right to smoke in the presence of others who may be adversely affected. In all places where such laws have been enacted, they have proven largely self-enforcing, and few fines or citations have been issued.[4]

Government regulation. The tobacco industry's argument that nonsmokers' rights legislation represents government intrusion into essentially private matters has proven to be an effective weapon. The counter argument must begin with the fact that the law is a measure to protect public health, which is one of the most important functions of government.

Civil liberties. The tobacco industry maintains that any infringement on the right to smoke is a violation of civil liberties. (In the black community advertising by the industry suggests that restrictions on smoking are a first step in bringing back segregation.[5] To Miami's Jewish community the tobacco industry warned "Don't let it happen here."[5]) The industry also compares clean indoor air laws to Prohibition. Such a comparison is not difficult to rebut, for there are numerous governmental restrictions on the use of alcohol to protect public health and safety that have almost universal support.

Scope of law. No matter how limited or comprehensive the proposed law may be, the tobacco industry will oppose all restrictions on smoking that may cut into cigarette sales. The industry will claim either that the law is discriminatory because it applies to some businesses and not to others or will claim that it is too sweeping and repressive.

Campaign financing. The sudden infusion of enormous funds from the tobacco industry to defeat clean indoor air laws will become a major issue in the campaign. The industry never enters a campaign halfheartedly and always provides nearly every penny of the opposition campaign. The proponents of the law must exploit this from the beginning of the campaign and must make the public and press realize that the opposition campaign and the tobacco industry are one and the same. Many of the local contributors to the opposition campaign are also likely to have direct financial ties to the sale of tobacco products.

Endorsements. Closely connected with the issue of campaign financing is the question of who supports and who opposes the law. Apart from the tobacco-growing and manufacturing states of Connecticut, Florida, Georgia, Kentucky, Maryland, North Carolina, South Carolina, and Virginia, or New York (where three of the six United States cigarette companies are headquartered)[6] voters will be interested to learn that the opposition is led by out-of-state interests. Moreover, as such legislation gains in popularity, local public figures are increasingly eager to lend their names to the campaign. The contrast between the quality and quantity of local support versus that of the outside tobacco interests is striking.

CAMPAIGN STRATEGY

Build a coalition. Essential to the passage of clean indoor air measures is was being steamrollered by the highly financed opposition, backed by developers and major corporations. Moments later the same individual was photographed in the elegantly furnished "No on P" headquarters in front of a sophisticated computer, which was putting out information on voter support by neighborhoods.

Despite the huge expenditures and deceitful tactics of the cigarette companies, they were beaten in this campaign because they encountered a resourceful group of nonsmokers' rights advocates who had been through the wringer twice before and who knew what to expect and what had to be done to win. It is hoped that their experience will be of help to people in other communities who might square off against the tobacco industry in the future. The accompanying article on issues to be faced and campaign strategy is designed to provide the basics of running a campaign.

The final tally in the election was 80,740 to 79,481—a remarkable victory in light of the overwhelming odds. As a result, Californians for Nonsmokers' Rights has succeeded in lobbying other communities throughout California to pass similar laws, the most recent of which is Los Angeles, whose strict ordinance was signed by Mayor Tom Bradley exactly one year after the vote on Proposition P. Perhaps with the possibility of a chain-reaction in mind, the tobacco industry tried one last desperate measure after the election. On the following Sunday the industry ran a full page newspaper advertisement complaining that the close vote was not sufficient to warrant imposition of such a controversial law and asking people to urge their legislators to amend Proposition P. The cigarette companies should have saved their money, for by placing the referendum on the ballot they had insured, under the terms of the City Charter, that the law could not be touched for a year. By that time, a news story in the anti-regulatory *Wall Street Journal* (August 15, 1984) would be pronouncing the law a success.

---

a broad base of support among constituencies such as health agencies, environmental organizations, and public interest groups. Environmentalists appreciate learning or being reminded that tobacco smoke is the largest contributor to indoor air pollution. Getting as many people from different organizations involved as early as possible gives them the feeling that they are part of the decision making process and gives their organizations a stake in the campaign.

Campaign staff. With the exception of a close-knit community where it is still possible for local values to prevail against commercial pressures from outsiders, a campaign cannot be run against the tobacco industry without a competent full-time staff. It is important to hire people who are committed to the issue.

Fund-raising. Although it is not possible to compete dollar-for-dollar with the tobacco industry for campaign money, there may be no need to. The tobacco industry can be beaten, even when it outspends the opposition by 10 to one. On the other hand, a minimum amount of money must be raised in order to run a creditable campaign. This means mounting a full-scale fund raising effort using both direct mail and person-to-person contact.

Campaign focus. The most effective issue for proponents of a local or state nonsmokers' rights law is the involvement and virtually complete financing of the opposition by the tobacco companies. The proponents' campaign should never focus on the tobacco industry's theme song of costs, enforcement, and government intrusion. A campaign that can focus the voters' attention on its issues (in this case, health and tobacco industry money) will stand a good chance of winning; a campaign that spends its time answering charges by the other side will inevitably lose.

Endorsements. It is important to secure key local endorsements early. Many subsequent endorsements will depend on who has already endorsed the initiative. Some individuals who may have no strong opinion on the issue may give their endorsement to the first person who approaches them with a reasonable presentation. Police, sheriffs', and firefighters' organizations have been prime early targets of the tobacco industry. For the proponents, enlisting the full commitment of the local medical association and specific physician-spokespersons and other health professionals for hearings, press conferences, and other public activities is imperative. Regrettably, there are too few physicians with sufficient political and media experience.

Fairness time. The tobacco industry will flood the airwaves with advertising. Nevertheless, the law requires that all radio and television stations must give each side of a ballot measure fair time (but not equal time) to present its views. Some stations attempt to fulfill this obligation by giving the "poor" side of a campaign a chance to present its views on an off-hours public affairs program, but others will give between one-fourth and one-third of the advertising time purchased by the tobacco industry. The effort to obtain this time should begin early, and legal advice should be obtained on how to pursue it most effectively.

Radio and television taglines. By law every radio and television advertisement must identify the source that paid for it. In seeking to defeat clean indoor air measures, the tobacco industry invariably organizes under a misleading name of an apparent local group (for example, "FAIR, Floridians Against Increased Regulation"). A challenge can be made to the use of such a name as the true sponsor of the advertisements. Even if the effort does not succeed in forcing a change in tagline, it can generate a great deal of journalistic scrutiny which will further highlight the issue of tobacco industry involvement.

Investigation of tobacco industry claims. Claims by the tobacco industry concerning the costs of implementing and enforcing the legislation as well as the "evidence of medical experts" should be fully investigated and exposed for whatever fraud is present. For example, tobacco industry claims relating to medical evidence often involve either a misquote or a quote out of context. It is important that the press and public be made aware of any such fraud as early and often as possible.

REFERENCES

1. *On Tobacco: 21 Questions and Answers.* Washington, DC, The Tobacco Institute, 1981.
2. *The Smoking Controversy: A Perspective.* Washington, DC, The Tobacco Institute, 1978.
3. *The Facts About . . . Public Smoking.* Winston-Salem, RJ Reynolds Tobacco Co, 1976.
4. Kahn PL: The Minnesota Clean Indoor Air Act. *NY State J Med* 1983; 83:1300-1301.
5. Tate Jr CF: A physician-led referendum for cleaner air in Florida. *NY State J Med* 1983; 83: 1302.
6. Blum A: Cigarette smoking and its promotion: editorials are not enough. *NY State J Med* 1983; 83: 1245-1247.

LIE #1: **The Tobacco industry lied about why Proposition P is on the ballot.**

The industry claims San Francisco faces "still another unnecessary vote on November 8" and implied Proposition P was put on the ballot by its supporters.
(Source: No on Proposition P support solicitation letter, September 26, 1983)

TRUTH: **The tobacco industry is responsible for this unnecessary vote.**

When the San Francisco Board of Supervisors adopted the workplace smoking ordinance on a vote of 10 to 1, smokers and nonsmokers throughout the city praised the law as even-handed and fair. The tobacco industry, sensing the law might have an effect on its sales, paid to put Proposition P on the ballot.
(Source: No on P support solicitation letter, September 25, 1983)

LIE #2: **The tobacco industry lied about government's role in enforcing Proposition P.**

[illegible] P will lead to government intrusion on [illegible]

TRUTH: **Proposition P is an entirely self-enforcing law.**

It simply requires that employers adopt a smoking policy to satisfy smokers and nonsmokers. There will be no government intrusion.
(Source: The Workplace Smoking Ordinance)

LIE #3: **The tobacco industry lied about who is affected by Proposition P.**

The tobacco industry claimed Proposition P is discriminatory because it covers all private employees but only 20% of public employees.
(Source: [illegible] campaign brochure)

TRUTH: **Proposition P does not cover all private employees and does not exempt government employees.**

The ordinance covers only office employees. It does not apply to construction workers, service employees or restaurants. Proposition P will extend to all local government offices. State office employees already are covered by a similar law, while the federal government must, by law, establish its own office smoking criteria. Proposition P is not discriminatory.
(Source: The Workplace Smoking Ordinance, Government Code Section 19262)

LIE #4: **The tobacco industry lied about the use of public funds.**

[illegible]

TRUTH: **Proposition P will cost nothing to the taxpayer.**

The San Fr[illegible] determined that Proposition P [illegible]

LIE #5: **The tobacco industry lied about workplace discrimination.**

The tobacco industry claimed "executives with private offices are not controlled by this law, yet secretaries, bookkeepers, key-punch operators, and every other office worker . . . must comply with the law."
(Source: [illegible] Voters' Handbook)

TRUTH: **Proposition P does not promote workplace discrimination.**

All office workers are covered by Proposition P from the highest-paid executive to the lowest-paid clerical worker.
(Source: The Workplace Smoking Ordinance)

LIE #6: **The tobacco industry lied about employee bargaining rights.**

[illegible]

TRUTH: **Proposition P broadens employees' bargaining rights.**

Currently, [illegible] smoking, there is [illegible] Proposition P [illegible] employees [illegible] Proposition P requires that [illegible] be devised.

LIE #7: **The tobacco industry lied about the City Attorney's role.**

One of the tobacco industry's ballot arguments pointedly asks "Should our City Attorney be investigating cigarette smoking instead of processing the heavy legal workload of the city?"
(Source: [illegible] Voters' Handbook)

TRUTH: **The City Attorney will not investigate cigarette smoking.**

Proposition P is an entirely self-enforcing law. The City Attorney's office will not investigate and will not use any public funds to enforce compliance with Proposition P. The City Attorney's workload will not be affected.
(Source: San Francisco Controller's Office, the Workplace Smoking Ordinance)

LIE #8: **The tobacco industry lied about the effects of second-hand tobacco smoke.**

[illegible]

TRUTH: **Second-hand smoke can be deadly!**

The U.S. Surgeon General has determined that second hand smoke is hazardous to our health. Experts throughout the country agree second hand smoke may be a cause of lung cancer and respiratory ailments.
(Source: [illegible] American Lung Association [illegible] Second-hand Smoke)

LIE #9: **The tobacco industry lied about its petitions.**

The tobacco industry claimed the 40,000 signatures they gathered on petitions "represents a great deal of interest on the part of the voters to place this issue on the ballot."
(Source: San Francisco Chronicle, July 2, 1983)

TRUTH: **The tobacco industry had to pay its petitioners to put Proposition P on the ballot.**

In all, the tobacco industry had to pay professional signature-gatherers $117,000 just to get the measure on the ballot. That is nearly $3.00 for every signature they received. In fact, two petitioners who received a total of $15,000 to gather signatures, registered to vote in San Francisco just days before the signature period began. Once that period was over, the two petitioners were registered to vote in San Diego in order to collect signatures for another political campaign.
(Source: San Francisco and San Diego Registrar of Voters)

LIE #10: **The tobacco industry lied about its local support.**

[illegible] contributions from local [illegible] they would donate themselves [illegible]

TRUTH: **There is little opposition to Proposition P.**

More than 99% of the $237,000 raised as of September 19, 1983 to defeat Proposition P has come from out-of-state tobacco companies. Local sources have contributed less than 1%. On the other hand, most of the money raised in support of Proposition P has come from San Francisco.

Local values versus tobacco industry power

The experience of GASP of Massachusetts

Edward L. Sweda, Jr, Esq

From its fledgling beginning in 1972 as a discussion group on the problem of the effects of tobacco smoke on the nonsmoker, Group Against Smoking Pollution (GASP) of Massachusetts has become a catalyst for legislative and judicial activity.

Statewide Initiative

In 1978, as the first step in an effort to lobby the Massachusetts Legislature to pass a Clean Indoor Air Act, GASP led a coalition of more than a dozen health and public interest groups (including the American Lung Association, the American Heart Association, and the Massachusetts Department of Public Health) in a petition drive. The collection of several thousand signatures of registered voters put a nonbinding public policy question (which is intended to gauge the degree of support for a piece of legislation) on the ballot in 10 legislative districts throughout the state. The question read as follows:

> Shall the Representative from this District be instructed to vote for the passage of legislation which would assure citizens clean air by restricting smoking by the general public to separate and clearly posted areas and which would provide for fines in the event of noncompliance?

To counter voter support for the question, the Tobacco Institute, based in Washington, DC, established a group in Massachusetts called Independent Citizens for Effective Government, which sent letters to registered voters in the 10 districts. The letter, which contained no mention of the group's connection with the tobacco industry, claimed that passage of the question would prohibit smoking in such areas as sidewalks and on the Boston Common. Following the November election, records from the Secretary of State's office revealed that the Tobacco Institute had spent $80,000 to try to defeat the question. Although GASP had raised only $3,500, voters approved the question in each of the 10 districts, by a statewide average of 70% to 30%.

This public vote was the crux of GASP's argument when approaching legislators on a bill to expand the state law restricting smoking in public places. Of the seven representatives in those districts who had voted against such legislation in previous years, five signed on as co-sponsors of the bill in 1979. The bill passed in the Senate but was never released from the House Ways and Means Committee, whose chairman was the brother of a Tobacco Institute lobbyist.

Shift to Local Level

After a lack of legislative success in 1980, GASP began to look to local governmental bodies for establishment of clean indoor air legislation. In Newton, a Boston suburb, the Board of Aldermen had adopted a resolution urging local restaurateurs to set up no-smoking sections for their customers; most restaurant owners ignored the resolution. In 1981, an ordinance was filed to require a no-smoking section in Newton restaurants. GASP contacted its Newton members and urged them to support this proposal. The Board of Aldermen overwhelmingly passed the ordinance, which has become the model for the other 350 cities and towns in Massachusetts.

The first direct vote on this issue in four years occurred in 1982 in Acton (pop 20,000). Acton has a form of government whereby any registered voter who comes to the town meeting is part of the legislative body. Thus, when a proposed by-law to require a minimum 25% no-smoking area in all restaurants seating 25 or more persons came up for a vote, a local Tobacco Institute lobbyist joined forces with some restaurant owners. In addition to paying a local teenager to hand out leaflets, the tobacco lobbyist urged voters to defeat the proposal because, for one reason, some nonsmokers were even using small portable fans to blow smoke back at smokers. The by-law passed by a vote of 138 to 88.

A similar proposal was being considered by Brookline (pop 55,062), the home town of then-president of GASP, Rita Addison. It was here that the tobacco industry spared no expense to stop GASP's momentum. The proposed by-law had to wind its way through several committees and nine public hearings before it could be considered at the town meeting. In addition to using two local lobbyists, the Tobacco Institute flew in a public relations operative from Washington, DC, to address one of the advisory committees. By the time of the town meeting vote, the Tobacco Institute and several restaurant owners had formed Brookline Restaurateurs Opposed to Excessive Regulation (BROER). While GASP was waging a grassroots effort to lobby more than 200 town meeting members, BROER launched a vituperative personal attack on the originator of the proposal. This tactic backfired; by a voice vote, Brookline became the third Massachusetts community to guarantee no-smoking sections in restaurants. One of the town meeting members stood up during the meeting to chastise the tone used in the BROER mailing and to note that unlike the GASP members, none of the opponents were residents of the town.

Since then, the Tobacco Institute has permitted the Massachusetts Restaurant Association to take the visible lead in opposing similar laws. Nevertheless, 25 Massachusetts communities have passed laws requiring no-smoking sections in restaurants, as of June 1985.

Ending Street Corner Pushing

GASP of Massachusetts has also endeavored for many

Address correspondence to Mr Sweda, Legislative Secretary, GASP of Massachusetts, 25 Deaconness Rd, Boston, MA 02215.

years to outlaw cigarette sampling—the tobacco industry's practice of handing out thousands of free packages of cigarettes or smaller 5-pack or 10-pack "samplers" to passers-by on the street. According to the Federal Trade Commission, the tobacco industry in 1981 spent more than $81.5 million nationwide on cigarette sampling (FTC report to Congress pursuant to the Federal Cigarette Labeling and Advertising Act for the year 1981, July 1984, p 25). Such sampling campaigns provide children with easy access to cigarettes and put the product in a socially acceptable light on the public streets.

On January 11, 1984, the 20th anniversary of the Surgeon General's Report on *Smoking and Health*, Boston Mayor Raymond L. Flynn signed a proclamation declaring the day "Boston Clean Indoor Air Day" and pledged that the "Government of the City of Boston shall take action to reduce the harm that smoking inflicts on the well-being of the people of Boston." On that day also, an ordinance to ban cigarette sampling was filed by several city councillors. (Minneapolis and St Paul, MN, and Cambridge, Newton, and Somerville, MA have passed similar bans.)

Unable to ally itself with the restaurant industry on this issue, the tobacco industry brought a representative from a Long Island-based sampling company (the bulk of whose business is with cigarette companies) to testify against the proposal at a public hearing. This individual defended the practice of giving free packs of cigarettes on public thoroughfares by pointing to the large number of black and Hispanic individuals employed by his company.

In the three months between the testimony and the vote, two local Tobacco Institute lobbyists worked behind the scenes to kill the ordinance. They were successful in getting two of the original sponsors to vote against the ban. However, GASP, with assistance from lobbyists for the Mayor, won a significant victory when the Boston City Council passed the ordinance by a vote of 7 to 6. On signing it into law, Mayor Flynn said that the "unregulated distribution of tobacco products gives our children and teenagers too much easy and cheap exposure to this health danger."

A few months later, however, two councillors filed an amendment to add the words "to any person under twenty-one years of age" to the ordinance. If passed, cigarette samplers would be welcomed back onto the streets of Boston. The City Council, without a public hearing, passed the amendment by a vote of 7 to 6. However, in January 1985, Mayor Flynn vetoed the amendment, saying that it would "effectively repeal" the ordinance. (GASP found that six of the seven councillors who voted for the amendment had received contributions from tobacco lobbyists.) The mayor's veto was upheld.

In September 1984, the City Council of Worcester—Massachusetts' second largest city—considered a similar ban on cigarette sampling. The individual from New York again appeared, as did a tobacco lobbyist who argued that it is silly to try to protect children by banning cigarette sampling when the city failed to enforce the state law prohibiting the sale of cigarettes to minors. Representatives of the Worcester Health Department, GASP, the major voluntary health organizations, and the public testified in support of the ordinance. The council, each member of which had been approached by the tobacco lobbyist the day before, voted 8 to 0 for the ordinance and to instruct the City Manager to take steps to enforce the state ban on cigarette sales to minors.

A SAFER WORKPLACE

Throughout the winter and spring of 1984, the Newton Board of Aldermen considered an ordinance to protect nonsmokers' rights in the workplace. At least a half dozen meetings were held, not only to receive public input on the issue itself, but also to draft an ordinance that would best apply to the working conditions that prevail in Newton.

While GASP again relied on its local members for advocating the adoption of a strong ordinance, the Tobacco Institute sent two letters to the Newton business community, pleading that if "you agree that you and your employees can solve the question of smoking and nonsmoking employees in the workplace without the iron fist of government regulation, then I suggest that you contact each of the members of the Board of Aldermen and express your opposition to them *immediately*." Not only did those letters fail to produce the intended result, but several members of the Chamber of Commerce responded by asking their Aldermen to vote for the ordinance.

The final result was a new law, the first of its kind in Massachusetts, that required employers with two or more employees to develop and post a smoking policy. The law also permits dissatisfied employees to appeal to the Newton Health Commissioner who may revise the policy if it fails to give "due consideration to the health of the complaining employee and to the reasonable needs of the employer's business."

The other major arena in which GASP has confronted the tobacco industry has been the courtroom. In *Lee v Department of Public Welfare*, Bristol County Superior Court No. 15385 (1983), a social worker in the Attleboro office of the Massachusetts Department of Public Welfare sued her employer for injunctive relief against the tobacco smoke in her workplace. Through the Tobacco Institute, a coworker who smoked cigarettes acquired the legal services of three law firms: Covington and Burling, Washington, DC; Sullivan and Worcester, Boston; and Shook, Hardy and Bacon, Kansas City. The coworker has acknowledged that the Tobacco Institute paid the legal bills. The basis of her intervention in the case was the contention that she was so addicted to smoking cigarettes that she had to be allowed to smoke at her desk in order for her to get her work done.

Paradoxically, the Tobacco Institute denies that cigarettes are addictive. The importance of this case did not escape the tobacco industry. This single instance of judicial action to curtail smoking in a typical office so threatened to decrease cigarette sales that the Tobacco Institute was willing to have its lawyers contradict its official public relations denial of the addictive qualities of its product, in order to be able to play a direct role in this case.

The attempt by the employer, the Commonwealth of Massachusetts, to dismiss the case was denied. In a decision that serves as a major precedent in Massachusetts, Superior Court Judge Robert Mulligan ruled the following:

> An employer has no duty to make the workplace safe if, and only if, the risks at issue are inherent in the work to be done. Otherwise, the employer is required to "take steps to prevent injury

> that are reasonable and appropriate under the circumstances." . . . Accordingly, this court cannot say that the plaintiff's claim fails to make out a legally cognizable basis for relief.

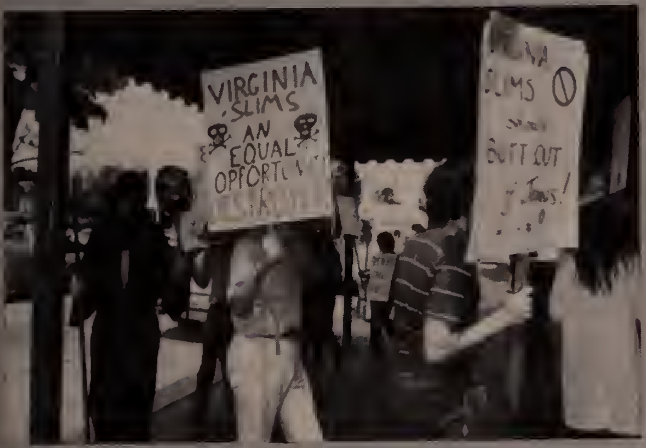
On January 8, 1985, the *Lee* case was settled by voluntarily dismissing the complaint based on assurances from the Department of Public Welfare that the nonsmokers in the Attleboro office will be provided both a separate no-smoking area in which to work and an entirely separate ventilation system so that none of the smoke from the smoking area will penetrate the no-smoking area. The lawsuit can be revived if the state reneges.

Acknowledgment. New England Deaconess Hospital has donated office space and other assistance to GASP since 1980.

Sham news and sports events exposed

The nonsmokers' rights movement turns to political theater as a means of calling attention to tobacco company-sponsored events. The tobacco industry has for many years attempted to improve its own public image by linking itself (by means of money) to other businesses and groups. Tobacco marketers have even pretended to be part of athletics, music, and news.

By November 1981, Philip Morris had launched the "Merit Report," a purported public opinion survey that questioned the public on virtually any subject—except smoking and health. The real purpose of the Merit Report was to promote Merit as the "intellectual's cigarette." The company sent computerized vans nationwide; one of the first stops was Boston's City Hall Plaza. While Deputy Mayor Katherine Kane welcomed Merit's van to Boston, more than twenty GASP picketers captured the lion's share of media attention that day, decrying the misuse of the public plaza to promote Merit cigarettes. Although there had been plans for the Merit van to return to Boston within a year, it never has. The van was given a similar welcome in 1982 in Springfield.



Protest by members of GASP of Massachusetts at Philip Morris' Virginia Slims Tennis Tournament, Newport, RI, July 1984.

GASP's appeal to Peter, Paul, and Mary thwarted RJ Reynolds' sponsorship of the "Camel Concerts on the Common" in the summer of 1982 (*NY State J Med* 1984; 84:162).

Perhaps the most outrageous tobacco-sponsored event of all is Philip Morris, Inc's Virginia Slims Tennis Tournament. Since 1978 when a group of 35 physicians and medical students from DOC (Doctors Ought to Care) paid a "house call" on the tournament in Miami, renaming it the "Emphysema Slims" matches, protests have been mounted in Washington, DC, New York, New Jersey, Boston, Newport, RI, and other sites on the circuit. At a Virginia Slims press conference to kick off the 1983 Boston visit, the then-President of GASP, Rita Addison, much to the consternation of the tournament's promoters, asked Billie Jean King how she could allow herself to be associated with a product that would soon cause lung cancer to overtake breast cancer as the number one cancer killer of American women. Media coverage of the press conference focused on this issue and King's praise for Philip Morris.

Logo of Emphysema Slims Tennis Tournament sponsored by medical students of the DOC chapter at the Medical College of Georgia, Augusta, March 1985. More such events are planned to call attention to the hypocrisy of tobacco company sponsorship of sporting events. At the tournament former Surgeon General Jesse Steinfeld, MD, presented DOC's First Annual W.A.R.M. Award (Worst Athlete Role Model) to tennis player Martina Navratilova for having worn the logos of brands of cigarettes on her tennis dress.

GASP has protested each of the tournament's 1983 and 1984 visits to Boston and to Newport, and has distributed hundreds of leaflets to spectators. Virginia Slim's 1985 visit to Boston was canceled; in February 1986 it will come to Worcester. —ES

An assessment of the effectiveness of nicotine chewing gum: unfulfilled expectations

JOSEPH FELDMAN, DRPH

There is a tendency to evaluate measures to reduce smoking less critically than might otherwise be the case because of the many detrimental effects of smoking. Such a tendency can lead to unrealistic expectations and possibly other side-effects if the measure involves a pharmacologic agent. This appears to have happened in the evaluation of nicotine chewing gum as an aid to stop smoking. For approval, the Food and Drug Administration (FDA) required two adequate and well controlled prospective studies showing a statistically significant difference between users and non-users of the gum. This requirement allows considerable room for judgment as to what constitutes "adequate." The two studies that formed the basis for FDA approval[1,2] did not adequately document a benefit of nicotine gum, although both were well controlled prospective studies showing significant differences between the groups tested. Some of the reasons for the inadequacy of these studies have already been described.[3] The present discussion will expand on these and comment on additional data and issues of methodology.

DOUBTFUL RANDOMIZATION

The most rigorous type of evaluation is the double-blind randomized control trial. Both of the studies noted above used this type of design. However, the use of such a design does not mean that the results apply universally. In such a trial, the results apply only to the target population—the population from which the sample subjects were drawn. For example, the classical randomized trial of antihypertensive medication was conducted among a sample of patients in a Veterans Administration hospital.[4] There was no doubt that the antihypertensive medication effected a significant reduction in mortality from hypertensive complications. But this unequivocal observation in a sample of male patients could not automatically be applied to female subjects. A second randomized trial that included women in the target population was required before inferences could be made.[5]

With nicotine chewing gum, several randomized trials have shown a higher rate of smoking cessation than other methods,[1,2,6-8] but each of these trials recruited highly selective subjects, and in some there were two levels of selection. In the British study (one of the two on which FDA approval was based) there were 116 subjects who attended a hospital smokers' clinic for treatment during a one-year period, and who agreed to participate in the trial.[1] One level of selection was based on willingness to volunteer to attend a smokers' clinic, and the second was agreement to participate in the trial. The authors did not indicate how many subjects refused to participate in the trial, and so it is not possible to estimate either level of selectivity. In the other study accepted by the FDA, subjects were recruited from a university campus and a nearby community by advertising campaigns.[2] The volunteers then participated in an eight-week pretest period. A total of 208 subjects participated, but again the reader is not told how many volunteers dropped out during the pretest period. In another study smokers were recruited through advertisements on a radio station's news broadcasts. Ninety-six of the volunteers participated, but again the reader is not told how many subjects failed to follow through. In another widely quoted study[7] 100 consecutive patients at a smoking withdrawal clinic were included in the trial. Patients were placed on the clinic's waiting list for a time period of two to three months. Therefore, subjects who participated in this study were not only volunteers, but they also had to have the perseverance to want to stop smoking for at least two months while on the waiting list. In a recent Swedish study subjects were recruited from a smoking cessation clinic in which the great majority of participants had smoking-related disorders such as chronic bronchitis, emphysema, asthma, and cardiovascular diseases.[8] Among this selective group half were excluded usually because of previous use of nicotine chewing gum.

Each of these trials was a randomized double-blind control trial that found a statistically significant difference in rates of cessation of smoking between users of nicotine chewing gum and subjects given a placebo. Yet none of these studies was adequate in the sense that the findings could be generalized to the smoking population.

Other factors in these trials also limit the applicability of their findings. For example, each of the studies combined the use of nicotine chewing gum with extensive counseling and support services. In one study a separate comparison was made of nicotine gum plus counseling versus nicotine gum without counseling.[6] In the absence of counseling, the gum was not effective.

CONSISTENT DRAWBACKS

Another issue that requires more study is the duration of cessation among gum users. Most studies have found that the benefit from nicotine chewing gum declines with the duration of follow-up.[2,6-8] Since all of the studies had a short follow-up period, usually one year or less, it is not

---

Address correspondence to Dr Feldman, Associate Professor, Department of Preventive Medicine and Community Health, SUNY-Downstate Medical Center, 450 Clarkson Ave, Brooklyn, NY 11203.

known whether the gain from nicotine gum will be permanent.

Three of the studies used a one-tailed test to determine statistical significance.[1,2,7] Some of the differences between gum users and non-users would not have been significant if two-tailed tests had been used. Theoretically a one-tailed test is appropriate if a difference in one direction has no importance or cannot conceivably occur. In the current context this implies that the patients using nicotine gum either could not do worse than non-users or that such an observation would be of no interest. Whether this situation applies to nicotine chewing gum is debatable. Some have noted that the rationale for a one-tailed test rarely occurs in practice.[9,10]

Two more recent studies to evaluate nicotine gum have attempted to sample from a broader target population.[11,12] In one of these, cigarette smokers visiting a general practitioner for reasons other than smoking cessation were invited to participate in a study to evaluate nicotine gum.[11] Of those invited 1,938 (89%) participated. These subjects were randomly allocated to either a control group with no intervention, a group that received doctor's advice and a booklet, or a group that received doctor's advice, a booklet, and nicotine gum. After statistical correction for refusals, failed biochemical validation, or switching to cigars or pipes, the proportions of subjects who had abstained from smoking after four and 12 months were 4% in the first two groups and 9% in the group that used the gum. Although the quit rate in the gum group was significantly higher, the authors were careful to avoid attributing this difference to pharmacologic factors. They noted that the study was not designed for that purpose and that it did not contain a placebo control group.

The most general double-blind randomized control trial failed to find a difference between the quit rates of subjects given a doctor's advice with or without the nicotine gum. This study included 1,550 patients between the ages of 18 and 65 years who smoked cigarettes and had visited the doctor for a condition related to or aggravated by smoking. Less than 4% of the subjects refused to participate. The trial involved multiple facilities, and subjects were randomly allocated to four groups: doctor's advice only; doctor's advice and booklet; doctor's advice and booklet and placebo gum; or doctor's advice and booklet and nicotine gum. A patient was considered to have quit if he or she reported not smoking at both six and 12 months; this was verified with biochemical tests at those times. In each of the four groups about 10% stopped smoking. Thus, the only double-blind randomized trial that was not limited to volunteers failed to find an effect of the nicotine gum. Even this study was selective in the sense that motivation to quit smoking may have been greater, since the subjects suffered from cigarette-related diseases from which they were seeking relief.

In another double-blind trial in a general practice, 429 smokers who had attempted and failed to give up smoking in the previous year were invited to participate.[13] Two hundred agreed; half were given nicotine gum and half were given a placebo gum. After six months, 10% of the subjects using the nicotine gum had stopped smoking, compared to 8% of the subjects in the control group. The authors concluded that the value of nicotine chewing gum is minimal when used in general practice.

Proceed with Caution

Nevertheless, nicotine gum may serve a useful purpose when coupled with supportive counseling for certain subgroups, such as highly motivated smokers or smokers highly dependent on nicotine. In one study, smokers using nicotine gum who scored high on a nicotine dependence questionnaire were five times more likely to remain abstinent than patients with high dependence who received placebo gum.[14] However, for those with low dependence, there was no difference in abstinence between nicotine gum and placebo users. The danger is that the gum will be used indiscriminately, possibly in lieu of an adequate support system. There is also the danger that the patient will form the impression that little effort beyond chewing the gum is required to stop smoking. One author has concluded that if a physician takes 60 seconds to tell a patient that cigarette smoking is ruinous, 3.5 million smokers would quit; if in addition the physician prescribes nicotine chewing gum, another 3.5 million smokers would quit.[15] This type of false optimism seems based more on wishful thinking than on hard data and is bound to lead to unfulfilled expectations. A more balanced appraisal has reminded physicians that the efficacy of the gum in general medical practice, the incidence of dependence and identification of the persons for whom the gum is effective remain to be resolved.[16] Until these data are available it would be prudent to proceed with caution in prescribing nicotine chewing gum.

References

1. Jarvis MJ, Raw M, Russell MAH, Feyerabend C: Randomized controlled trial of nicotine chewing-gum. *Br Med J* 1982; 285:537–540.

2. Christen AG, McDonald JL, Olson BL, Drook CA, Stookey GK: Efficacy of nicotine chewing gum in facilitating smoking cessation. *JADA* 1984; 108:594–597.

3. Blum A: Nicotine chewing gum and the medicalization of smoking. *Ann Intern Med* 1984; 101:120–122.

4. Veterans Administration Cooperative Study Group On Antihypertensive Agents: Effects of treatment on morbidity in hypertension. II. Results in patients with diastolic blood pressure averaging 90 through 114 mm Hg. *JAMA* 1970; 213:1143–1152.

5. Hypertension Detection and Follow-Up Program Cooperative Group: Five-year findings of the Hypertension Detection and Follow-Up Program. II. Mortality by Race-Sex and Age. *JAMA* 1979; 242:2572–2577.

6. Schneider NG, Jarvik ME, Forsythe AB, Read LL, Elliott ML, Schweiger A: Nicotine gum in smoking cessation: a placebo-controlled, double-blind trial. *Addict Behav* 1983; 8:253–261.

7. Fagerström KO: A comparison of psychological and pharmacological treatment in smoking cessation. *J Behav Med* 1982; 5:343–351.

8. Hjalmarson, AIM: Effect of nicotine chewing gum in smoking cessation. A randomized, placebo-controlled, double-blind study. *JAMA* 1984; 252:2835–2838.

9. Armitage P: *Statistical Methods in Medical Research*. London, Blackwell Scientific Publications, 1971, p 104.

10. Fleiss J: *Statistical Methods For Rates and Proportions*, 2nd ed. New York, John Wiley & Sons, 1981, p 29.

11. Russell MAH, Merriman R, Stapleton J, Taylor W: Effect of nicotine chewing gum as an adjunct to general practitioners' advice against smoking. *Br Med J* 1983; 287:1782–1785.

12. Report by a subcommittee of the Research Committee of the British Thoracic Society: Comparison of four methods of smoking withdrawal in patients with smoking related diseases. *Br Med J* 1983; 286:595–597.

13. Jamrozik N, Fowler G, Vessey M, Wald N: Placebo controlled trial of nicotine chewing gum in general practice. *Br Med J* 1984; 289:794–797.

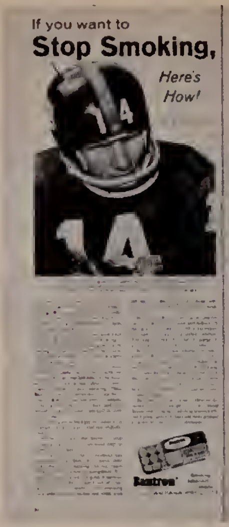
14. Jarvik M, Schneider N: Degree of addiction and effectiveness of nicotine gum therapy for smoking. *Am J Psychiatry* 1984; 141:790–791.

15. Sachs OPL: Office strategies to help your patients stop smoking. *J Respir Dis* 1984; 5:35–40, 44, 46–48.

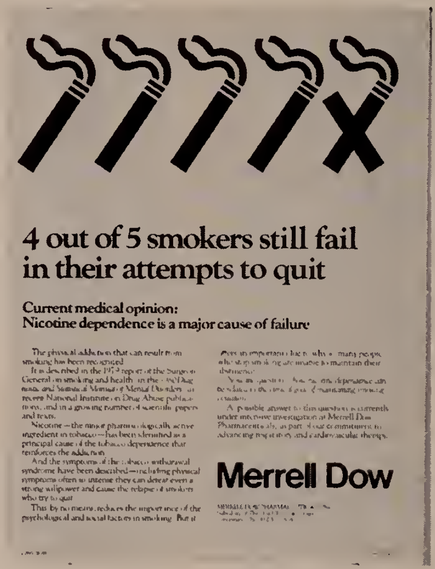
16. Hughes JR, Miller SA: Nicotine gum to help stop smoking. *JAMA* 1984; 252:2855–2858.

Nicotine chewing gum is the latest in a long line of chemical agents for smoking cessation. Other pharmacologic approaches such as the nicotine substitute lobeline (sold as Bantron or Nikoban) have been backed by major advertising campaigns. On January 22, 1964, one week after the release by the Surgeon General of the landmark report on *Smoking and Health*, M. R. Thompson, Inc., manufacturers of Nikoban, a lobeline sulfate lozenge, held the First Annual M. R. Thompson Symposium on Recent Advances In the Medical Aspects of Smoking. Two speakers reported smoking cessation rates at two months of 90% and 50%, respectively with Nikoban. In contrast Schwartz' review (*Pub Health Rep* 1969; 84:483–506) of more than 20 studies of lobeline found only 8 in which placebo was used as a control; and only two of these showed better results for lobeline than for placebo.

In 1983, prior to the approval of Nicorette by the FDA, Merrell Dow laid the groundwork for the gum's introduction with advertisements in medical journals that pinned the blame for failure in smoking cessation on nicotine dependence. After the gum was approved, advertisements portrayed it as a unique prescribable treatment for nicotine dependence. Numerous advertisements and press releases in the mass media suggested that physicians were at last able to treat smoking successfully.

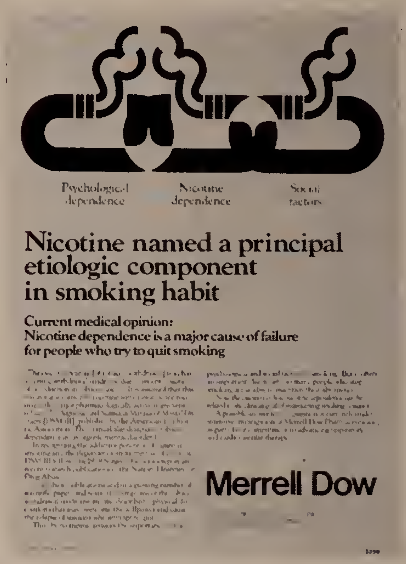

If you want to
Stop Smoking,
Here's How!

RECENT ADVANCES
IN THE
MEDICAL ASPECTS
OF SMOKING


4 out of 5 smokers still fail in their attempts to quit

Current medical opinion:
Nicotine dependence is a major cause of failure

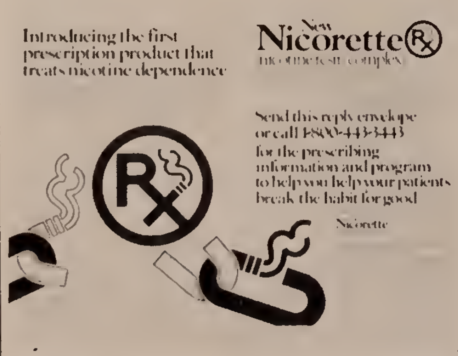
Merrell Dow


Psychological dependence
Nicotine dependence
Social factors

Nicotine named a principal etiologic component in smoking habit

Current medical opinion:
Nicotine dependence is a major cause of failure for people who try to quit smoking

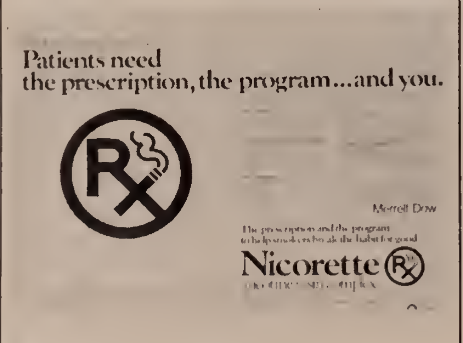
Merrell Dow


Introducing the first prescription product that treats nicotine dependence

New
Nicorette℞
nicotine resin complex

Send this reply envelope or call 1-800-443-3443 for the prescribing information and program to help you help your patients break the habit for good

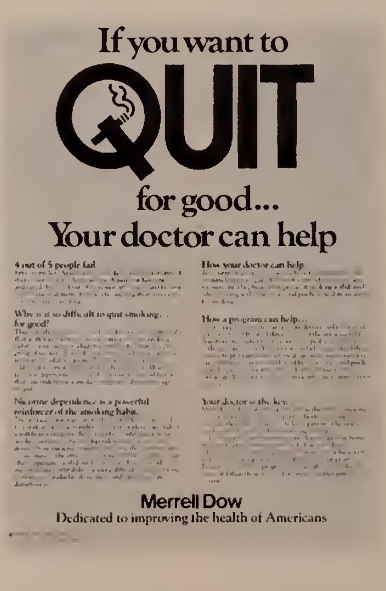
Nicorette


Patients need
the prescription, the program...and you.

Merrell Dow

The prescription and the program to help smokers break the habit for good

Nicorette℞
nicotine resin complex


If you want to
QUIT
for good...
Your doctor can help

4 out of 5 people fail

How your doctor can help

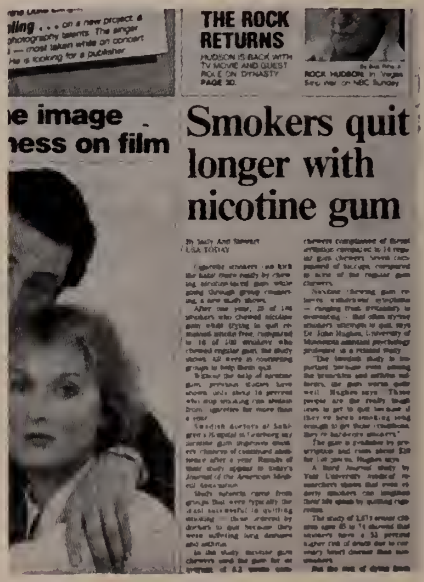
Why is it so difficult to quit smoking... for good?

How a program can help...

Nicotine dependence is a powerful reinforcer of the smoking habit.

Your doctor is the key.

Merrell Dow
Dedicated to improving the health of Americans


THE ROCK RETURNS
HUDSON IS BACK WITH TV MOVIE AND GUEST ROLE ON DYNASTY PAGE 2D.

ROCK HUDSON: In 'Vegas Strip War' on NBC Sunday

...e image
...ness on film

Smokers quit longer with nicotine gum

By Sally Ann Stewart
USA TODAY

An indoor air quality standard for ambient tobacco smoke based on carcinogenic risk

JAMES L. REPACE, MS, ALFRED H. LOWREY, PHD

ABSTRACT. An indoor air quality standard for ambient tobacco smoke in workplaces in the United States is derived based on limiting the involuntary carcinogenic risk to nonsmokers to the maximum level considered acceptable by federal regulatory agencies for environmental carcinogens in air, water, or food. This risk level corresponds to a 1-in-100,000 chance of contracting fatal lung cancer in a working lifetime of 40 years. To achieve acceptable risk, the daily average concentration of tobacco tar for a typical office must be at most 0.75 micrograms per cubic meter of air. At typical smoking occupancies for an office environment, achieving this standard would require impractical amounts of ventilation or prohibitive costs for air cleaning equipment. It appears that the only practical control measures are complete physical separation of smokers and nonsmokers on different ventilation systems, or prohibition of smoking in the workplace.

(NY State J Med 1985; 85:381-383)

Exposure of nonsmokers to indoor air pollution from tobacco smoke[1] (also known as involuntary or passive smoking) has become a public health problem[2] for several reasons: tobacco smoke is a human carcinogen,[2] exposure of nonsmokers to ambient tobacco smoke is widespread,[1,3] existence of a threshold for carcinogenesis is doubtful,[2,4,5] and there are a number of studies reporting a dose-response relationship between passive smoking and cancer.[6-11] Based in part on these studies, it is estimated that about 5,000 deaths from lung cancer occur among nonsmokers in the United States each year due to passive smoking.[12] In 1982, when there were only three such studies, the US Surgeon General asserted that "nonsmokers should avoid exposure to tobacco smoke to the extent possible."[2] This raises the question of how much exposure to tobacco smoke is too much, and whether excessive levels of ambient tobacco smoke can be controlled by ventilation or air cleaning.

In earlier work[1,13,14] factors affecting nonsmokers' exposure to tobacco smoke were studied, and field surveys of the levels of respirable particles indoors and outdoors, in both smoke-free and smoky environments, were conducted. This work established that ambient tobacco smoke imposed significant air pollution burdens on nonsmokers, and, using controlled experiments, a model was developed to estimate those exposures. This model predicts that the exposure of nonsmokers in the United States ranges from 0 to 14 milligrams of cigarette tar per day, depending on the nonsmoker's lifestyle, and that the average tar exposure for adults of working age is about 1.5 mg/day.[1,12,14] By comparison, smoking three very low tar cigarettes per day would result in an inhalation of about 1.5 mg of tar.[1] This finding is consistent with the results of a study which showed that passive smoking can produce urinary levels of cotinine (a nicotine metabolite) in nonsmokers that are comparable to those of active smokers of from one to three cigarettes per day.[15]

Due to higher smoker densities and similar air exchange rates, passive smoking in the workplace appears to dominate domestic passive smoking by a factor of about 4 to 1, and thus constitutes the major source of exposure for most adults.[12,14] This makes passive smoking in the workplace a prime candidate for public health control strategies.[16]

Currently, the primary control measure for ambient tobacco smoke is ventilation.[1,13,16] Therefore, this paper examines the impact of ventilation on nonsmokers' lung cancer risk as a basis for the formulation of an indoor air quality standard for ambient tobacco smoke.

DAILY PASSIVE TAR INHALATION

The quantity, Q, of tobacco tar (in units of micrograms per day) that would be inhaled out of the air by a nonsmoker in a typical work site is given by the expression $Q = ZB_rH$ (Z, equilibrium concentration of tobacco tar in units of $\mu g/m^3$; B_r, nonsmoking worker's respiration rate in units of m^3/hr; and H, length of the average workday in hours per day).[1,14] Earlier work[1,13,14] showed that the equilibrium concentration of tobacco smoke in an occupied space is given by the equation $Z = 217\ D_{hs}/C_v$ (D_{hs}, smoker density in units of habitual smokers per hundred cubic meters (where the number of habitual smokers is taken to be one third[1] of the design occupancy, O_c); 217, a constant proportional to the average emission rate for a typical cigarette smoker; and C_v, design ventilation rate in units of air changes per hour (ach)).[1,13,14] O_c and C_v may be determined for a typical office from tables of ventilation standards published by the American Society of Heating, Refrigerating, and Ventilating Engineers (ASHRAE).[17] If a certain risk level R_a (in units of annual probability of lung cancer mortality) is adopted as the acceptable level of risk as defined by federal regulatory practice,[18] the maximum daily inhaled exposure may be determined as Q_{max} (in units of $\mu g/day$) which corresponds to such a risk: $Q_{max} = R_a/E_R$ (E_R, exposure-response relationship relating lung cancer risk to passive exposure to tobacco smoke, in units of annual probability of lung-cancer mortality per microgram of tobacco tar in-

---

Mr. Repace is a physicist and policy analyst for the US Environmental Protection Agency and Dr Lowrey is a research chemist at the US Naval Research Laboratory, Washington, DC.

Address correspondence to Mr Repace, US Environmental Protection Agency, Washington, DC 20460.

Portions of this paper were presented at the Third International Conference on Indoor Air Quality and Climate, Stockholm, August 20-24, 1984.

The views presented in this article are solely those of the authors and do not necessarily represent the official policies of their respective agencies.

haled per day).[12,19]

The exposure-response relationship, E_R, has been estimated to be five lung cancer deaths per year per 100,000 persons at risk, per milligram daily exposure to tobacco tar,[12,19] where the population at risk is taken as nonsmokers aged $\geq$ 35 years. The method of estimation is outlined as follows: the daily probability-weighted average exposure of a typical adult in the United States has been calculated to be 1.43 mg/day, averaged over mean estimated workplace and domestic passive smoking exposures.[12,14] Exposure probabilities were estimated from a survey of the smoking policies of 1,000 American corporations (both white collar and blue collar; large, medium, and small), and from the percentage of 40,000 children raised in homes with one or more smokers.[12,14] Hirayama[7] has reported an annual rate of lung cancer mortality from passive smoking of 6.8 per 100,000 persons. A comparable age-standardized estimate of 7.4 deaths per 100,000 person-years for lung cancer mortality from passive smoking has been derived[12,19] from a study[20,21] of lung cancer in Seventh Day Adventists in the United States. The exposure-response relationship is derived from the ratio of the average risk to the average exposure: E_R = (7.4 lung-cancer deaths per hundred thousand person-years)/(1.43 mg of tobacco tar passively inhaled per day), yielding a value $E_R = 5 \times 10^{-5}$ lung cancer deaths per person-year mg tar/day.[12]

Level of "Acceptable" Exposure

Anderson[18] has reported an upper limit target risk of 10^{-5} (1 per 100,000) used by federal regulatory agencies for lifetime involuntary carcinogenic risk to the public. A 10^{-5} lifetime risk is a 1.3×10^{-7} annual risk, assuming a 78-year lifetime exposure. (The Food and Drug Administration has used a lifetime risk level of 10^{-6} as acceptable, and the Environmental Protection Agency, in the case of water quality criteria, has used lifetime risks of 10^{-5} to 10^{-7} as acceptable).[18] Scaling the acceptable annual risk level to adjust for a 40-year workplace exposure instead of a 78-year lifetime exposure yields $R_a = 78/40 \times 1.3 \times 10^{-7} = 2.5 \times 10^{-7}$ lung cancer deaths per person-year.

Using the given values of acceptable risk and exposure-response, $Q_{max} = R_a/E_R = 2.5 \times 10^{-7}/(5 \times 10^{-5}) = 5\ \mu g$/day. An expression for the maximum workplace tobacco tar concentration, Z_{max}, allowable to keep the inhaled exposure from exceeding Q_{max} can then be derived: $Z_{max} = Q_{max}/B_rH$ (Z_{max} represents the maximum acceptable concentration of ambient tobacco smoke (in units of $\mu g/m^3$) averaged over an H hour workday that would be permissible in the workplace to keep the annual lung cancer risk within "acceptable" levels for involuntary risk). Assuming an average respiration rate of $B_r = 1\ m^3/hr$ (alternate between sitting and light work) from tables of respiration rates for different degrees of human activity,[22] and an H = 6.7 hour daily average workday,[12,14] Z_{max} = 5 micrograms per day/6.7 m^3 per day = 0.75 $\mu g/m^3$. In other words, to produce an acceptable cancer risk from passive smoking in the workplace, the daily concentration of ambient tobacco tar during a nonsmoker's working lifetime would have to average three quarters of a microgram per cubic meter. By contrast, ambient tobacco tar concentrations in commercial buildings have been found to be 100 $\mu g/m^3$ to 1,000 $\mu g/m^3$.[1,13] In Switzerland, Weber and Fischer[23] reported a value of 202 $\mu g/m^3$ for 20 offices with natural ventilation, and an average of 120 $\mu g/m^3$ for 24 other offices, including 10 with both natural and mechanical ventilation, and 14 with mechanical ventilation only; 8 rooms had some smoking restrictions.

Effect of Ventilation and Air Cleaning on Risk Reduction

How much ventilation would be required in a typical American workplace to achieve an acceptable passive smoking risk? According to ASHRAE Standard 62-1981,[17] a ventilation rate (outdoor air) of 10 L/sec per occupant is recommended for office space at a design occupancy of seven persons per 100 m^2 of floor area (20 ft^3/min per occupant at seven persons per 1,000 ft^2). Assuming a ceiling height of 3 m and that one third of the workers smoke,[1] the smoker density D_{hs} = (seven persons) × (one smoker/three persons)/3.00 hundred cubic meters = 0.78 habitual smokers per 100 m^3 (a habitual smoker is assumed to smoke 2 cigarettes per hour for 16 hours per day).[1] The design ventilation rate is calculated as (70 L/s × 3,600 sec/hr)/(1,000 L/m^3/300 m^3) = 0.84 ach.[16]

Thus, in a typical office, the expected average concentration of tobacco smoke would be $Z_{av} = 217 \times .78/.84 = 202\ \mu g/m^3$. Reducing this concentration to the acceptable level of 0.75 $\mu g/m^3$ would require about 226 ach, or 2,700 L/sec per person (nearly 5,400 ft^3/min per occupant), a 270-fold increase in effective ventilation rate, which is impractical.[16]

Can air cleaning be a cost-effective substitute for ventilation? A heavy-duty room air cleaner with a fan capacity of 1,000 ft^3/min and a 99.97% efficiency HEPA filter cost about \$1,800 in 1982.[24] Thus, one can achieve an effective air exchange rate of (1,000 ft^3/min × 60 min/hr)/(9,800 ft^3) = 6.12 ach for a cost of \$1,800, or a total cost of \$66,471 to obtain 226 ach by air cleaning, or about \$28,000 per smoker, exclusive of fan-operating costs, an uneconomical proposition.

Physical separation of nonsmokers and smokers into sections within a given space reduces peak concentrations,[13] but not average concentrations to which nonsmokers are exposed, and therefore does not affect average lifetime risk.

Conclusions

A health-based standard of 0.75 $\mu g/m^3$ (daily average) for respirable particulate matter from ambient tobacco smoke in the workplace appears to be necessary to reduce carcinogenic risk to nonsmokers to acceptable levels. However, to attain such a standard, complete physical separation of smokers and nonsmokers on separate ventilation systems, and prohibition of smoking in the workplace appear to be the only feasible alternatives for reducing the lung cancer risk from passive smoking to below the maximum risk considered acceptable by the standards of federal regulatory agencies for carcinogens in air, water, or food.

References

1. Repace JL, Lowrey AH: Indoor air pollution, tobacco smoke, and public

health. *Science* 1980; 208:464-472.

2. *The Health Consequences of Smoking: Smoking and Cancer.* A report of the Surgeon General, US Dept of Health & Human Services, Washington, DC, DHHS Pub No 82-50179, Government Printing Office, 1982.

3. Friedman GD, Petitti DB, Bawol RD: Prevalence and correlates of passive smoking. *Am J Public Health* 1983; 73:401-405.

4. Scientific bases for identification of potential carcinogens and estimation of risks. Report of the Interagency Regulatory Liaison Group, Work Group on Risk Assessment. *J Natl Cancer Inst* 1979; 63:241-268.

5. International Agency For Research on Cancer (IARC), Monographs V 1-20, suppl I, "Chemicals and Industrial Processes Associated With Cancer in Humans," Lyon, IARC, 1979.

6. Trichopoulos D, Kalandidi A, Sparros L, MacMahon B: Lung cancer and passive smoking. *Int J Cancer* 1981; 27:1-4.

7. Hirayama T: Nonsmoking wives of heavy smokers have a higher risk of lung cancer: a study from Japan. *Br Med J* 1981; 282:183-185.

8. Hirayama T: Passive smoking and lung cancer. *Br Med J* 1981; 282:1393-1394.

9. Correa P, Pickle LW, Fonham E, Lin Y, Haenszel W: Passive smoking and lung cancer. *Lancet* 1983; 595-597.

10. Sandler DP, Wilcox AJ, Everson RB: Cumulative effects of lifetime passive smoking on cancer risk. *Lancet* 9 February 1985, p 312.

11. Hirayama T: *Passive Smoking and Lung Cancer.* Proceedings of the 5th World Conference on Smoking and Health, Winnipeg, July 10-15, 1983.

12. Repace JL, Lowrey AH: A quantitative estimate of nonsmokers' lung cancer risk from passive smoking. *Environment International,* 1985; 11:3-22.

13. Repace JL, Lowrey AH: Tobacco smoke, ventilation, and indoor air quality. *ASHRAE Transactions* 1982; 88:894-914.

14. Repace JL, Lowrey AH: *Modeling Exposure of Nonsmokers to Ambient Tobacco Smoke.* Proceedings of the 76th Annual Meeting of the Air Pollution Control Association, Atlanta, June 19-24, 1983.

15. Matsukura S: Effects of environmental tobacco smoke on urinary cotinine excretion in nonsmokers—evidence for passive smoking. *N Engl J Med* 1984; 311:828-832.

16. Repace JL: *Effect of Ventilation on Passive Smoking Risk in a Model Workplace.* Proceedings of An Engineering Foundation Conference on Management of Atmospheres in Tightly Enclosed Spaces, Santa Barbara, October 17-21, 1983. American Society of Heating, Refrigerating, and Air Conditioning Engineers, Atlanta, GA 30329.

17. American Society of Heating, Refrigerating, and Air Conditioning Engineers, Inc: *ASHRAE Standard 62-1981, Ventilation for Acceptable Indoor Air Quality.* ASHRAE, 1791 Tullie Circle, NE, Atlanta, GA 30329.

18. Anderson EL and the Carcinogen Assessment Group of the US Environmental Protection Agency: Quantitative approaches in use to assess cancer risk. *J Risk Analysis* 1983; 3:277-295.

19. Repace JL: Consistency of Research Data on Passive Smoking and Lung Cancer, *Lancet* 1984; 2:506.

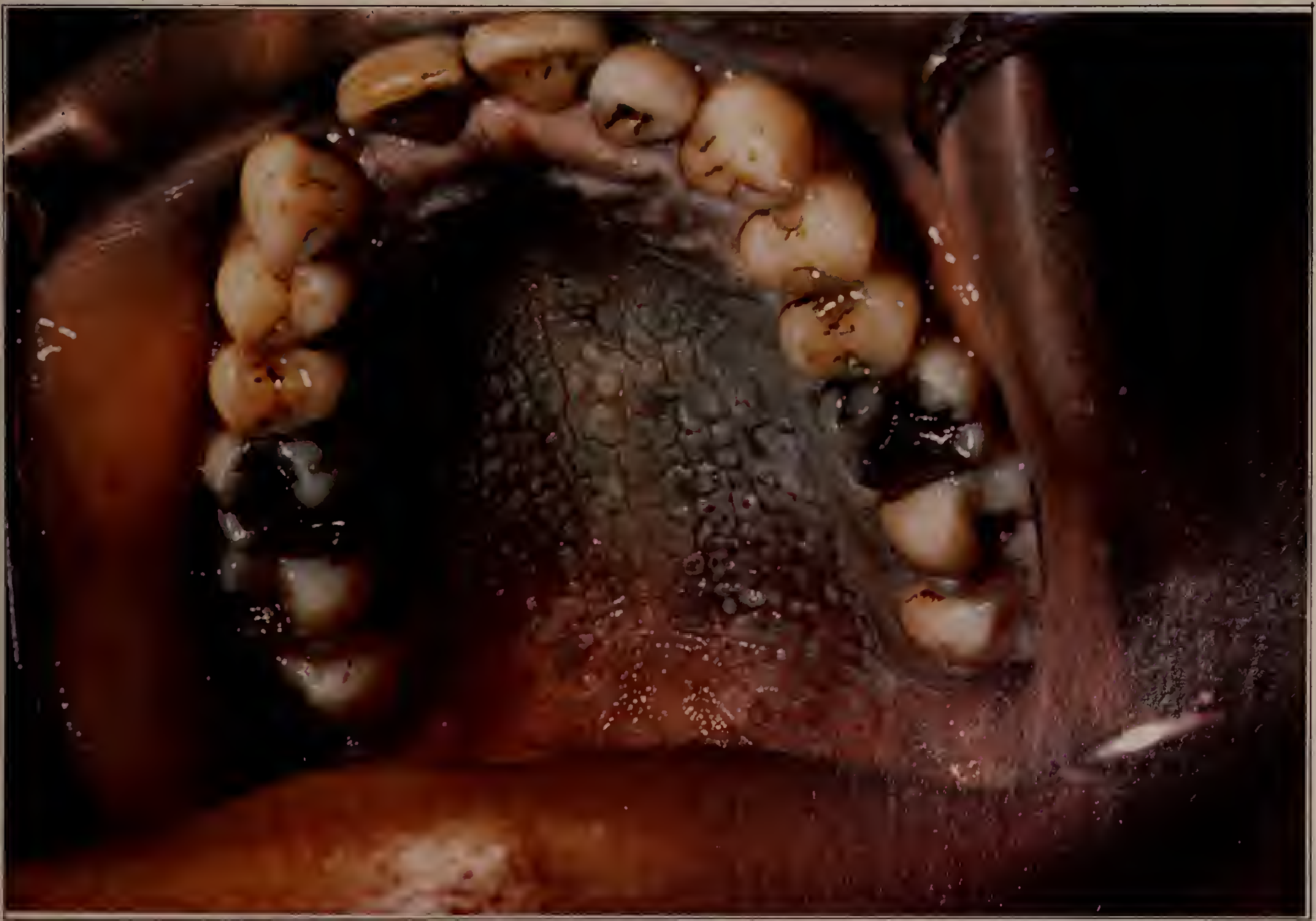
20. Phillips RL, Kuzma JW, Beeson WL, Lotz T, Brin B: Mortality among California Seventh-Day Adventists for selected cancer sites. *J Nat Cancer Inst* 1980; 65: 1097-1107.

21. Phillips RL, Kuzma JW, Beeson WL, Lotz T: Influence of selection vs lifestyle on risk of fatal cancer and cardiovascular disease among 7th-Day Adventists. *Am J Epidemiology* 1980; 112:296-314.

22. Altman PL, Ditmer DS: *Respiration and Circulation.* Bethesda, Federation of American Society for Experimental Biology, 1971.

23. Weber A, Fischer T: Passive smoking at work. *Int Arch Occup Environ Health* 1980; 47:209-221.

24. *Indoor Air Quality Handbook for Designers, Builders, and Users of Energy-Efficient Residences.* Sandia Report SAND-82-1773, 1982. Sandia, Albuquerque National Laboratories.



Nicotinic stomatitis. Inflammation of the palate in a man over 50 who took up cigarette smoking as a teenager.
Photograph submitted by William H. Wood, DDS, 1271 Seventh St, Slidell, LA 70458.

A pilot's guide to cabin air quality and fire safety

H. B. FULTON, JR

Airline travelers need more protection from exposure to tobacco smoke. An important step in this direction would have been the proposed ruling to prohibit smoking on flights of less than two hours—a measure that was passed by the Civil Aeronautics Board in May 1984, only to be rescinded a few hours later, allegedly following political pressure.

IS VENTILATION ADEQUATE?

At hearings of the Senate Subcommittee on Aviation in 1982 and 1983 on the subject of cabin air quality the Federal Aviation Administration has testified that aircraft ventilation rates compare favorably with recommended standards for non-aviation environments. This position was echoed by the Air Transport Association of America (ATA), the trade organization of airlines in the United States, which based its testimony on a survey of ventilation data reported by airlines. However, much of the ATA data appear to have been based on system design values compiled by aircraft manufacturers. Once the airplane is put into service, these values begin to erode. Actual ventilation capacity is dependent on many factors, such as engine compressor section efficiency, bleed valve operation, air duct cleanliness, security of duct connectors, pressure regulator valve function, air conditioning pack operation, door and window seal integrity, filter porosities, aircraft altitude, and engine revolutions per minute.

During the summer, inadequate airflow through the cabin is a common complaint on the Boeing 727 and the Douglas DC-9. In one case, upon investigation by maintenance workers, a large clamp that secured a rubberized flexible duct connector was found to be loose, and air was leaking out of the connection. Water separator bags on the DC-9 remove condensation from conditioned air and can become clogged and impede air flow. The 727 has a muffler-like device in its duct system to enable quiet and smooth airflow. This muffler uses a fiberglass packing which can work loose and clog a downstream filter. This, too, will impede airflow. The efficiency of air cycle machines (packs) which heat, cool, and dehumidify the air entering the cabin erodes with time and usage. These deficiencies are common to all operators of these jets. The ventilation systems simply do not perform at design specifications after prolonged usage.

In addition to the DC-9 and the 727, the Boeing 757 flies a major share of Eastern Airlines' flights that are less than two hours. This is the newest and most sophisticated jet in the air today, and consequently its ventilation system will operate closer to design specifications than those in older airplanes. However, this is a "catch-22" because the 757's air conditioning system is designed to recirculate a substantial percentage of cabin air. This recirculated air is passed through two filters on its way back to the cabin. Both of these filters are of the fiberglass type which effectively trap only particulate matter and accomplish little or no filtration of odors or gases.

The most obvious evidence that smoking introduces pollutants at a higher rate than the ventilation systems can effectively dissipate is the haze in the back of the airplane. A similar observation is that within seconds after turning off the "No Smoking" sign the pilot of a DC-9 can detect tobacco smoke in the cockpit, which is 20 feet from the passenger section. Flight attendants complain of having to work in a cloud of smoke which hangs at head level. If ventilation capacities were as the engineers say they are, this smoke cloud should never occur, because conditioned air enters the cabin from overhead and is exhausted through floor-level vents. The fact is that this top-to-bottom airflow pattern is not forceful enough to dispell the volume of smoke produced by numerous cigarettes.

The photograph of the outflow valve of a DC-9 (Fig 1) is a graphic illustration of "what you see is what you breathe." The outflow valve is the major exhaust port through which cabin air is vented overboard and is the device that controls cabin pressurization. Its movement between open and closed positions is accomplished by mechanical linkages which become gummed up when tobacco smoke condenses. This stickiness makes smooth operation of the pressurization difficult, if not impossible, much to the discomfort of the passengers' and crews' ears. It can even cause a mechanical failure if the valve becomes stuck in one position.

The figure shows an outflow valve with average contamination. Such a picture is common to all airliners on which smoking is permitted. The B-757 had a higher percentage of dirty valves, which is noteworthy, because the recirculation systems on this airplane and the B-767 are harbingers of aircraft design. The purpose of a recirculation ventilation system is to increase engine fuel economy, which is achieved at the expense of cabin air quality.

CIGARETTE FIRES ALOFT

A fire in the cabin is a dire emergency. Although the

Mr Fulton is a pilot for Eastern Airlines.
Address correspondence to Mr Fulton, 4116 Kuykendall Rd, Kennesaw, GA 30144.

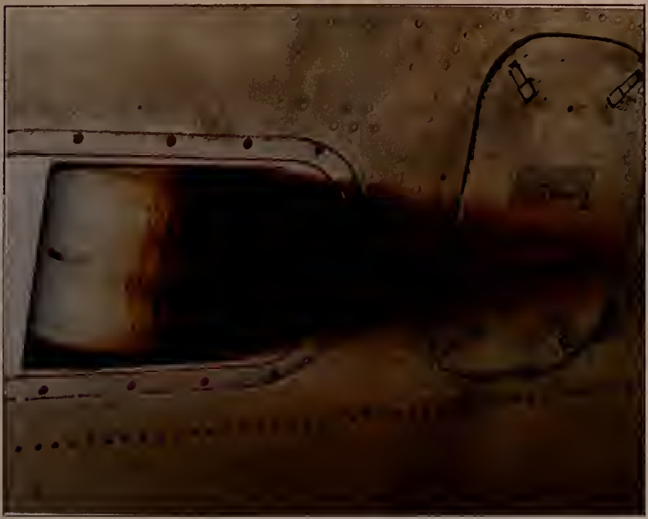


FIGURE 1. The stain adhering to the airplane skin behind the outflow valve of a DC-9 is tobacco smoke condensate, "tar," which can cause a malfunction or even failure of the pressurization system.

airline industry goes to great lengths to make flying the safest form of transportation that exists today, the lighting of cigarettes in airliners is courting disaster. The originally suspected cause of the 1983 Air Canada DC-9 fire that killed 22 persons was the lavatory flush motor; but this has been eliminated as a fire ignition source after tests by the National Transportation Safety Board. Although the contents of the waste bin were not burned due to activation of the fire extinguisher, which is positioned within the waste bin compartment, this does not rule out the possibility of an improperly discarded cigarette as an ignition source. The chute that guides discarded paper towels, tissues, and similar refuse into the trash bin on the DC-9 has narrow dimensions (approximately 2½ in × 7 in). Passengers push paper towels through the spring loaded door opening and release them, expecting them to fall into the waste receptacle. More often than not, however, the towel lodges in the chute, which stops others from falling through; soon the chute is packed with paper towels. This creates two waste bins, one in the container protected by a fire extinguisher and the other in the chute with no fire protection. Any paper wedged in the chute could have been ignited if a lit cigarette were dropped on top of it.

Another distinct possibility is the tendency of passengers who are not familiar with airplane lavatories to put waste paper into improper places. Since the trash receptacle chute is covered by a spring loaded door and one must read the message on it to know what it is, much waste paper finds its way into any opening, such as an empty Kleenex container, the slot for toilet seat covers, or the paper towel holder. An incompletely extinguished cigarette could just as readily be placed into these areas. The oxidizers added to American tobacco make cigarettes even more hazardous, because the cigarettes will smolder until they are completely burned out. One has only to ask any experienced flight attendant about passengers who have dropped or lost a burning cigarette to learn about such harrowing incidents.

In pilots' flight manuals there is a remedial procedure for almost any failure or emergency that can be imagined, except for an in-flight cabin fire. This is not an inadequacy in training or in the manufacturer's aircraft manual. The truth is that little can be done except fight the fire with a hand-held extinguisher, for which the crew is trained, and land the airplane as soon as possible, hoping the passengers have not suffocated or the crew become incapacitated before they can get safely on the ground. Pilots cannot release the passengers' oxygen masks because they operate under positive flow, and the pure oxygen would turn the airplane into a giant torch.

Prohibiting smoking on short flights would virtually eliminate the possibility of accidental fires, but would raise the specter of surreptitious smoking in the lavatories. Indeed, a lavatory fire allegedly occurred on a DC-9 inbound to Tampa International Airport in 1983. The fire was of sufficient intensity to make the door too hot to touch, and, afterward, the plastic fixtures were found to be melted. It is not known if an attempt was made to fight the fire or if the door was kept closed to contain the smoke, but the flight was concluded safely and the fire was put out on the ground. A discarded cigarette was the suspected cause. These continuing incidents are ample reason to require smoke detectors in the lavatories. The battery powered home-type detector should work well, but the cover should be tamperproof so the battery cannot be disconnected. Also, the lavatory signs should be more prominently displayed and should include a warning about the danger of fire and the presence of a smoke detector.

There is little question that the vast majority of the public desires a ban on smoking aboard aircraft. Sensing this, in 1983 the Tobacco Institute, the public relations unit of the tobacco industry, paid workers to collect signatures of air travelers opposed to such restrictions. Individuals reported they were first asked if they smoked. If the answer was "No," the interview was concluded with a "Thank you." If "Yes," they were asked if they would sign a petition to the Civil Aeronautics Board that was worded in such a way as to imply an imminent threat to the freedom of airline passengers (Fig 2). In order to provide an alternative viewpoint, a group of volunteers from the American Lung Association of Georgia asked 5,502 people in the Atlanta Hartsfield International Airport the following question: The Civil Aeronautics Board has proposed that all smoking be banned on airline flights of one hour or less. Would you be in favor of, neutral to, or opposed to such a regulation? The results were 62% in favor, 24% neutral,

TO THE U.S. CIVIL AERONAUTICS BOARD:

As airline passengers, we OPPOSE any government regulation

- To ban smoking *entirely* on flights of one or two hours or less
- To ban smoking *entirely* on planes with fewer than 30 or 60 seats
- Under which smoking might be banned *entirely* in a compartment if a single passenger complains of "physical illness" from smoke

Smoker/nonsmoker seating works well! Please keep it that way!

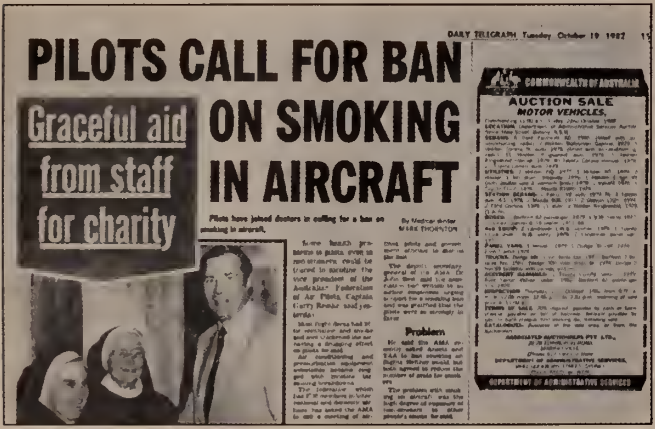
FIGURE 2. Top of petition circulated in airports by employees of the Tobacco Institute.

and 14% opposed. The survey was reinforced by subsequent surveys by Midway Airlines, Wein Air Alaska, and *Newsweek*. In view of the frequency of unsolicited comments from individuals critical of the proposal for focusing only on short flights and not on a total ban, it is unlikely that the results of the survey would differ were there a proposal for a ban on smoking on flights of two hours.

From its founding in 1981 to its demise in 1985, Texas-based Muse Air imposed a total smoking ban on its flights. Other carriers such as Rocky Mountain Airways, Hawaiian Air, Aloha Airlines, Mid Pacific Air, and Air Maritime in Canada have also banned smoking. Chicago-based Midway Airlines has offered total nonsmoking flights on an experimental basis, and numerous commuter airlines have been operating nonsmoking flights. Not only is a ban on smoking necessary for improved comfort, health, and safety, but it also appears to be desired by the vast majority of air travelers.

THE HEALTH OF PILOTS

Although the passenger cabin has been the focus of concern of hearings on smoking aloft, many airline pilots feel that tobacco smoke in the cockpit is a greater threat to their health and the safety of the flight (Fig 3). In 1976 the Public Citizen's Health Research Group, the Aviation Consumer Action Project, and 76 pilots of seven major airlines attempted to have smoking banned in the cockpit but were rebuffed by the Federal Aviation Administration (FAA). The position of the FAA, according to J. Robert Dille, MD, chief of the Civil Aeromedical Institute, is that "our two studies and those of Heimstra suggest that abrupt smoking withdrawal poses a greater threat to aviation safety than crew smoking" (personal communication). What this position fails to address is that there are far more nonsmoking pilots than pilots who smoke, and that a decided majority of the nonsmoking pilots believes that the smoke of others in the close confines of the cockpit is a detriment to one's health and one's ability to be a safe, alert, and competent pilot.



DAILY TELEGRAPH Tuesday October 19 1982

PILOTS CALL FOR BAN ON SMOKING IN AIRCRAFT

Graceful aid from staff for charity

Pilots have joined doctors in calling for a ban on smoking in aircraft.

By Medical Writer MARK THORNTON

Problem

COMMONWEALTH OF AUSTRALIA

AUCTION SALE

MOTOR VEHICLES,

DEPARTMENT OF ADMINISTRATIVE SERVICES

FIGURE 3. Pointing to the possibility that some health problems in nonsmoking pilots are related to exposure to tobacco smoking, the 2,700-member Australian Federation of Air Pilots joined the Australian Medical Association in urging airlines to ban smoking aloft (*Daily Telegraph* (Sydney), October 19, 1982). Airlines have responded by cutting the number of seats at which smoking is permitted.

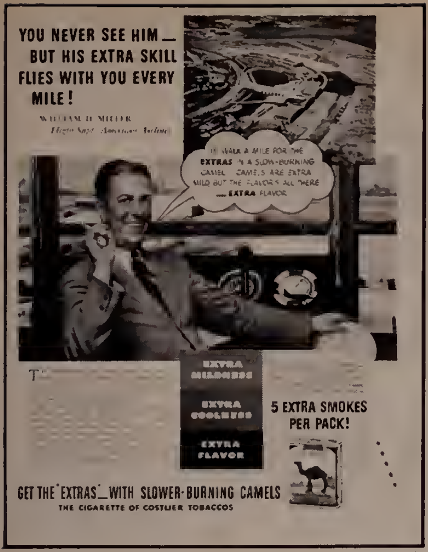
The primary study referred to by Dille was a Multiple Task Performance Battery administered to 17 non-aviation oriented individuals who were smokers. The sessions consisted of two days of testing. The first day the subjects smoked immediately before the first 30-minute test period and during the 10-minute interludes between the five subsequent 30-minute test periods. The scores obtained were used as baseline scores. The second test day the subjects were allowed to smoke immediately before the first test period, but were denied smoking for the remainder of the three-hour testing period. The results indicated that "when smoking was prohibited, performance was decreased, largely as a result of decrements in tracking [ability]. Decrements in performance were associated with decreased attentiveness, both prior to and after the experiment."

This sample of 17 subjects is too small to use as a basis for a ruling that affects thousands of pilots. To address both sides of this issue the Multiple Task Performance Battery should be administered to a group of nonsmokers. The first day the subjects would breathe ambient air to establish baseline scores. The second day another person would join them in the test compartment who would smoke one or two cigarettes during each 30-minute test period. A decline in scores on the second day might provide revealing information on the ability of nonsmokers to function in a smoky environment. If this were to occur, and in light of the overwhelming number of nonsmoking pilots, then a cockpit smoking policy should be decided in favor of the majority.

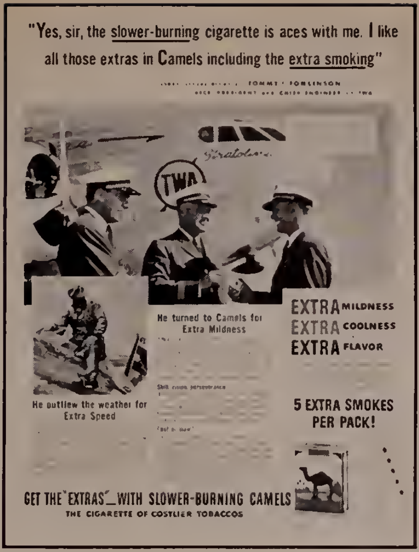
Preliminary results of a mail survey of 1,287 Eastern Airlines pilots conducted by the Air Line Pilot Association support such a policy. Four hundred and eighteen pilots replied, of whom 343 identified themselves as nonsmokers. An overwhelming number of respondents felt that smoking in the cockpit is detrimental to health (85% of nonsmokers and 33% of smokers agreed), that the cockpit ventilation system inadequately removes smoke, that smoking in the cockpit is detrimental to crew coordination, and that smoking should be restricted or prohibited in the cockpit.

Federal Aviation Regulation 91.11 states "No person may act as a crewmember of a civil aircraft while using any drug that affects his faculties in any way contrary to safety." Though aviation literature contains some mention of the many detrimental effects that smoking can have on a pilot, there is a total lack of information on the effects of smoke on the nonsmoking pilots who share the same air supply and the same small space. In the recent survey of the Air Line Pilot Association, 85% of the nonsmoking pilots reported some type of adverse physical reaction—principally, eye irritation (74%) and headache (40%)—whereas only 14% of the smoking pilots reported any of these reactions. In the category of non-physical reactions, 76% of the nonsmoking pilots reported some type of adverse reaction (annoyance, 55%; resentment, 31%), whereas only 18% of the smokers did so.

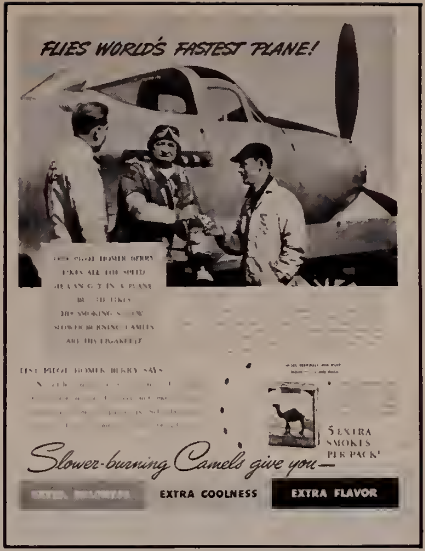
The Soviet airline Aeroflot has a policy that prohibits smoking in the cockpit for five hours, and two hours before landing. Since most flights in the United States are less than two hours a policy should exist that during any such flight smoking should be prohibited unless expressly


YOU NEVER SEE HIM — BUT HIS EXTRA SKILL FLIES WITH YOU EVERY MILE!
EXTRA MILDNESS
EXTRA COOLNESS
EXTRA FLAVOR
5 EXTRA SMOKES PER PACK!
GET THE "EXTRAS" — WITH SLOWER-BURNING CAMELS
THE CIGARETTE OF COSTLIER TOBACCOS

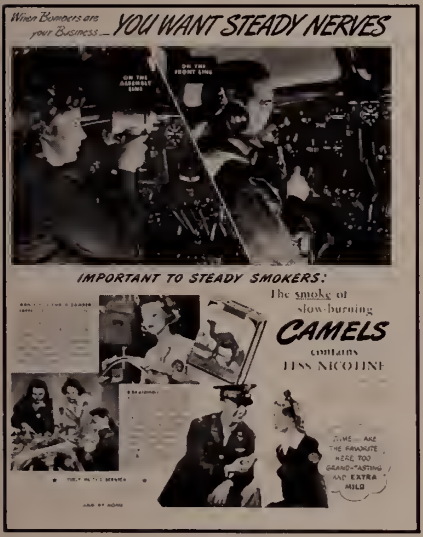
1940


"Yes, sir, the slower-burning cigarette is aces with me. I like all those extras in Camels including the extra smoking"
TOMMY TOMLINSON
TWA
Stratoliner
He turned to Camels for Extra Mildness
He outflew the weather for Extra Speed
EXTRA MILDNESS
EXTRA COOLNESS
EXTRA FLAVOR
5 EXTRA SMOKES PER PACK!
GET THE "EXTRAS" — WITH SLOWER-BURNING CAMELS
THE CIGARETTE OF COSTLIER TOBACCOS

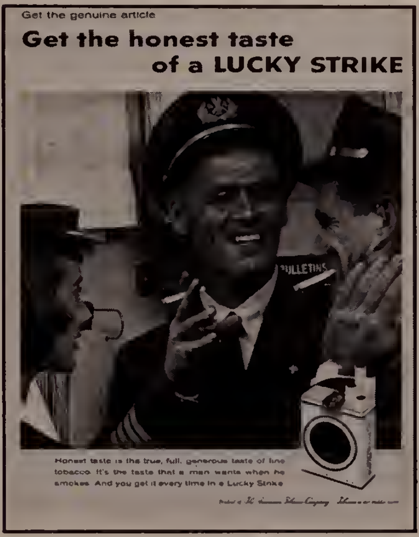
1940


FLIES WORLD'S FASTEST PLANE!
5 EXTRA SMOKES PER PACK!
Slower-burning Camels give you —
EXTRA MILDNESS
EXTRA COOLNESS
EXTRA FLAVOR

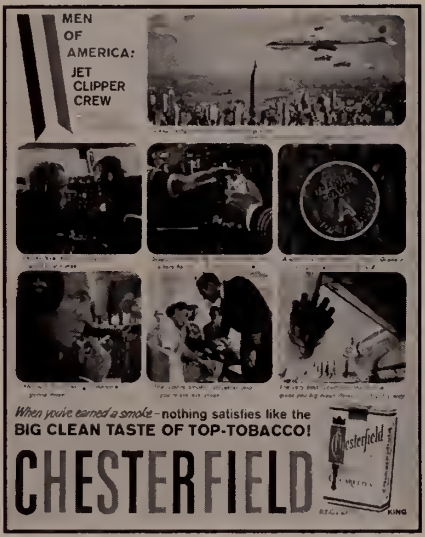
1940


When Bombers are your Business — YOU WANT STEADY NERVES
IMPORTANT TO STEADY SMOKERS:
The smoke of slow-burning CAMELS contains LESS NICOTINE

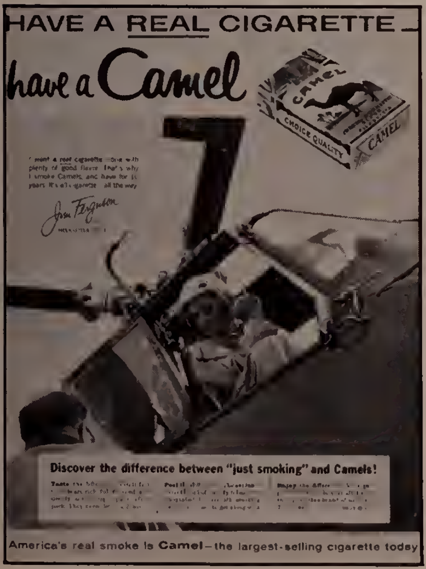
1942


Get the genuine article
Get the honest taste of a LUCKY STRIKE
BULLETINS
Honest taste is the true, full, generous taste of fine tobacco. It's the taste that a man wants when he smokes. And you get it every time in a Lucky Strike

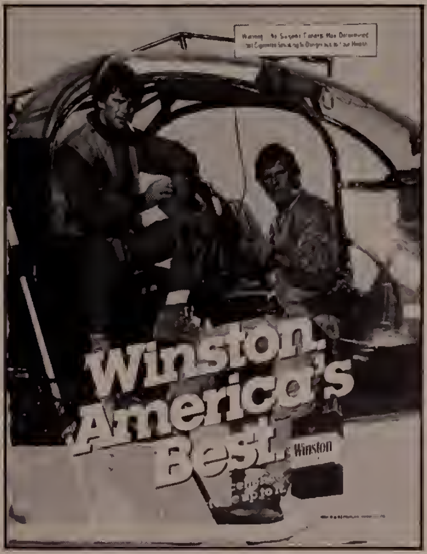
1958


MEN OF AMERICA:
JET CLIPPER CREW
When you've earned a smoke — nothing satisfies like the BIG CLEAN TASTE OF TOP-TOBACCO!
CHESTERFIELD
KING

1958


HAVE A REAL CIGARETTE —
have a Camel
CAMEL
CHOICE QUALITY
Discover the difference between "just smoking" and Camels!
America's real smoke is Camel — the largest-selling cigarette today

1957


Winston America's Best.
Winston

1985

From stunt pilots in the 1920s to astronauts in the 1960s, the airman as hero has been a frequent theme of cigarette advertising aimed at millions of readers of *LIFE* and other magazines. At New York's LaGuardia Field (top left), slower-burning, cooler Camels were depicted as a vital asset for a radio control-room flight superintendent. Just before flying the maiden transcontinental flights of TWA Stratoliner aircraft (top center), the pilots and a national record-holder for flying speed and endurance smoked Camels. Test pilots (top right) and bombardiers (middle left) smoked Camels for steady nerves. Other pilots chose Lucky Strikes (middle center) and America's first commercial jet pilots (middle right) on Pan American smoked Chesterfield in the cockpit. According to RJ Reynolds, helicopter pilots in 1957 (bottom left) and 1985 (bottom center right) demonstrate their manliness by lighting up cigarettes near fuel supplies. [DOC ARCHIVE] waived by each pilot of the crew.

The rigid code of command authority emanating downward from the captain in an airline cockpit is not well known outside the industry. A first or second officer is hesitant to speak out against a smoking captain in defense of his or her preference for a smoke-free cockpit, for fear of reprisal.

A new cockpit smoking policy would do much to alleviate disputes in airline cockpits and to improve the health of pilots and the safety of flights.

Who to complain to about smoking aloft?

With the closing of the Civil Aeronautics Board on December 31, 1984 (mandated in airline deregulating legislation enacted in 1978), all matters related to consumer protection in air travel (including smoking) have been shifted to the Department of Transportation (DOT). DOT handles questions and complaints by telephone (202-673-6047) or by mail (Consumer Affairs Division, Room 10405, Office of Community and Consumer Affairs, Office of Governmental Affairs, US Department of Transportation, 400 7th Street SW, Washington, DC 20590).

The Airline Transport Association [spokespersons have] talked about their legal responsibilities as common carriers. The legal responsibility of a common carrier, according to common law going back to the days of stage coaches, is that it must provide the highest possible standards of safety and health for passengers. There is nothing in the common law that requires it to permit anybody to smoke or to drink or to burn incense or to chew or to spit. Yet they talk in terms of "preferences." Somehow they have to accommodate preferences. Yet the Civil Aeronautics Board held from the beginning, and the US Court of Appeals recently reaffirmed— and many airlines have demonstrated— that there is no legal right to smoke aboard an airplane.

For these reasons ASH (Action on Smoking and Health) strongly supports the banning of smoking on short flights. There is no longer any doubt that it would solve the problem. After 10 years of complaints the airlines have done virtually nothing. They have not experimented with other proposals. They have not come up with different ventilation or physical barriers, or a barrier of people who don't care between the smoking and non-smoking sections. They do absolutely nothing and then when proposals are made, they come in and say no, no, no, we oppose it.

It works. Many carriers have banned smoking, and smokers are not climbing the walls or setting fires to the restrooms. Indeed, it is hard to see why anyone should really have to smoke on a short flight. On short flights many smokers fly in the no-smoking section anyway.

And I wonder why it is in the Air Transport Association that they feel that cigarette smokers are somehow entitled to more protection than pipe and cigar smokers. Pipe and cigar smoking is banned by most carriers. Those who derive the same satisfaction from chewing and spitting or who desire to burn incense or do a variety of other things can't do so aboard a plane.

Other privileges such as the use of computers on flights are now being restricted by some of the airlines where it appears to be necessary to protect passengers. Is smoking that much more important?

What arguments do they present to the contrary? Well, it is the usual lawyers' parade of horrors. Let's sit down and find every possible thing that might go wrong. Any regulator knows that these frequently and usually do not occur. They say that it is going to be confusing. Passengers won't know when they can smoke and when they cannot. Could anything be more confusing than their ticket structure? Could anything be more confusing than when you are going to get a movie or not or when you are going to get a snack versus a meal or when you can hang up your large bag or many other amenities? They say that drawing the line is arbitrary. Of course, any line is somewhat arbitrary. We have to draw lines on how much people can drink, or baggage size, or a variety of other limitations . . . .

The biggest argument, of course, is that it somehow disadvantages people in certain cities, not recognizing that living in certain cities creates lots of problems with regard to air transportation. Smoking is certainly just a small one.

We strongly favor the idea of banning smoking, not just on flights of one hour, but of two. And there are several reasons. First, two hours is consistent with many other situations in everyday life: movies, courtrooms, college seminars, many meetings, and so on. We know of many non-smoking jurors who get excused from jury duty because they can't stand the smoke in the jury room. I have never seen anybody go before a judge and say, "Your Honor, I can't participate in this trial. I've got to smoke every hour on the hour." Smokers can and do reasonably refrain from smoking up to two hours.

Based on the testimony of
John F. Banzhaf, III
Action on Smoking and Health (ASH)
Hearings of Civil Aeronautics Board
February 1983

Endobronchial foreign body (cigarette filter tip) with inflammatory pseudotumor of the lung

A "True" xanthogranuloma

ANIL K. BATRA, MD, LLOYD REICH, MD, MARTIN BERNSTEIN, MD

Benign tumors of the bronchus are rare. The majority of these are adenomas, with small numbers of papillomas, fibromas, lipomas, fibroxanthomas, plasma-cell granulomas, amyloids, and hamartomas. The patient presented in this report had an obstructing lesion of the bronchus subsequently found to be a non-neoplastic tumor formed by a partially organized cigarette filter tip in the intermediate and right lower lobe bronchus with distal xanthogranulomas.

CASE REPORT

A 59-year-old man was admitted for evaluation of a persistent cough, productive of white sputum, of six months' duration, and an infiltrate in the lower lobe of the right lung. A year prior to admission he had had a severe coughing paroxysm lasting 10 to 15 minutes while smoking a filter-tipped cigarette, after which the patient no longer smoked. Three months before admission, a febrile illness developed that lasted 10 days. It was treated with oral penicillin for one month without any response. A chest roentgenogram taken at that time was within normal limits. The patient admitted to episodic wheezing unrelated to change of body posture during the four months preceding admission. There was no history of bronchial asthma.

Physical examination showed a temperature of 38.3 °C (101 °F); pulse, 84 beats/min; blood pressure, 150/80 mm Hg; and respiratory rate, 16/min. There was no lymphadenopathy, clubbing, or cyanosis. Examination of the chest revealed localized wheezing in the right midlung field posteriorly, with intermittent rhonchi. The remainder of the physical examination results, including those involving the pharynx and vocal cords, were within normal limits.

Laboratory examination results, including hemogram, urinalysis, and blood and liver chemistry study values, were within normal limits. Chest roentgenograms showed an infiltrate in the lower lobe of the right lung without any evidence of pleural effusion or hilar and mediastinal adenopathy.

The patient underwent fiberoptic bronchoscopy, which showed normal upper airways. The right upper lobe carina was markedly edematous, but the segmental orifices were patent. There was a lobulated endobronchial mass arising from the medial wall that almost occluded the bronchus intermedius. Bronchial brushing and multiple biopsy specimens of the mass and of the mucosa from the right upper lobe and main carina were obtained.

Bronchial brushings revealed polymorphonuclear leukocytes and isolated, irregular columnar cells. Endobronchial biopsy of the mass showed evidence of chronic inflammation with squamous metaplasia and dysplastic changes.

The patient's general condition improved during the next few days, and the temperature normalized. He underwent mediastinoscopy and right thoracotomy. The mediastinal lymph nodes were seen to be hyperplastic and benign on frozen section. A right middle and lower lobectomy was performed, and the patient had an uncomplicated recovery.

Pathologic findings. The right lower lobe appeared enlarged, weighing 427 g and measuring 16 × 18 cm (Fig 1). The lower-lobe bronchus contained a plastic tubular structure that appeared to be a cigarette filter tip filled with a bloody purulent exudate and surrounded by fibrinopurulent material and scar tissue. The lower-lobe lung tissue itself was uniformly firm and appeared consolidated and studded with tan nodules. The middle lobe and its bronchus were unremarkable.

The lower lobe showed a pattern of xanthogranulomatous inflammation consisting mainly of large histiocytes and plasma cells, with some small lymphocytes and occasional polymorphonuclear cells. The alveoli had thickened septae containing fibrinous tissue infiltrated with plasma cells and lymphocytes characteristic of chronic organizing pneumonia. The right middle lobe was normal (Figs 2, 3).

When the patient was told of these findings, he recalled choking on a cigarette filter

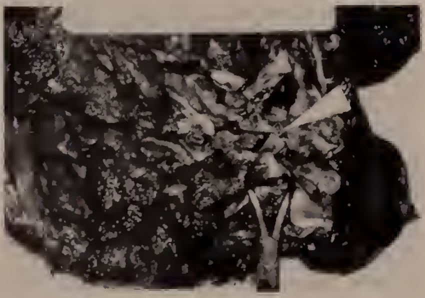


FIGURE 1. Right lower and middle lobectomy specimen with main and segmental bronchi opened. Arrow points to cigarette filter tip totally occluding main bronchus of the lower lobe. Lower lobe parenchyma is completely consolidated.

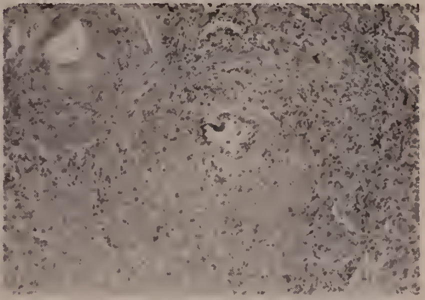


FIGURE 2. Right lower lobe parenchyma. Normal alveolar pattern is completely replaced by inflammatory infiltrate. A bronchus remains (center); blood vessels can also be distinguished (lower right). (hematoxylin and eosin, original magnification ×40)

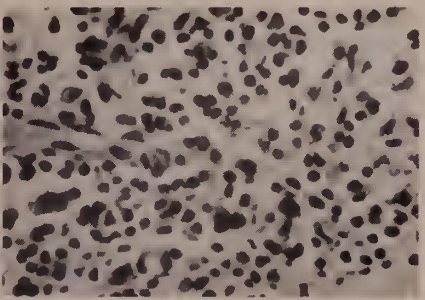


FIGURE 3. Right lower lobe parenchyma. Inflammatory infiltrate consists of round cells, mainly plasma cells and large foamy histiocytes with small lymphocytes. (hematoxylin and eosin, original magnification ×400)

From the Division of Pulmonary Disease, Department of Medicine, (Drs Batra and Bernstein), and Department of Pathology (Dr Reich), Maimonides Medical Center, Brooklyn, NY, and Downstate College of Medicine, Brooklyn, NY.

Address correspondence to Dr Batra, 5701 W Charleston Blvd, Suite 203, Las Vegas, NV 89102.

tip (True) more than a year ago. He had presumed that it was expectorated during a coughing paroxysm.

DISCUSSION

This case illustrates the pathologic features consistent with xanthogranuloma of the right lower lobe.[1] The possibility of a foreign body as the causative factor in the pathogenesis of pulmonary xanthogranuloma has been considered by others,[2–4] but the presence of a foreign body has not been documented. Other suspected causes of xanthogranuloma in the lung include viral infections, immunologic reaction such as Arthus reaction, disorders of cholesterol metabolism, and exogenous steroid administration.[1,2] Postobstructing pneumonitis has been described, and morphologic features vary according to the size of the obstructed bronchus, the presence of an underlying pathologic condition in the obstructed segment of the lung, the duration of obstruction, and the nature of the foreign body.[5] The appearance of these lesions may mimic carcinoma on clinical, radiologic, and sometimes pathologic examination.[5] They occur most commonly in young adults and are usually asymptomatic or associated with nonspecific symptoms such as persistent cough, as in this patient.[6] Radiologic manifestations include a solitary nodule, an infiltrate, or, rarely, an endobronchial lesion.[2,6]

This case illustrates that the aspiration of a filter tip caused a clinical and bronchoscopic picture suggestive of a neoplasm. Xanthogranulomas of the lung may be a manifestation of postobstructive pneumonitis with or without an identifiable foreign body.

REFERENCES

1. Spencer H: Collapse, bronchial obstruction and its sequelae, shock lung and foreign bodies in the lung, in *Pathology of the Lung*. New York, Pergamon Press, 1977, Vol 1, pp 479–504.
2. Pearl M: Postinflammatory pseudotumor of the lung in children. *Radiology* 1972; 105:391–395.
3. Mason WE, Keats TE, Baker GF: Inflammatory pseudotumor of the lung: A case report. *Radiology* 1963; 81:824–827.
4. Reeder MM, Hochholzer L: Large (greater than 4 cm) solitary pulmonary mass. *JAMA* 1974; 229: 1493–1494.
5. Titus JL, Harrison EG, Clagett OT, Anderson MW, Knaff LJ: Xanthomatous and inflammatory pseudotumours of the lung. *Cancer* 1962; 15:522–538.
6. Bates T, Hull OH: Histiocytoma of the bronchus: Report of a case in a six-year-old child. *Am J Dis Child* 1958; 95:53–56.

TOBACCO PRODUCTS LIABILITY PROJECT

Wrongful death and personal injury product liability lawsuits against tobacco manufacturers may prove to be an effective strategy in the public health battle against cigarettes. The Tobacco Products Liability Project (TPLP) is attempting to encourage and advance this strategy on a national level. The goal of the TPLP is to curtail tobacco-induced morbidity and mortality and to shift the economic burden now imposed on society onto the tobacco industry. The litigation method offers several means of action to curb the use and acceptability of cigarettes:

- Awards of massive damages may drive up the price of cigarettes to as much as three or four dollars a pack, which in turn will reduce tobacco consumption especially among the young.
- Each personal injury case against tobacco producers that is reported in the mass media will further educate the public as to the negative consequences of smoking.
- Increased risk of liability may force tobacco companies and their advertising agencies to place conspicuous and detailed health warnings on packages and advertisements, comparable to medication inserts, and to desist from making misleading statements and using deceptive advertising imagery.
- Legal discovery and interrogatories may expose evidence of criminal negligence by cigarette manufacturers in failing to warn consumers.
- Such individual suits may open the way to large actions by insurers, states, and municipalities to recover damages directly from the tobacco industry for medical and disability costs now paid for by state and local taxes.

TPLP seeks the systematic development and coordination of such lawsuits. On January 26, 1985, at Northeastern University School of Law, TPLP convened the First National Conference on Tobacco Product Liability. With participation from attorneys, public health officials, physicians, and economists, the conference established both the research and logistical bases for TPLP (a project of the non-profit, tax-exempt Clean Indoor Air Educational Foundation. The second national conference will be held in Boston on January 10–12, 1986).

TPLP is seeking individuals with tobacco-induced diseases or cigarette-related burn injuries (and the families of such persons) who are willing to seek compensation from tobacco companies. It is also seeking experts willing to testify in all parts of the country, as well as endorsements from medical and public health societies.

TPLP is establishing a clearinghouse on tobacco product litigation to assist plaintiffs' attorneys, a directory of expert witnesses in a variety of fields, and a publication to report on medical and legal developments related to tobacco liability law.

Information on the selection criteria for potential plaintiffs and on other aspects of the Tobacco Products Liability Project may be obtained from its co-chairmen: Michael Charney, MD, 82 Marlborough St, Boston, MA 02116; Richard Daynard, JD, PhD, Northeastern University School of Law, 400 Huntington Ave, Boston, MA 02115.

First National Conference on

SUING THE TOBACCO INDUSTRY: A Product Liability Approach to Public Health

Saturday January 26, 1985
from 1:30-3:30 at
Northeastern University School of Law
400 Huntington Avenue, Boston, Mass.

—attorneys from five states who are currently bringing product liability cases against cigarette companies will discuss how to do it

—scholars from law and public health will discuss the implications for reallocating the costs of cigarette-induced diseases and for combatting the cigarette pandemic

—members of the Bar, law students, public health professionals, and the press are cordially invited

For further information call 437-3343 or write to the
"Tobacco Products Liability Project" at
Northeastern University School of Law
400 Huntington Avenue, Boston, Mass. 02115

The Tobacco Products Liability Project has stimulated widespread interest in the legal profession (and a drop in the prices of tobacco stocks following the publication of an article on it in *The Wall Street Journal.*) An example of the growing interest among lawyers in litigating claims based on disease caused by cigarette smoking is CigLit, organized by three trial attorneys in Texas. The litigation group will seek to hold tobacco companies accountable for promoting an epidemic "fed by a continuous harvest of young people as replacement smokers for those who die and quit." CigLit's address is PO Box 1152, Beaumont, TX 77704.

The clinician and smoking in Great Britain

DONALD J. LANE, DM

Although hints that smoking might pose a health problem appeared in medical journals more than 50 years ago, reliable epidemiological evidence that smoking is a killer did not appear until the 1950s. It was soon after that, in 1962, that the issues were put before the British public in the first Royal College of Physicians of London's report, *Smoking & Health.*[1] The British Royal Colleges are largely educational bodies: they set examinations, serve as a forum for postgraduate education, and oversee professional training. Until recently it has been unusual for the Colleges to issue reports for a wider public, but they have done this increasingly in recent years, and in no field more insistently than that of smoking. The Colleges have also advised the government on issues affecting public health, but for no issue has their voice been raised with so much determination as that of smoking.

That first report, factual and carefully argued, concluded that smoking-related diseases pose "the most challenging of all opportunities in preventive medicine." It contained a list of recommendations for public and government action. Neither paid much attention. When, after nine years had elapsed, the College produced its second report, *Smoking and Health—Now* in 1971,[2] it sadly had to record an increasing number of deaths from all smoking-related disorders. It also reported action by the government on only one front—a voluntary code on advertising negotiated with, and largely to the advantage of, the tobacco industry. This was backed up by just one piece of legislation: the banning of cigarette advertising on television in 1965.

The recommendations listed in the second report echoed in principle those recorded in the first. In the third report published in 1977,[3] the list reads depressingly the same, and by the time of the fourth report in 1983,[4] which again listed similar areas where action was required, the rising tide of frustration felt within the medical profession burst into print: "failures on the part of successive governments to take action to curb a lethal habit have angered and depressed those concerned for the public health." What is it that the profession demands so insistently, and why is it so difficult to achieve? The facts of the case are clear enough. In the United Kingdom, with a population of 55 million, there are about 100,000 deaths each year attributable to the effects of smoking. Physicians have fought for action to curb smoking on many fronts, but perhaps the issues can be crystallized into two areas of activity—education and legislation.

EDUCATIONAL EFFORTS

The dissemination of knowledge about the harmful effects of smoking and encouragement of a healthy lifestyle was at first delegated to local government authorities, but from 1968 has been in the hands of the Health Education Council (for England and Wales) and the Scottish Health Education Group. These bodies have been very active, particularly in schools and through the mass media; an evaluation suggests that their methods are effective. Admittedly they are financed by the government, but on a budget of £9 million (1 £ = approximately $1.17 US), they have to tackle all health issues, not just smoking. Their annual expenditure on smoking is about £2.5 million, which pales into insignificance beside the figure of at least £100 million, some 40 times as great, estimated to be spent by the tobacco industry on promotion. So this is a question of scale—of limited public resources set against the almost limitless funds of the opposition. And it is a question of public bodies trying to set smoking correctly in a context of overall health issues, against an industry hell bent on one all-consuming aim—to promote tobacco.

In addition to direct educational approaches, the Royal College of Physicians saw the need for a public information agency after the publication of its second report in 1971. Thus it set up Action on Smoking and Health (ASH). Again this has received financial support from the government, but this is currently running at a mere £136,000 per annum. ASH is used as a resource unit for the profession and for enquiries from all citizens. It services a group of Members of Parliament (MPs) committed to its aims and acts as a powerful and effective antismoking lobby. Factual evidence that it passes to individual doctors who have to face the media is so good that opponents from the tobacco industry will seldom, if ever, engage in public debate over the smoking issue. Although its stones are well aimed, it cannot yet claim the success of David over Goliath.

LEGISLATIVE OBJECTIVES

1. Taxation

The College sees two main areas where governmental action could make a real difference, and yet in which the government has been disappointingly indifferent: taxation and advertising. As the 1983 report detailed, the tax frac-

---

Dr Lane served as Secretary of the Working Party of the Royal College of Physicians that produced the College's fourth report on smoking in 1983. Sir Raymond Hoffenberg served as chairman.

Address correspondence to Dr Lane, Consultant Chest Physician, Osler Chest Unit, Churchill Hospital, Oxford, United Kingdom.

tion in the price of cigarettes was close to 0.70 in the early 1960s but fell to 0.63 in 1973. Since then there has been a steady return to 0.71, but if the government had not allowed the tax fraction to fall—particularly from 1969–1973—it is estimated that there could have been a sharp fall in cigarette consumption some 10 years earlier than that which is now occurring in Britain. The medical profession has no direct voice in fiscal matters. Like others, including the tobacco industry, it has the right to petition the Treasury and does so, especially during the period before the annual budget is set. Because governmental departments keep their spheres of influence to well circumscribed limits, it is unusual for health matters to influence fiscal decisions, but it is notable (much to the satisfaction of the profession) that on three occasions the health risk has been used as the reasoning behind the increase in tobacco taxation—twice under a Labor Government, and most recently in 1984 under the Conservatives. The increase of 10p (100p = 1 £) on the price of 20 cigarettes (about 10%) in the last budget was much more than would have resulted from inflation alone, and the Chancellor made clear that the excess was to discourage smoking. The evidence is that it does. But if the government really believed in this fiscal tool as a means of enforcing its health policy, it would have acted in this way earlier and much more drastically. It has waited until an increasing wave of public opinion has moved against smoking before stepping in: moving with the flow is easier and politically more acceptable than swimming against it.

2. Advertising

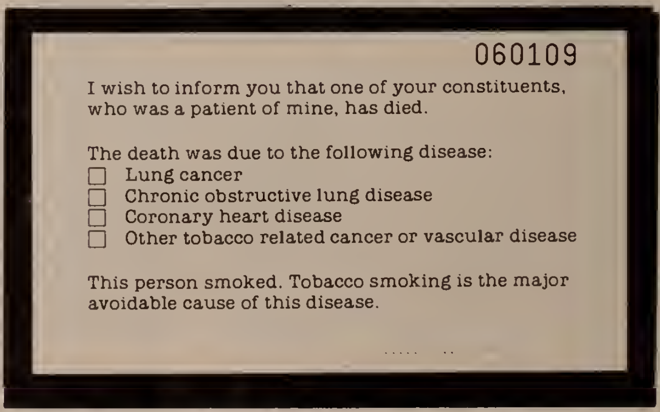
Even more vexing is the question of advertising. The ban on television advertising of cigarettes, enacted in 1965, is still the only legislation on the issue. The rest is uncontrolled or a matter for voluntary agreements. Such agreements prohibit the association of smoking with manliness, feminine allure, and social, sexual, business, or sporting success. In addition, smoking advertisements are not to appear in media directed to the young or on billboards adjacent to schools or playgrounds. Advertising should not be designed to persuade nonsmokers to take up smoking. Regrettably, the guidelines are frequently ignored, and the tobacco companies have no difficulty in finding alternative methods of bringing attention to their products. Sports sponsorship—Benson and Hedges cricket (Gallahers), John Player motor racing (Imperial Tobacco), State Express tennis (British American Tobacco), Peter Stuyvesant water sports (Rothmans), and many others—brings smoking before the public eye. Philip Morris' move of Marlboro cigarettes as a sponsor of Formula 1 racing flagrantly flouted the guidelines. The company clearly stated that they wanted "to promote a particular image of adventure, of courage, of virility." Sports sponsorship gets through to a large, often young and impressionable audience through television and newspaper coverage. Attempts to overcome these violations of the guidelines have largely failed. The sponsorship of art and cultural events is in a similar category, but the more recent use of cigarette brand names in the promotion of other interests such as clothing and travel is much more sinister. Again, the trouble here is that the medical profession has no direct say in these matters. Indeed the situation in sports sponsorship is such that even the Government Department of Health and Social Security has no say, for the matter is decided by the Department of the Environment. So the medical profession has to remain content to state its views, to make its recommendations, and to voice its discontent through College reports and public utterances.

A CHANGE FOR THE BETTER

Yet, this is not to say that all is gloomy on the anti-smoking front in Britain. This is far from the truth. The prevalence of smoking and the average number of cigarettes smoked per head of population is falling rapidly. Government surveys carried out biennially showed the prevalence of cigarette smoking in men to have been 52% in 1972 and 38% in 1982, the corresponding figures for women being 41% and 33%. This has occurred as a result of a change in public attitudes. Smoking is no longer acceptable in many circles. This change in attitude has been stimulated by the Colleges and is reflected in the educational strategy whereby the motivation to stop is changed from "cigarettes cause illness" to "healthy living means not smoking." The latest Health Education Council campaign, supported by a band of dedicated non-smoking athletes, is headed with the theme "pace setters don't smoke." Regrettably, success is often limited, even with these excellent techniques, in the lower socio-economic groups.

In 1984 ASH added to its public information lists of hotels and restaurants that provide space for non-smokers, insurance companies that give discounts for non-smokers, and transport authorities that are increasing their facilities for non-smokers. London Transport tops the list: they are currently running an experiment of totally non-smoking underground trains. British Rail has introduced entire non-smoking cars as a result of a passenger survey.

Also in 1984 the doctors' own "trade union," the British Medical Association, so successful in its lobbying for the campaign to introduce seat belts in cars, entered into the anti-smoking arena. Its campaign to harrass the tobacco industry and lobby Parliament is underway with pressure on MPs to promote anti-cigarette advertising legislation, media interviews, and a close scrutiny of all breaches of the existing voluntary agreements on advertising. Doctors are encouraged to send black-edged cards announcing the death of a constituent from a smoking-related disease to their MPs:



060109

I wish to inform you that one of your constituents, who was a patient of mine, has died.

The death was due to the following disease:

☐ Lung cancer
☐ Chronic obstructive lung disease
☐ Coronary heart disease
☐ Other tobacco related cancer or vascular disease

This person smoked. Tobacco smoking is the major avoidable cause of this disease.

Britain carries the lamentable reputation of having one of the highest death rates from smoking-related disorders in the world. Yet it is the place where medical scientists first established the relationship between smoking and fatal disease. Despite recent successes, it is sad but true that the profession has had the greatest difficulty in not only transmitting the significance of observed facts to the government, but also in persuading the government to take appropriate action—action which, if carried out when the first warnings were given, could have saved many hundreds of thousands of lives.

REFERENCES

1. *Smoking and health. A report of the Royal College of Physicians.* London, Pitman Medical, 1962.
2. *Smoking and health—Now. A new report of the Royal College of Physicians.* London, Pitman Medical, 1971.
3. *Smoking or health. A third report of the Royal College of Physicians.* London, Pitman Medical, 1977.
4. *Health or Smoking. Follow-up report of the Royal College of Physicians.* London, Pitman Medical, 1983.

Copies of *Health or Smoking?* can be purchased for $12.50 each (postpaid) from Urban & Schwartzenberg, 7 E Redwood St, Baltimore, MD 21202.

ELECTED OFFICIALS MUST ALSO BEAR RESPONSIBILITY

For the past year it has been my practice to send a letter to the Senators and Congressmen of each patient in whom I diagnose lung cancer, trying to point out that officials of our government must also bear some responsibility for the current pandemic of tobacco-induced disease.

More recently, I have begun asking patients who are dying of lung cancer to write for themselves to their representatives in Congress and also to write to the presidents of tobacco companies.

I am maintaining a file of these letters, which is expanding daily. Perhaps at some point they can be put to additional use.

JOHN J. MAY, MD
Attending Physician
Pulmonary Medicine
The Mary Imogene Bassett Hospital
Cooperstown, NY 13326

The Honorable Daniel Patrick Moynihan
United States Senate
Russell Senate Office Building
Room 464
Washington, DC 20510

Dear Senator Moynihan:

I have just had the onerous task of notifying one of your constituents that he has cancer of the lung. Like virtually all patients in whom I have made this diagnosis, this 64-year old mechanic is a cigarette smoker. Over the next one to two years, tens of thousands of dollars will be spent to provide him with medical care. The likelihood that he will survive these two years is well under 20%. Less than 10% of patients with lung cancer survive for five years.

I continue to be impressed that despite its avowed concern about the high cost of health care, Congress not only permits but actually fosters the production and sale of an addicting drug known to cause a variety of cancers as well as heart disease, stroke, emphysema, chronic bronchitis, etc. This is a drug which the Surgeon General has implicated in the deaths of one thousand Americans each day. A drug which has cost our society over 900 billion dollars in the 20 years since the initial Surgeon General's report.

Some portion of the responsibility for my patient's tragedy must rest with those in positions of public responsibility. I urge you to place the welfare of your constituents and your society over the welfare of a dishonest and avaricious industry.

Sincerely,
John J. May, MD

Physicians, smoking, and health in the Netherlands

HANS ADRIAANSE, DRS, RUUD HALFENS, DRS, MARIA J. DROP, PHD, JAN VAN REEK, DRS

Only recently has Holland (population 14 million) turned into a country with a majority of non-smokers. Although the percentage of men who smoke has gradually decreased from 90% to 41%, the percentage of women who smoke increased until the late 1960s before decreasing. The present percentage of women smokers (33% in 1982) is higher than the 1958 level (29%) (Table I).[1]

TABLE I. Smoking Behavior (%) in the Dutch Population Over Age 15.[1]

	1958	1963	1967	1970	1975	1979	1981	1982
Men	90	82	78	75	66	52	47	41
Women	29	32	42	42	40	38	36	33

Teenagers (especially boys) and older women smoke less than the national average and less than the overall adult population of 21 and over (Table II).[1]

TABLE II. Smoking Behavior (%) in the Netherlands for Age Classes 15–19 Years and Over 64 Years.[1]

	1958	1963	1967	1970	1975	1979	1981	1982
Men 15–19 yr	—	—	58	55	46	29	27	18
Men 65+ yr	88	76	83	74	66	57	51	43
Women 15–19 yr	—	—	57	57	48	39	30	27
Women 65+ yr	5	3	13	13	12	13	13	13

In 1981, the University of Limburg Health Panel Study was initiated. This is a nationwide survey on health attitudes and behavior in the adult population. In 1981 the study found that 49.1% of adults smoked, whereas in 1983 the percentage of current smokers was found to be 49.8%. It seems that the rapid trend toward non-smoking has stabilized, at least in the adult population.[2,3]

On the basis of 23 surveys carried out in the Netherlands during the last 25 years, van Reek[1] concludes that the emancipation effect among women has fully evolved and the influence of puritan norms on changes in smoking behavior is considered to be small. A further downward trend in smoking is expected due to increased awareness of the hazards of smoking.

Another argument for this expectation can be found if one considers the impact of the youngest cohorts on the national average (Table III). The cohort of men over 65, 43% of whom smoke, will be replaced by the young male cohort, 18% of whom smoke; and the cohort of women over 65, will be replaced by the young female cohort with a smoking rate more than twice as high. Taking into account the difference in life expectancy (72 years for men, 76 years for women) only a slight decrease in smoking in the overall population might be expected.

TABLE III. Smoking in the Netherlands by Age and Gender.[1]

Age (yr)	Percentage of smokers Men	 Women
15–19	18	27
20–34	45	45
35–49	44	36
50–64	45	27
65+	43	13

The Health Panel Study has found that the average starting age of smoking is 17 years. Age at the time of initiation of smoking was not related to the age of those who stopped smoking. More than half of the respondents think they smoke too much. Intentions to stop smoking are prevalent among 38% of those who smoke, with women indicating a greater interest in stopping. Young people and persons from lower social classes report more failed attempts to stop smoking. The reasons most commonly cited for failure to stop smoking include the following: "Smoking is tasty," "It calms you down," "It's a way of experiencing togetherness," "I just feel like it is what I want." Only 22% of the respondents (most of them women) smoke so-called "low-tar" cigarettes. Eighty-one percent of women who smoke choose filter cigarettes; 50% of male smokers choose filtered brands.

A comparison of the data of the first (1981) and second (1983) surveys of the University of Limburg Health Panel Study, leads to the conclusion that the cigarette pandemic has not declined. Between 1981 and 1983 5.5% of the sample started smoking, while 4.9% of the sample stopped smoking. Nearly 75% of those who started within that two-year period were former smokers. There appears to be no correlation with gender, age, or socio-economic status.

One of the most astounding results of the survey is that the percentage of physicians who still smoke (56%) is higher than that of the population at large (Table IV).[2,4] Similar results have been reported in previous surveys.

Forty-nine percent of physicians who smoke believe they are smoking too much (Table V). Fifty-three percent of smoking physicians do not plan to stop; 22% intend to

---

From the Departments of Health Education (Dr Adriaanse), Nursing (Dr Halfens), and Medical Sociology (Drs Drop and van Reek).

Address correspondence to Dr Adriaanse, Associate Professor, Department of Health Education, University of Limburg, PO Box 616, 6200 MD Maastricht, The Netherlands.

TABLE IV. Smoking Behavior (%) of General Physicians and the Population in the Netherlands, 1981–1983.[4]

Tobacco Consumption	Current Smokers		Former Smokers		Never Smokers		Sample Size		Response Rate	
Physicians	55*	56	30	33	11	11	74	100	55	60
Population	49	50	22	25	29	25	1,278	884	63	77

* Figures in first column under each heading represent 1981 values; second column, 1983 values.

TABLE V. Judgements about Personal Smoking Behavior (%) among Physicians in the Netherlands, 1981–1983.[2,4]

	Physicians		General Population	
	1981	1983	1981	1983
Smoke too much	54	49	63	63
Intend to stop smoking	32	22	37	36
Tried to stop smoking*	36	23	63	35†
Sample size	74	100	1278	884
N = smokers	41	56	627	440

* Overlap with "intend to stop smoking" group.
† Tried to stop last year but failed.

stop; 13% intend to cut down; and 2% intend to cut down to 50% of their current level. Twenty-three percent tried to stop one or more times during the year prior to being surveyed.

Physicians' perception of their professional responsibility in regard to the smoking habits of patients is paradoxical. Although 94% agree with the statement that "It is part of my task to point out to patients the dangers of smoking," and 61% agree that "My personal smoking behavior is relevant to the patient by whom I am perceived as an exemplar," only 22% agree with the statement that "In general, physicians' health behavior should set an example for patients." [1] When asked to mention healthy aspects of their own lifestyles, both the general population and the physicians rank highly frequent physical exercise, not smoking, and good nutrition. Among the unhealthy aspects of their lifestyle, physicians rank working situation higher than smoking, followed closely by stress. To the general population, smoking, alcohol consumption, and lack of exercise are ranked highest as unhealthy aspects of one's lifestyle.[8] It is hardly surprising that only 5% of the physicians think they have a healthier lifestyle than the general population.

In the United States, the percentage of physicians who smoked began rapidly declining in the 1950s following the publication of reports on smoking's relationship to lung cancer. By the late 1970s this rate, which had been as high as 67% in 1950, had dropped below 20%.[6,7] In the Netherlands, the medical profession seems to be lagging behind by at least two decades. The Dutch physician still preaches the double standard of "Do as I say, not as I do."

REFERENCES

1. van Reek J: Rookgedrag in Nederland van 1958–1982. Tÿdschrift *Alcohol & Drugs* 1983; 9:99–103.
2. Adriaanse H, Drop MJ, Halfens R, Philipsen H: *Leeft Nederland oke?* Zeist, 1981.
3. Halfens R, Drop MJ, Philipsen H: *Leefwijze en gezondheid,* een panelstudie. Maastricht, University of Limburg, 1984.
4. Adriaanse H, Drop MJ, Mey K de, Verboom W, Witte L de: *Gezondheidsopvatting en beroepsuitoefening.* Maastricht, 1984.
5. Vandenbroucke, JP, Kok FJ, Matroos A, Dekker E: Rookgewoonten van Nederlandse huisartsen vergeleken met die van de bevolking. *Ned Tijdschr v Geneeskunde* 1981; 125:4–6.
6. Centers for Disease Control: Smoking behaviour and attitudes of physicians, dentists, nurses and pharmacists. *Morbid Mortal Wkly Rep* 1977; 26:185.
7. Glanz K: Preventive health behaviour of physicians. *J Med Educ* 1982; 57:637–639.
8. Rijntjes AG, Dubois VE, Adriaanse HP: *Health Beliefs, Health-Related Behaviour and Professional Performance of General Practitioners: Their Role in Health Education.* 10th WONCA Conference Proceedings, Singapore, 1983, pp 203–208.

IK ROOK NIET, IK SPORT
als niet-roker heb je een sprintje voor
roken vermindert de conditie
Stichting Volksgezondheid en Roken

"I don't smoke. I play sports. As a non-smoker you are way ahead." Poster by the Netherlands Foundation on Smoking and Health, adopted by the Dutch Sports Federation. The Netherlands Foundation on Smoking and Health (address: Stichting Volksgezondheid en Roken, Frederik Hendriklaan 34a, 2582 BC, Den Haag, The Netherlands) was established in 1974 by the government in conjunction with the national heart foundation and the cancer association. The foundation is developing mass media campaigns to counteract smoking. Regrettably, although tobacco advertising has never been permitted on television or radio, all other types of promotion have proliferated. Local cigarette manufacturers are largely owned by American and British companies, but Dutch cigar factories produce 60% of the world export trade in cigars. Hence, the tobacco industry is a powerful lobby.

Economic dependence on tobacco manufacturing in Northern Ireland

An obstacle to prevention

GABRIEL J. SCALLY, MB

The hazardous effects of tobacco use by humans continue to come to light, as a small section of the medical profession attempts to combat this serious public health problem in the developed world. It has become increasingly clear, however, that the traditional health education message—that smoking will damage health—is of limited value in the absence of structural changes in society. What has also become apparent is that the tobacco industry will respond to threats to its market and its very existence in a highly sophisticated manner. Consideration of this response should rank as highly as does clinical and basic science research on the health effects of smoking. The web of political and economic interests that protects the tobacco industry has been dubbed "The Smoke Ring" [1] and can be seen to operate at a local level in Northern Ireland.

In common with the rest of the world, Northern Ireland has a smoking problem. Some 33% of its population smokes (1981, unpublished data, Department of Finance) and, using a method pioneered in Scotland,[2] more than 2,000 deaths per year can be attributed to smoking. With a population of only one and a half million, Northern Ireland is by far the smallest of the countries that make up the United Kingdom (UK). It is also the poorest, the most unhealthy and, as the world knows, the most troubled.

Recession has hit hard in an area already economically weak. The unemployment rate in Northern Ireland (21.9% in September 1984) approaches twice that of Great Britain (13.4%). Against this bleak background, the tobacco industry occupies a prominent position in the economic fabric. The history of the industry in Northern Ireland dates from 1810 and is now represented by two companies: Carreras Rothmans (a subsidiary of the South Africa-based Rembrandt Group) and Gallaher Limited (a subsidiary of American Brands Inc, NY). A report[3] prepared in 1983 for the tobacco industry showed that although Northern Ireland had only a 2.8% share of the UK market for cigarettes. the local industry accounted for about 20% of the total annual output of the UK tobacco industry, employed over 15% of the industry's UK workforce, and produced personal income of $50.2 million in 1981. Despite a decline of 30% in tobacco industry employment between 1971 and 1981, the general industrial decline in Northern Ireland has increased the tobacco industry's relative importance, and in 1981 it accounted for 4.9% of total manufacturing employment (Table I). A comparison of Northern Ireland with other Western countries is revealing (Table II): the number of persons employed in the tobacco industry per head of population is more than twice that of its nearest rival, the Netherlands.

GOVERNMENT SUPPORT FOR THE TOBACCO INDUSTRY

The economic importance attached to the tobacco industry in Northern Ireland can be seen from the high level of financial assistance provided by the government. Although by no means financially insecure, the industry is

TABLE I. Contribution of Tobacco Industry Employment to Total Manufacturing Employment in Northern Ireland, 1971 to 1981.

Year	Total Number of Employees in Manufacturing Industry	Total Number of Employees in Tobacco Industry	Percentage of Contribution of Tobacco Industry to Total Manufacturing Employment
1971	162,952	7,447	4.57
1972	157,775	7,104	4.50
1973	155,295	6,964	4.48
1974	153,219	7,021	4.58
1975	146,279	6,971	4.77
1976	139,542	6,616	4.74
1977	138,290	6,364	4.60
1978	133,380	6,459	4.84
1979	125,686	6,160	4.90
1980	120,880	5,823	4.82
1981	106,689	5,249	4.92

Source: Department of Economic Development, Northern Ireland.

TABLE II. Numbers Employed in Tobacco Manufacturing per 1,000 of Total Population, 1979.

Country	Total Population (×1,000)	Number Employed in Tobacco Manufacturing (×1,000)	Number of Employees per 1,000 Population
Canada	23,690	9	0.38
United States	220,584	59	0.27
Belgium	9,848	7	0.71
Denmark	5,117	3	0.59
France	53,480	11	0.21
F R Germany	61,439	21	0.34
Italy	56,914	20	0.35
Netherlands	14,038	23	1.64
United Kingdom:			
Great Britain	54,338	21	0.39
Northern Ireland	1,545	6	3.88

Sources: *United Nations Yearbook of Industrial Statistics 1980*, and *World Health Statistics Annual 1982*.

Address correspondence to Dr Scally, Senior Tutor, Department of Community Medicine, The Queen's University of Belfast, Institute of Clinical Science, Grosvenor Road, Belfast, BT12 6BJ, Northern Ireland.

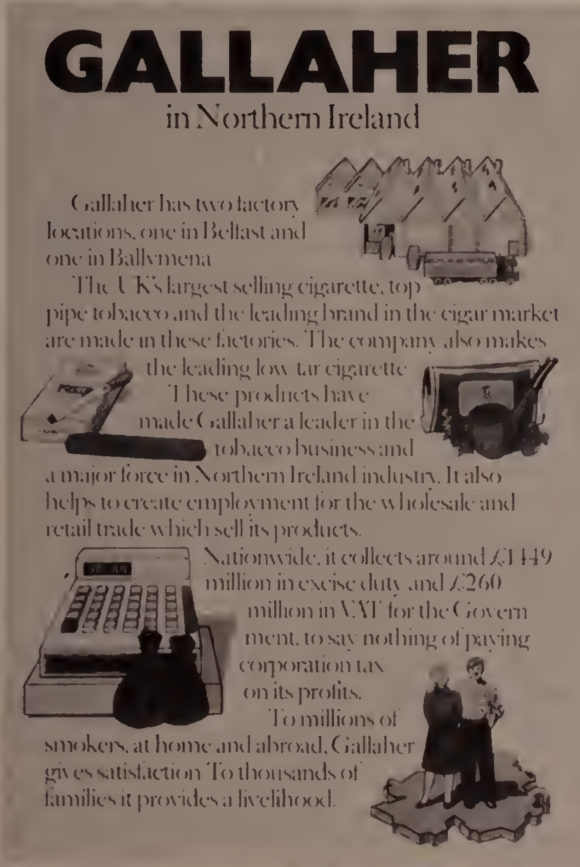
consistently among the leading recipients of financial assistance in the form of Standard Capital grants. In the financial year 1982–1983 the tobacco manufacturing industry received a total of $3.2 million. During the same period, government spending on health education—via the four Northern Ireland Health Boards and the Northern Ireland contribution to the London-based Health Education Council—totaled approximately $600,000. In terms of public expenditure, this gives a ratio of more than 5:1 in favor of the tobacco industry. In 1982–1983 the Health Education Council spent 27% of its income on anti-smoking activities. If this percentage were applied to all health education spending, the ratio of grants to the tobacco industry to spending on anti-smoking health education was almost 20:1.

TOBACCO COMPANY SPONSORSHIP

Sponsorship of sporting and cultural events by the tobacco industry has been interpreted as a belated attempt to buy respectability. In the face of demands from the medical profession for a complete ban on the sponsorship of sporting activities,[4] spending has been regulated by means of voluntary agreements between the industry and the UK government. Nevertheless, the sponsorship activities by tobacco interests in Northern Ireland include activities ranging from cricket to fly-fishing and country and western music to classical music.

Of particular note is the close connection between Gallaher Limited and the Belfast-based Ulster Orchestra. It is often argued by tobacco interests that advertising and sponsorship are aimed at persuading smokers to change brands rather than recruiting new smokers. If this is so, then associating with an orchestra that plays classical music is irrational, since the audience is likely to be drawn from the section of society least likely to smoke. There can be no other explanation than a quest by the tobacco industry for respectabilty. Since Gallaher's association with the orchestra began in 1981 there have been 30 performances dubbed "Gallaher Concerts," and of the 24 concerts held in Belfast during the 1984 to 1985 season, six were sponsored in this manner. Gallaher Limited is particularly proud of its sponsorship of the orchestra's 1983 tour of England and Wales, the highlight of which was a concert given in London in the presence of Her Majesty Queen Elizabeth the Queen Mother. That such prestigious associations were valued by the industry was confirmed by the announcement of a further three-year sponsorship deal with the orchestra amounting to $340,000.[5]

In the present economic climate in the UK, academic institutions have suffered reductions in their government-provided funding. Obtaining research funds from other sources has therefore increased in importance. Research

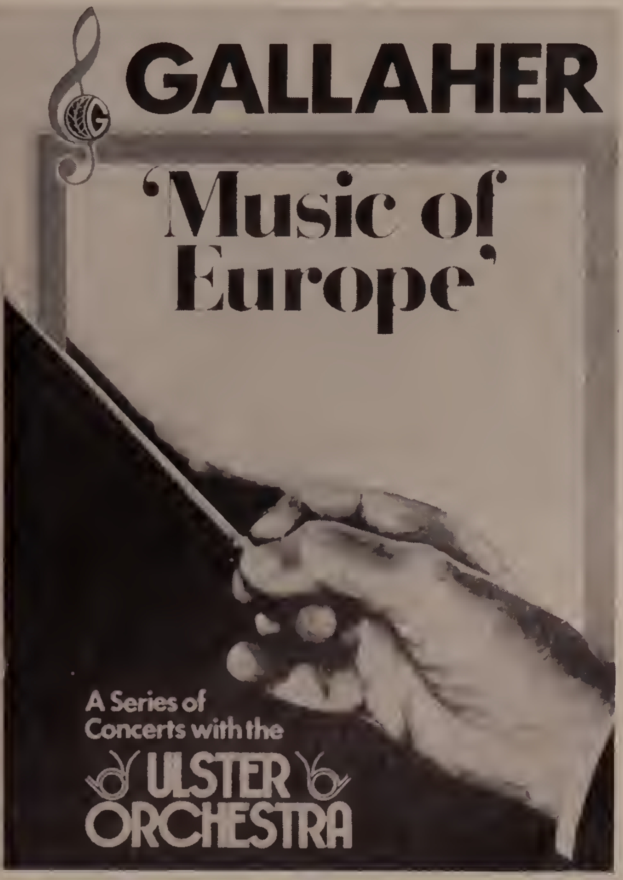

GALLAHER
in Northern Ireland

Gallaher has two factory locations, one in Belfast and one in Ballymena

The UK's largest selling cigarette, top pipe tobacco and the leading brand in the cigar market are made in these factories. The company also makes the leading low tar cigarette These products have made Gallaher a leader in the tobacco business and a major force in Northern Ireland industry. It also helps to create employment for the wholesale and retail trade which sell its products.

Nationwide, it collects around £1449 million in excise duty and £260 million in VAT for the Government, to say nothing of paying corporation tax on its profits.

To millions of smokers, at home and abroad, Gallaher gives satisfaction To thousands of families it provides a livelihood.


GALLAHER

'Music of Europe'

A Series of Concerts with the
ULSTER ORCHESTRA

funding given by Gallaher Limited to academic institutions in Northern Ireland totaled at least $210,000 in the three years from 1981 to 1983. The present vice-chancellor of The Queen's University of Belfast is the current chairman of the Independent Scientific Committee on Smoking and Health, which is a UK body established in 1973 and appointed by the government to advise the government and the tobacco companies on scientific aspects of smoking and health. The credibility of the committee was seriously compromised when the previous chairman, upon his retirement in 1981, accepted a post with Imperial Tobacco. Six months previously a prominent member of the committee's scientific secretariat had joined Gallaher Limited.[6]

It is clear that the tobacco industry in Northern Ireland is well integrated into the sporting, cultural, and academic life of the community and that such a close association is mutually desired. The net effect of this integration is that the purveyance of death and disease is rendered socially acceptable.

PREVENTIVE MEASURES ATTACKED

Prevention can be divided into soft and hard (or popular and unpopular[7]) forms. Soft prevention is that which is universally acceptable: health education films, leaflets and posters, and group lectures or talks. It is centered on individuals and aimed at encouraging behavioral change while displaying indifference to external anti-health pressures. Hard prevention, on the other hand, challenges social and economic structures and seeks to build collective support for political change. Such collective action inevitably threatens or seeks to restrict the commercial activities of the anti-health forces. Both hard and soft preventive measures have come under attack from a variety of quarters in Northern Ireland.

While surveys in Northern Ireland have demonstrated that the public attitudes towards anti-smoking initiatives are generally supportive, there is little evidence of this being translated into activity. The limited activity that exists is initiated by the health education departments of the four Health Boards and also, notably, by the pressure group Action on Smoking and Health, which has a strong history of campaigning. Anti-smoking activity is not regarded as a priority by health professionals in Northern Ireland; this includes some doctors working in public health who cite a slight decline in the number of smokers as a reason for continuing to ignore this pressing public health problem. Similarly, in the local press, little attention has been given to the problem. It might be suggested that this is due to the lucrative annual Rothmans Press Awards, but the real reason is probably that there is little or no activity to report.

It is generally accepted that progressive increases in taxation of tobacco products is an effective means of reducing consumption. For the government, this step has the added attraction of increased revenue. Early in 1984 the predictions were that a substantial tax increase on tobacco products would be imposed in March. A nationwide campaign was launched by tobacco interests to fight off this threat. In Northern Ireland this campaign was launched by spokesmen for the local tobacco industry, with support and participation from trade unions and the retail trade. Taxation of tobacco products was called "unfair and discriminatory" and much was made of the threat to jobs. A leading trade unionist stated,

> The North has suffered so many devastating manufacturing job losses in recent years that jobs in the tobacco industry must be safe-guarded. We are saying to the Chancellor, enough is enough. If there is to be any tax increase let it be a fair increase in relation to other industries and other products.[8]

The head of Gallaher Limited in Belfast put it more bluntly: "An increase in tobacco tax will mean jobs lost in Northern Ireland."[9] By coincidence, that same day his company announced record sales and record profits of $132 million.

The campaign failed in that the tax on cigarettes was increased significantly, but limited success could be claimed in that the tax on pipe tobacco was not increased. A major theme of the tobacco industry campaign had been that the tax on pipe tobacco was higher than that on cigarettes. It was also noted that in excess of half of the UK production of pipe tobacco was made in Northern Ireland. During a debate on the budget in the Northern Ireland Assembly, an elected body with limited powers, the tax increase on cigarettes was attacked by several representatives and defended by none, a rare display of political unity. Responding to the debate, the Minister of State confirmed that the possible effect on the Northern Ireland economy has been taken into account in the formulation of the budget and hinted that this was the reason behind the absence of a tax increase on pipe tobacco.[10]

Aspects of soft prevention have also come under attack. The first National No-Smoking Day was held on February 29, 1984, when smokers were asked to quit at least for the day. Among other activities, the campaign recruited a number of prominent people in Northern Ireland who promised to give up smoking for the day. The efforts of the anti-smoking lobby attracted widespread media attention. These efforts were, however, condemned by another prominent Northern Ireland trade unionist speaking on behalf of a trade union branch that represented 500 white collar workers in the tobacco industry: "It feels that such campaigns can do nothing but add to the sustained attack on the industry, which inevitably threatens jobs of the thousands of workers here."[11] The position of this branch clearly contradicted the policy of the national union which had stated its support for health promotion programs and smoking restriction in the workplace.[12]

TOBACCO AND UNEMPLOYMENT

In his stirring call for action at the close of the fourth World Conference on Smoking and Health, in 1979, Mike Daube described tobacco manufacturers as "merchants of death." If anyone still doubted their all-pervading influence, then the blocking by government of the appointment of Mr Daube to a post with the Health Education Council in London has surely dispelled any such illusions.[13] The apparent reason for the blocking of the appointment was that he had unnecessarily alienated tobacco interests.

In Northern Ireland these same merchants of death have integrated themselves into the fabric of society to such an extent that to dislodge them will require a major offensive by public health advocates. It would be tempting to take false comfort in what is termed soft prevention. Rather, concrete proposals must be brought forward that can both tackle this most pressing public health problem and prevent widespread unemployment. In the absence of well thought-out alternatives to employment in the tobacco industry, it would be easy for the pro-health forces to be suppressed and discredited particularly in a place as hungry for work as Northern Ireland.

There are at least two approaches by which an alternative employment strategy could be developed. First, industrial engineering consultants could be commissioned to undertake a study of non-harmful uses for existing plants and machinery. Cooperation in the study would have to be sought from the tobacco companies and the government. Second, using the example of employees in an English arms manufacturing company (Lucas Aerospace), encouragement and possibly finances could be made available to interested groups of employees, enabling them to explore alternative and socially useful ways to utilize their skills. Together, these suggestions could provide a constructive response to the "There is no alternative" argument frequently used in the defense of the tobacco industry and indeed in defense of current economic and industrial policy in the UK. It is important to avoid the simplistic equation that to prevent 2,000 deaths a year 5,000 jobs would be sacrificed. We should aim to have both a non-smoking population and full employment—both are essential components of health.

REFERENCES

1. Taylor P: *Smoke Ring: The Politics of Tobacco.* London, The Bodley Head, 1984.
2. Brotherston KG: *The Scottish Epidemic.* Edinburgh, Action on Smoking and Health, 1982.
3. Mackay DI, Edwards RT, Sladen JP: *The Tobacco Industry: Its Economic Significance in Northern Ireland.* Edinburgh, PEIDA, 1983.
4. Robson JG, Black D, Clark DH, et al: sponsorship of sport by tobacco companies. *Br Med J* 1982; 284:395–396.
5. Orchestra wins support to the tune of £260,000. *Belfast Telegraph* 1984; June 12, p 1.
6. Smoked in. *Financial Times* 1981; June 25, p 24.
7. Worden M: Popular and unpopular prevention. *J Drug Issues* 1979; 9: 425–433.
8. Tax 'threat' to North tobacco jobs. *Irish News* 1984; Feb 1, p 1.
9. Tobacco tax 'a threat to jobs.' *News Letter* 1984; Feb 1, p 6.
10. Northern Ireland Assembly: Official Report 1984. 1984; April 10, 9: 814–831.
11. No 'butts,' addicts will make an effort. *Belfast Telegraph* 1984; Feb 25, p 1.
12. Association of Scientific, Technical and Managerial Staffs: *Occupational Stress.* (An ASTMS policy document.) London, ASTMS, 1983.
13. Turner J: Government blocks top Health Education council appointment. *The Health Services* 1983; Oct 21, p 1.

The Scottish Epidemic

Estimated annual deaths from smoking in Scotland

	Men	Women
Ischaemic Heart Disease	2512	1567
Lung Cancer	2664	396
Bronchitis and Emphysema	854	220
Total deaths due to smoking		8213
Total deaths from all causes	63299	

Estimated annual admissions to hospital of residents of Scotland as a result of their smoking:

	Men	Women
Ischaemic Heart Disease	3703	1749
Lung Cancer	5466	853
Bronchitis and Emphysema	2855	1148
Total admissions due to smoking	15774	

Every day 772 constituents will, as a result of the three main diseases caused by smoking, occupy a hospital bed which could otherwise be used for the treatment of other patients. This represents a total cost of £5848076 per year in salaries for nurses and junior medical staff. It does not include consultant salaries, hospital running costs, community, ambulance or primary care costs.

—BROTHERSTON KG: *The Scottish Epidemic — Deaths and Hospital Admissions Attributable to Smoking by Parliametary Constituency, Region and Health Board.* Edinburgh, Scottish Committee Action on Smoking and Health at Royal College of Physicians, 1982, p. 3.

Slow but steady progress against smoking in Ireland

EMER B. SHELLEY, MB

Tobacco was probably introduced into Ireland towards the end of the 16th century;[1] it was imported from North America. Until the 20th century, tobacco was either smoked in a pipe or taken as snuff. Cigarette smoking did not become popular in Ireland until some decades after consumption had increased in other developed countries.[1]

In 1920, the average yearly consumption of cigarettes per adult in Ireland was 640, with a moderate increase to 930 by 1930. There was a dramatic rise in consumption during World War II and the post-war years, to 2,510 per adult per year in the 1950s. There was a further increase during the 1960s. By 1974, annual consumption reached 3,550 cigarettes per adult. In 1973, the three countries with a consumption higher than the Irish rate of 3,340 cigarettes per person were Canada (3,870), the United States (3,850), and Australia (3,440).[2]

The prevalence of cigarette smoking among adults in Ireland declined from 43% in 1972 to 35% in 1981 and 32% in 1983.[3] A similar reduction in smoking occurred in the population of the European Economic Communities (EEC) as a whole—from 44% cigarette smokers in 1960 to 39% in 1979.[4] The prevalence of smoking has been lower in the United States, having decreased from 40% in 1964 to 33% in 1981.[5]

In Ireland, as in other countries, the decline in cigarette smoking has been greatest among the more affluent and the better educated. In 1983, 40% of those in unskilled occupations were cigarette smokers, 36% of clerical and skilled workers, 27% of the professional and administrative groups, and 24% of farmers (Health Education Bureau, personal communication).

WOMEN AND CHILDREN

The increase in cigarette consumption during this century occurred first in men. In 1961 the average yearly consumption for adult women in Ireland was approximately one-third that of men. By 1975, women's consumption had doubled to 2,800 cigarettes per year. The increase in consumption was even greater in women of child-bearing age, with a particularly sharp increase between 1971 and 1975. The 1975 average of 3,250 cigarettes per year represents almost 10 cigarettes per day per woman between the ages of 16 and 44 years.[1]

The decline in smoking prevalence during the 1970s occurred later in women and was proportionately less than in men.[3] In smoking prevalence, as in other facets of life, the gap between men and women has become narrower. In 1983, smoking prevalence was 34% in men and 30% in women.

The decline in smoking has occurred in all age groups but has been slowest in the 25 to 34 year age group (Health Education Bureau, personal communication).·In 1983, 37% of those in this age group were current cigarette smokers, compared to 42% in 1972. The proportion of current smokers was lowest (24%) in those aged 55 years and over. The prevalence of smoking was 35% among those aged 16 to 24 years, 35 to 44 years, and 45 to 54 years.

Probably the most important and urgent public health problem in Ireland now is the prevention of addiction to tobacco among young people. This is especially so because of the increasing number of young people and the high proportion of the population they now represent. In 1979, 40% of the population was aged less than 20 years (total population 3.4 million).

There is evidence that there has been an increase in the prevalence of smoking among students in Ireland. In surveys of second-level students in 1967, 1970, and 1980, the proportion of boys who were regular smokers was 32%, 35%, and 34%, respectively.[5] The corresponding proportions for girls were 11%, 18%, and 26%. In the 1980 survey, 65% of regular smokers smoked 20 or more cigarettes per week. In addition to the one third of boys and the one quarter of girls who said they were regular smokers, a further one quarter of both boys and girls said they were occasional smokers. Sixteen percent of boys and 11% of girls described themselves as ex-smokers. In the absence of effective intervention, it can be predicted that addiction to tobacco and the resulting mortality and morbidity will continue to exert its toll on future generations in Ireland.

MORTALITY FROM SMOKING-RELATED DISEASES

None of the major prospective studies on the effects of smoking on health were carried out in Ireland. It is reasonable to assume, however, that the impact on mortality is similar to that in the United Kingdom and the United States. In such studies, more than 50% of all adult deaths have been shown to result from smoking-related diseases.

Mortality from all causes in Ireland started to decline from the mid-1970s.[6,7] This trend is apparent for both sexes and for all ages, except for men aged 55 to 64 years. A similar trend of lesser magnitude is suggested for mortality from coronary heart disease for men, but not for women. It is postulated that the trends in mortality are related to the decrease in smoking prevalence, particularly among men. Mortality rates from coronary heart disease for Irish women were the highest of any EEC country in the 1970s, with an average standardized mortality ratio of 220, when this ratio was 100 for women in all the EEC countries.[8]

The age-standardized death rate from all cancers combined has been increasing since at least 1940, and smok-

---

Dr Shelley is a co-author of "Smoking and health—the facts in Ireland," published under the auspices of the Research Working Party on Smoking and Health as a supplement to the *Irish Med J* (1983; 76:(suppl)1-29).

Address correspondence to Dr Shelley, Project Leader, Kilkenny Health Project, Parnell St, Kilkenny, Ireland.

ing-related cancers have increased as a proportion of all cancer deaths.[1] Since 1950, there has been an increase in lung cancer mortality rates for men, although rates for men under the age of 65 started to level off in the late 1970s.[7] Lung cancer mortality rates for women are lower than the male rates but can be expected to continue to increase, as women did not start to smoke in large numbers until the 1960s.

GOVERNMENT CONTROLS

The three main tobacco manufacturers in Ireland provided direct employment for 1,881 people in 1982, in addition to those employed in the wholesale and retail trade.[3] In that year, government revenue from excise duty and value-added tax on tobacco products was 272 million Irish pounds (1 Irish pound = $0.97), and this increased to 318 million pounds in 1983.[3,4] To put this figure in context, it is only in recent years that public expenditure on health services exceeded one billion pounds.

Despite revenue yield from tobacco taxation, governments in recent years have made efforts to control the activities of the tobacco industry. In 1964, according to a code of practice, there were restrictions on publicity and advertising of tobacco products. Advertising was prohibited on television in 1971 and on radio in 1976. Existing legislation, under the 1908 Children's Act, prohibits the sale of tobacco to those under the age of 16.[1] The Tobacco Products (Control of Advertising, Sponsorship and Sales Promotions) Act of 1978 and the 1979 regulations made under the act, updated and extended Irish anti-smoking legislation.[4,9] The advertising of tobacco products on radio, television, or outdoor billboards is forbidden, though it is permitted in adult newspapers and magazines. Advertising is also allowed within premises that serve as points of retail sales of tobacco products. Advertising that is allowed may only include a picture of the cigarette packet, its brand name, the corporate name or emblem, text referring to quality, and a health warning. Cigarette packets must also carry one of the following warnings: "Smokers Die Younger—Royal College Physicians of Ireland"; "Cigarettes Can Cause Cancer—Government Warning"; and "Smoking Seriously Damages Your Health—Government Warning."

CIGARETTES AND SPORT

The 1978 act and the regulations associated with it also relate to sponsorship by tobacco companies. Events may not be sponsored by the industry if participants are mainly under the age of 18 years. In addition, sponsored events cannot be advertised in publications directed at young people. Advertisements, while naming the sponsor, cannot be part of, or alongside, a tobacco product advertisement. The money which the tobacco companies may spend on advertising and sponsorship is controlled by the Minister for Health. This was first determined in 1980, following consultation with the industry. In 1983, permitted expenditures by the industry were almost 9 million pounds on advertising and 2 million pounds on sponsorship.

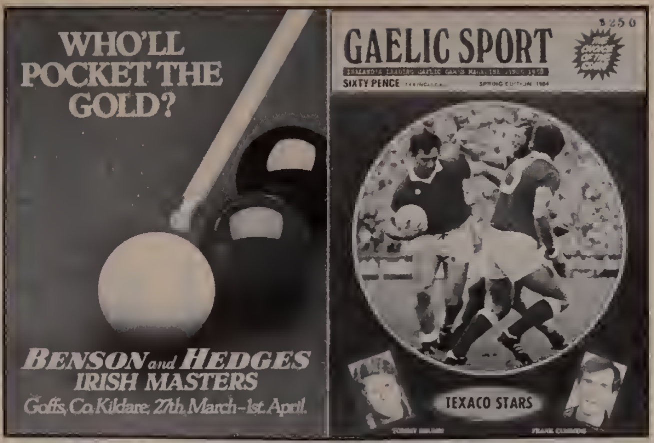

WHO'LL POCKET THE GOLD?
BENSON and HEDGES IRISH MASTERS
Goffs, Co Kildare, 27th March–1st April
GAELIC SPORT
SIXTY PENCE
TEXACO STARS

FIGURE 1. Irish sports magazine, front and back covers. Tobacco companies sponsor some sporting activities and place cigarette advertisements alongside images of athletic prowess.

The tobacco companies have continued to promote their products in association with sporting events (Fig 1). The Irish Open Golf Tournament in 1984 was advertised in a leading Sunday newspaper, with a golfing cartoon figure in color on the front page. The picture showed a crowd control barrier behind the golfer, complete with advertising banners—with "OLLS" to one side and "CARR" on the other, and, underneath, the caption "Carrolls Irish Open." There was also a large color advertisement showing the relevant cigarette packet on the newspaper's back page (Fig 2).

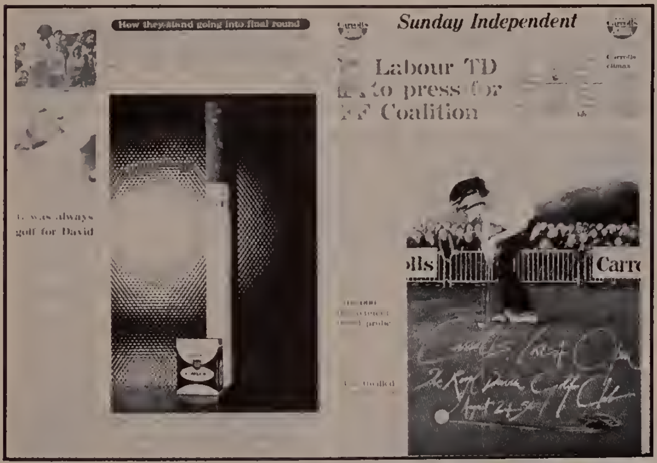


FIGURE 2. Artful dodge. Front page and back page advertisements for Carrolls cigarettes in Irish newspaper.

The tobacco manufacturers have also used their resources, where possible, to influence policy. The Irish Tobacco Manufacturers' Advisory Committee, which represents the major manufacturers—Carrolls, Gallahers, and Player & Wills—made a submission to the government-appointed Commission on Taxation.[3] The commission's Report on Indirect Taxation presented the economic situation of the tobacco companies and the tax yield from tobacco, with relatively little reference to the associated health, social welfare, and other deficits.

STRONGER LEGISLATION ANNOUNCED

The Health Education Bureau expanded its activities during the 1970s. In 1980, when it carried out a major anti-smoking campaign, the bureau spent 391,000 pounds on anti-smoking activities (Health Education Bureau,

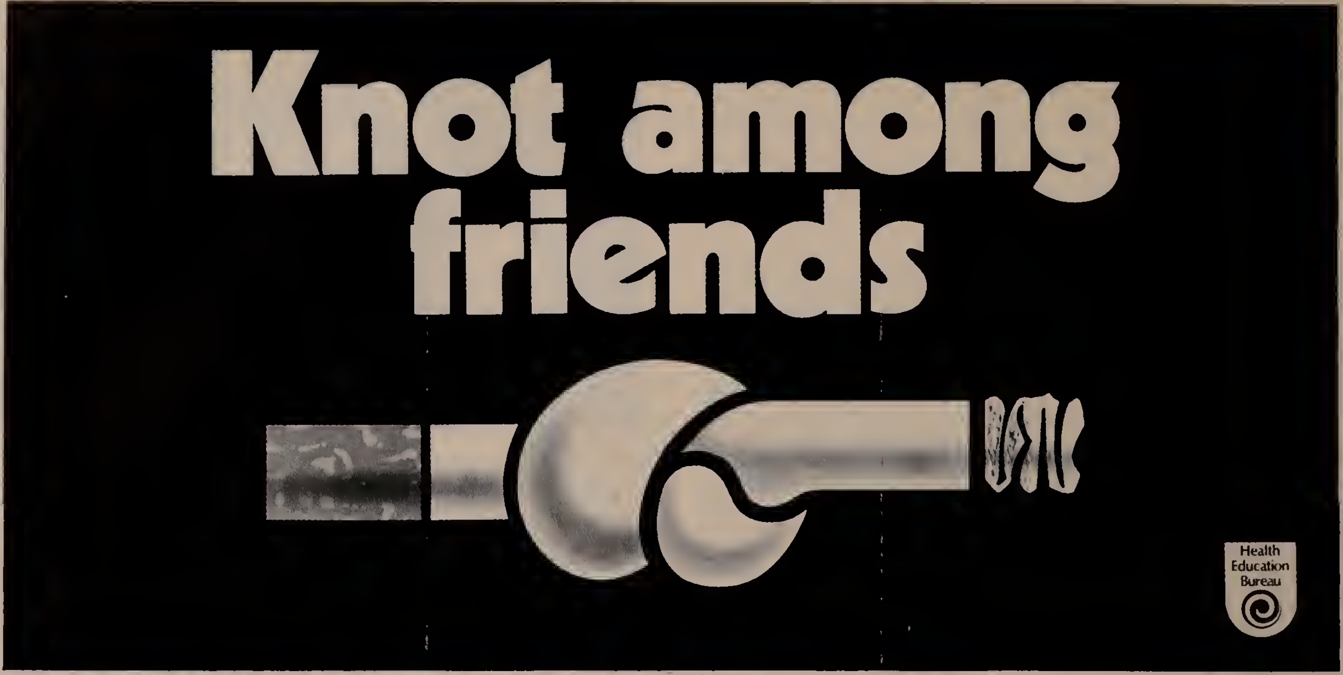


FIGURE 3. Recent theme of an Irish Health Education Bureau health campaign.

personal communication). This was not repeated in subsequent years, when expenditures were 115,000, 44,000, and 45,000 pounds. In 1984, it is estimated that 160,000 pounds was spent on anti-smoking activities. In that year, the bureau published Fact Sheets on Smoking, and it launched an advertising campaign, including the use of outdoor billboards—a medium forbidden to the tobacco industry. The advertisements use a picture of a knotted cigarette, with captions such as "Knot on," "Knot here," "Knot among friends" (Fig 3), and "Smoking is choking—don't flash the ash."

It is evident from the number of young people experimenting with cigarettes and the proportion who are regular smokers that existing legislation, its enforcement, the extent to which the industry breaches the spirit of the law, and current levels of health education are inadequate to reduce the smoking epidemic. The Health Education Bureau has been developing an educational approach to encourage the responsible use of all drugs, including tobacco, and the development of decision-making skills. Such "life-skills" programs are being implemented in many schools throughout the country.

The Minister for Health, Mr Barry Desmond, has announced his intention to introduce further legislation during 1985. Smoking will be restricted in public places, including hospitals, schools, public transportation, cinemas and theatres, and public sector offices to which the public has access. He has also proposed to update the legislation in relation to the sale of cigarettes to children. A 2% levy may be imposed on tobacco advertising, and the resulting revenue (estimated at 200,000 pounds per annum) used for health education. A system of rotational warnings on cigarette packets is also being considered.

The publication of "Smoking and health—the facts in Ireland"[1] defined the dimensions of the problem for policy-makers. The proposed legislation is a welcome recognition by the Minister for Health that tobacco smoking is a serious public health problem and that existing control measures are inadequate. The extent to which the legislation is enforced, monitored, and evaluated will reflect the true level of awareness by government and by health professionals of the importance of smoking as a public health problem in Ireland.

REFERENCES

1. Cleary A, Shelley E, Bourke G, Graham I (eds): Smoking and health—the facts in Ireland. *Irish Med J* 1983; 76:12(suppl):1-29.
2. Lee PN (ed): Tobacco consumption in various countries. London, Tobacco Research Council, 1975.
3. The Commission on Taxation: *Indirect Taxation*, third report. Dublin, The Stationery Office, 1984.
4. Health Education Bureau: *Fact sheets. Smoking*. Dublin, 1984.
5. O'Rourke AH, Condron L, O'Byrne DJ, Wilson-Davis D: Smoking—a study of post-primary schools 1980-81. *Irish Med J* 1983; 76:285-289.
6. Hickey N, Mulcahy R, Daly L, Graham I: Mortality from coronary heart disease—trends for the Republic of Ireland. *Postgrad Med J* 1984; 60:9-14.
7. Central Statistics Office: Report on Vital Statistics, 1950-1980. Dublin, The Stationery Office.
8. Dean G, Kelson M: The reported mortality pattern in the countries of the EEC for six common cancers and ischaemic heart disease. *Irish Med J* 1984; 77:98-100.
9. The Oireachtas: Tobacco Products (Control of Advertising, Sponsorship and Sales Promotion) Act, 1978. Dublin, The Stationery Office.

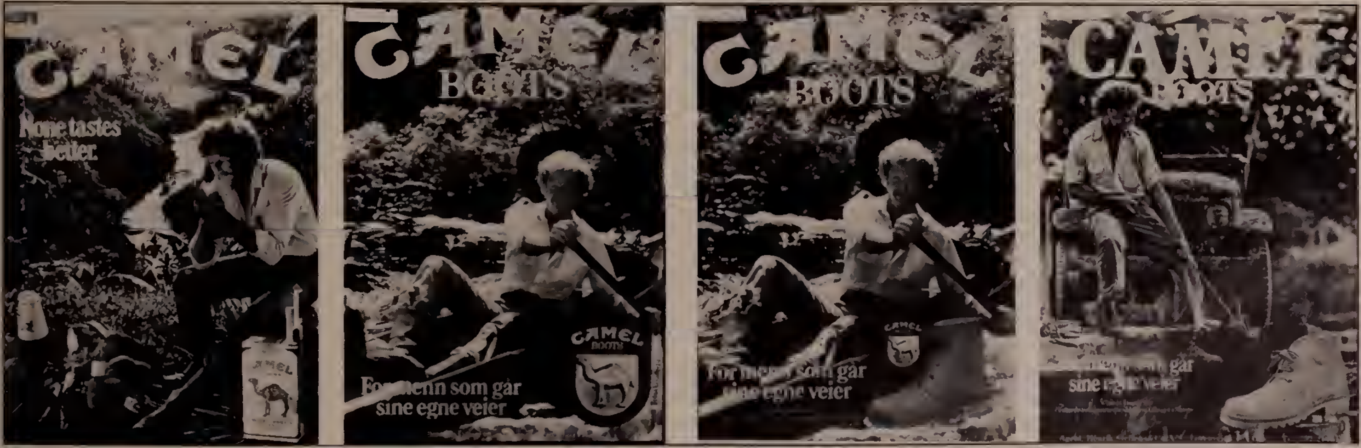
An attempt to circumvent the ban on cigarette advertising in Norway

ASBJØRN KJØNSTAD

The Norwegian Tobacco Act was passed in 1973 and came into force two years later. It contains a ban on advertising of tobacco products which is virtually without exemptions. The act brought about the disappearance of cigarette advertising in newspapers and magazines, ended television commercials, and removed all outdoor advertising for cigarettes such as posters, signs, neon lights, and other kinds of outdoor advertising, as well as shop displays. This reduction must be regarded as positive in view of the national health situation and the purpose of the act. Confronted with a decline in sales since the act took effect, the industry has tried numerous ways to circumvent the law.

During the first eight years in which the Norwegian Act was in force, Norwegian authorities dealt with approximately 100 violations of the law: that is an average of one case a month. Most of them ended with a statement by the Ministry of Social Affairs saying that the law had been violated. So far no such case has been brought before the courts; however, the following case has received considerable attention.

The largest Norwegian newspaper issues a weekly magazine called *A-Magasinet*. In 1981–1982 this magazine presented three versions of advertisements for "Camel boots." The first shows a man in a canoe going down a river. Most prominently featured in the picture are the man, the landscape, the heading ("Camel boots"), the trademark (picturing a camel), and text ("For men who go their own ways"). One can see that the man in the picture is wearing boots, but they do not show very well, as they are partly hidden down in the canoe and are partly of the same color as the canoe.


CAMEL
None tastes better
CAMEL
CAMEL BOOTS
CAMEL BOOTS
For menn som går sine egne veier
CAMEL BOOTS
For menn som går sine egne veier
CAMEL BOOTS
For menn som går sine egne veier

From left to right: cigarette ad; first version of boot ad; second version; third version.

The question is whether this is in conflict with the advertising ban of the Tobacco Act. The act says that tobacco products must not appear as part of advertisements for other goods or services. The provision must be interpreted on the basis of the pre-legislative works. It says that "also the use of tobacco products and the featuring of smokers or smoking is banned in advertisements of other goods and services."

In the "Camel boots" advertisement there are no persons smoking and no showing of packets of cigarettes or tobacco. But the entire advertisement is apt to turn the attention to a certain brand of cigarettes—"Camel." The trademark is the same for both the cigarettes and the boots, and the spelling is the same. The lettering is similar, and the letters appear in the same curved shape on the packets of cigarettes as in the "Camel boots" ads. The same symbol is used for both products.

Also, the ad used for Camel boots in Norway is very similar to ads used for Camel cigarettes in several foreign magazines, such as *TIME*, that are sold in Norway. The ad for cigarettes features the same man, in the same clothes and boots, and in the same surroundings. The National Council on Smoking and Health and the Ministry of Social Affairs agreed that the "Camel boots" ad was in conflict with the advertising ban of the Tobacco Act, and *A-Magasinet* was informed.

Two months later the other version of the "Camel boots" ad appeared in *A-Magasinet*. This one was very like the first version. The person, the canoe, the surroundings, and the heading are identical in the two versions. What is new in the second version is the prominence of a boot; the name, address, and telephone number of the agent; and that the picture of the camel is a bit smaller. These differences did not, however, make the second version of the ad legal.

The third version appeared in *A-Magasinet* one month later. The owner of *A-Magasinet* stated that the following changes had been made in the ad:

> The name "Camel" is now different from the name as it appears on the packets of cigarettes. The camel has been removed. The background motive is not shown in cigarette ads in international

---

Address correspondence to Mr Kjønstad, Professor of Law, The University of Oslo, Oslo, Norway.

Presented at the Fifth World Conference on Smoking and Health, Winnipeg, Canada, July 10–15, 1983.

publications. There is no doubt that the ad is for *boots*.

Many of the features of the first two versions making them seem like advertisements for tobacco have been removed. However, there can be no doubt that this is an ad for cigarettes. This becomes evident from the use of the name Camel, the same man, and the same surroundings as can be seen in foreign publications.

The fact that the intention is to advertise a brand of cigarettes has been admitted by RJ Reynolds in connection with the introduction of the offer. In tobacco trade publications such as *World Tobacco*, RJ Reynolds has said that this type of marketing was adopted "to expand awareness of the Camel brand and to fit in with the life-style of potential customers by providing catalogue shipping convenience for the man who wants to spend more leisure time at rugged outdoor activities."

In addition, the name Camel could not have been used without the consent of the manufacturer. Accidental use of the same name and trademark might be in conflict with both the Act on Trade Marks and the Act on Marketing and is therefore not likely.

Many people will not be able to distinguish between the different versions of the ad. In this case, a chain of associations is maintained to the ad for tobacco products. If it is possible to create a chain of associations by advertisements starting with a breach of the law and ending in a product which in itself is innocent, it would be quite easy to circumvent the advertising ban. Therefore, series of ads should be considered together.

The advertisements appearing in *A-Magasinet* for Camel boots constituted an infringement of the advertising ban of the Tobacco Act. *A-Magasinet* has been informed accordingly by the Ministry of Social Affairs, and the advertising of Camel boots has ceased.

During the preparation of the Norwegian Tobacco Act, such legislation was criticized by powerful opponents who maintained that the act was in conflict with constitutional principles of freedom of expression. Yet, since the act took effect no one in Norway, apart from those in the tobacco companies, has called for a return of tobacco advertising.

SMOKING CONTROL IN SWEDEN

The first anti-smoking society in Sweden was established in Stockholm in 1886 and remained active through the 1920s. In 1955 the National Smoking and Health Association (NTS) was founded—the first such agency in the world. Since 1964 the informational programs of the NTS have been funded mainly by the government as the result of a petition by a group of 25 scientists. The National Board of Health and Welfare helps coordinate smoking control measures with other public and private organizations.

The initial funds allocated to the NTS were not sufficient to begin a comprehensive anti-smoking campaign. To secure additional funding, in 1971 a special tobacco committee of the Ministry of Health and Social Affairs was created to present a better smoking program, and in 1981 another committee was formed to upgrade the program.

Sweden's smoking control program has several objectives: to prevent young people from smoking; to influence persons who smoke to change their habits; and to create a favorable public opinion to support program activities. Education about smoking is mandatory in Swedish schools. All cigarette advertising has been banned from television. Since 1977, all tobacco packages are required to carry health warnings and data on tar, nicotine, and carbon monoxide levels; there are 16 warnings that appear in rotation. Outdoor tobacco advertising is prohibited, and tobacco advertising in newspapers is restricted in size. No advertisements can appear in publications directed at young people. Moreover, no people or beautiful scenery can be shown in tobacco ads.

In 1978, the NTS began a new campaign aimed at creating a nonsmoking generation by the year 2000. Rather than publicize the health consequences of tobacco use, the campaign, by extensive use of the mass media, stresses a nonsmoking lifestyle as popular, modern, and fashionable.

Efforts to control smoking appear to be successful. From 1970 to 1980, the percentage of men reported smoking daily declined from 53% to 31%. Among women, the figure dropped from 34% in 1978 to 26% in 1980. Moreover, since 1978 there has been a downward trend in male lung cancer rates. Since the early 1970s smoking among adolescents has also decreased from more than 40% in 16-year-old boys and girls to about 30%.

In 1981, the special tobacco committee recommended that a separate law be enacted to limit smoking in public places. In 1983, the National Board of Health and Welfare and the National Board of Occupational Safety and Health recommended that no one should be subjected to discomfort or to the health hazards of tobacco smoke in public places and the work place. The recommendation accommodates nonsmokers and smokers, but priority is given to nonsmokers.

A detailed booklet *Smoking Control in Sweden*, can be obtained from the National Smoking and Health Association (NTS), Wenner-Gren Center, 22nd floor, S-113 46 Stockholm, Sweden.

Innovation in Iceland: graphic health warnings on tobacco products

THORSTEINN BLÖNDAL, MD, GUDJON MAGNÚSSON, MD

In Iceland (population 245,000) about 1,600 people die each year, and the excess mortality due to smoking is believed to be between 200 and 300 deaths a year. On January 1, 1985 new provisions of the Icelandic Tobacco Act* took effect, aimed at reducing the death toll by discouraging the consumption of tobacco and protecting non-smoking individuals against the effects of tobacco smoke. The new law, which was passed in Parliament without a single dissent, specifies that all packs of cigarettes, cigars, pipe tobacco, and snuff carry a health warning; that tobacco not be sold to persons under 16 years of age; and that smoking be illegal in public areas of government buildings, institutions, and businesses. Smoking is now limited in hospitals and is banned in clinics, day care centers, primary schools, and recreational facilities for teenagers. Restaurants are required to establish non-smoking sections. All advertising for tobacco and smoking accessories is banned, including window displays and imitations of tobacco products. (Tobacco ads were banned in 1971.)

Also under the law, strengthened educational programs about the harmful effects of tobacco are being implemented in schools and on the state-owned radio and television stations. Two pro mille of the gross sales of tobacco by the State Wine, Spirit and Tobacco Monopoly will be spent on government efforts to counteract smoking. In 1984 the state income from tobacco was 800 million Icelandic kroner ($20 million). (The entire state budget for 1985 is $780 million.) A Committee for Tobacco Use Prevention, appointed by the Minister of Health and Social Security, submits proposals to the minister on ways to use the funding.

A NEW KIND OF WARNING

The introduction of pictorial warnings on tobacco products is a major innovation in Iceland and may be unique in the world. Behind the new health warnings lie the following concepts and objectives:

- to establish better contact with the smoking population of 70,000
- to use clear, short, and unsophisticated messages
- to use several messages and to change them regularly
- to use a colored warning label of appreciable size, covering one quarter of the front or the reverse of cigarette packs, and of the front of cigar, pipe, snuff, and chewing tobacco packets
- to avoid "scare tactic" propaganda

Throughout the day smokers handle their pack of cigarettes—an excellent but too little used medium of communication. The use of a different color for each warning makes the message more readable, and simultaneously makes it more difficult for the tobacco manufacturers to hide the message by adapting the warning to the background of the packet. As smokers usually purchase the same brand of tobacco for an extended period of time, it is important to use different warnings on the same brand. A new color and illustration announce each different warning. It was decided to use eight warnings, two for cigars, and one for snuff and chewing tobacco. One of the warnings is used on cigarette packs as well as on packages for cigars and pipe tobacco. During the development of the various warning texts the graphic designers proposed the use of illustrations to reinforce the message. The experience with previous health warnings was that after a while smokers would no longer read them. The use of an illustration in connection with a certain text reminds the consumer of the association between a particular message and a specific illustration.

The warnings (Fig) are not dramatic, except that which accompanies the black figure in number 6: "Hundreds of Icelanders die each year due to smoking."

The cost of the warning labels is to be paid by the tobacco manufacturers, who are not permitted to place the warnings on transparent foil or other outer wrappings. Any person who violates the regulation shall be subject to a fine or, in cases of serious or repeated offenses, to imprisonment. The regulations apply equally to imported cigarettes, or the tobacco will be returned to the export country.[†]

COMMENT

In 1984, more than 40% of the adult Icelandic population smoked. The effect of the new legislation on the prevalence of smoking will be monitored. One of the important lessons that has been learned since the law took effect is that people are much more in favor of actions of this kind

---

Dr Blöndal is Chief Physician, Heilsuverndarstödin, Reykjavík. Dr Magnússon is acting Director General of Public Health.

Address correspondence to Dr Blöndal, Heilsuverndarstöd Reykjavikur, Baronsstigur 47, 101 Reykjavík, Iceland

* Copies of the Icelandic Tobacco Act, including the Act on Prevention of the Use of Tobacco, of May 28, 1984, and the regulation concerning warnings on tobacco products, of December 28, 1984, are available from Dr Magnússon, Landlaeknir, Laugavegur 116, 105 Reykjavík, Iceland.

[†] As might have been expected, foreign tobacco companies have threatened to end all cigarette exports to Iceland. Although the Icelandic tobacco market is very small, tobacco companies doubtless fear that other nations will follow Iceland's example.

In February 1985 the largest morning newspaper *Morgunbladid* alleged that in January an operative of an American tobacco company, using an assumed identity, visited the Icelandic embassy in Washington, ostensibly seeking information about Iceland on behalf of an anonymous American corporation interested in investing money in the country. The report cited questions by the woman about an American cargo shipping company, Rainbow Navigation, which controls all shipping to the US Naval Base in Keflavik. The woman is also reported to have contacted the State Department and the Pentagon under an assumed name. Sources for the newspaper article suggested that the tobacco company is looking for weak spots in the trade links between the two countries, as a way of influencing American congressmen to persuade the Icelandic government to abandon its new warning label law.

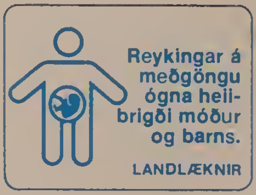
—Editor

than might have been anticipated. In a recent opinion poll, 59% of respondents considered the law as appropriate; 31% considered it too radical; and 10% wanted tougher legislation.

There has always been a problem for health educators to bypass the "psychological blockade" present in the minds of tobacco smokers against health information. The use of an illustration supporting the conventional health warning text on tobacco packs is a key advance toward more effective health education.

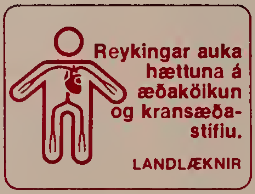
Health warnings

on tobacco packages in Iceland according to regulation no. 499/1984


Reykingar á meðgöngu ógna heil-brigði móður og barns.
LANDLÆKNIR

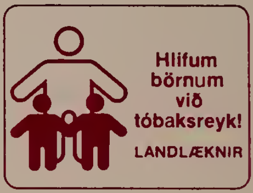
1. Smoking during pregnancy endangers the health of mother and child.

DIRECTOR GENERAL OF PUBLIC HEALTH.


Reykingar auka hættuna á æðakölkun og kransæða-stíflu.
LANDLÆKNIR

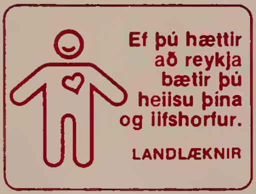
2. Smoking may damage your arteries and cause heart attack.

DIRECTOR GENERAL OF PUBLIC HEALTH.



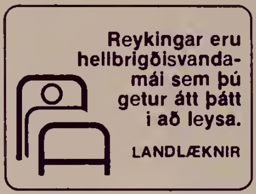
3. Protect children from tobacco smoke.

DIRECTOR GENERAL OF PUBLIC HEALTH.


Ef þú hættir að reykja bætir þú heilsu þína og lífshorfur.
LANDLÆKNIR

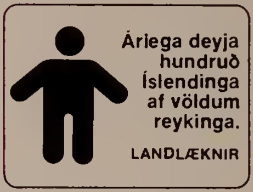
4. If you stop smoking you improve your health and increase your life expectancy.

DIRECTOR GENERAL OF PUBLIC HEALTH.


Reykingar eru heilbrigðisvanda-mál sem þú getur átt þátt i að leysa.
LANDLÆKNIR

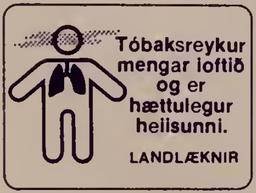
5. Smoking is a health problem you can help to solve.

DIRECTOR GENERAL OF PUBLIC HEALTH.


Árlega deyja hundruð Íslendinga af völdum reykinga.
LANDLÆKNIR

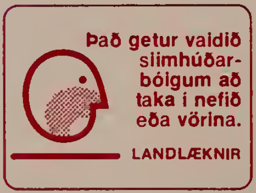
6. Hundreds of Icelanders die each year due to smoking.

DIRECTOR GENERAL OF PUBLIC HEALTH.


Tóbaksreykur mengar loftið og er hættulegur heilsunni.
LANDLÆKNIR

7. Tobacco smoke pollutes the air and is a health hazard.

DIRECTOR GENERAL OF PUBLIC HEALTH.


Það getur valdið slímhúðar-bólgum að taka i nefið eða vörina.
LANDLÆKNIR

8. Snuff and chewing-tobacco may damage the mucous membranes.

DIRECTOR GENERAL OF PUBLIC HEALTH.

Cigarettes, labels no. 1,2,3,4,5 and 6
Cigars and pipe tobacco, labels no. 3 and 7
Snuff and chewing tobacco, label no. 8

Smoking in France: a dismal picture

RENÉE BITTOUN

> *After being in Paris for several months, my three-year-old son was in need of a pediatrician. As I was a visiting research fellow at one of Paris' largest teaching hospitals, I availed myself of the staff pediatrician. Entering his rooms we were struck by a pall of smoke emanating from an ashtray overflowing with cigarette butts. Though convivial, the doctor began to examine my son, cigarette in mouth. After glaring at him for several moments, I commented that some of his patients were likely to remain ill from passive smoking. He said he was aware of these effects, but exposure has to be longer than just one short visit. I asked whether he had ever considered the exemplare role that he played within the community and to children particularly. This had never occurred to him. This encounter was typical—smoking was open and very common among the staff at the hospital.*
>
> *—account of a concerned parent*

Despite the laws of 1976 banning outdoor tobacco advertising, France continues to suffer one of the highest adult rates of smoking in the world (49% among men; 25% among women).[1] Surprisingly, 57% of medical practitioners in France (half of whom are women) are regular smokers—far higher than the national average.[2]

A survey of the smoking habits of student health professionals in France showed that 54.3% of medical students, 50.4% of pharmacy students, 47.5% of dental students, and 64.7% of nursing students were regular smokers.[3] Doctors who smoked believed that smoking, far from being socially degrading like alcohol abuse, is enhancing, characterizes a privileged social class, and is associated with stress in those overwhelmed with work and responsibilities.[2] Many thought smoking to be a minor, banal habit, and most were not convinced of the dangers of tobacco usage. Most admitted that they did not adopt the role of intervenor with patients who smoked or who had smoking related diseases.[2]

As in other countries, various agencies work for the advancement of non-smoking status in France, the most vocal being the private Comité National Contre le Tabagisme (CNCT) and the government Comité Français d'Éducation pour la Santé (CFES), set up in 1976 by then Minister for Health Simone Veil to augment the anti-tobacco advertising laws. Though publicly in agreement, these two agencies prefer to work independently.

The CFES deals with educational materials on all health issues, preparing briefs and reports, but it seldom has used the mass media for preventive health care issues. The CNCT publishes a quarterly journal, *Tabac et Santé*, with articles that range from local news to international surveys and research in the current status of smoking control. Within Paris alone, there are 45 centers affiliated with the CNCT for the treatment of "nicotinomanie." Treatments listed as available include acupuncture, homeopathy, and vaccines. Few results have been published (none in the English literature), and there has been sharp criticism of acupuncture as ineffective and scientifically unfounded.[4,5]

Traditionally, the nationalized tobacco industry (Service d'Exploitation Industriel des Tabacs et Allumettes, known as SEITA) has held a monopoly in the French market with a popular black tobacco; the French prefer Gauloises (the brand provided freely during National Conscription), followed by Gitanes.[6] A recent trend towards imported "blonde" cigarettes, often filtered, has led to foreign cigarette sales (Fig 1) that increased from 13% in 1977 to 37% in 1983.[7] Despite the changes in advertising laws in 1976, units of cigarettes sold in France have increased by 6% from 1978 to 1983, without a concomitant rise in population. To take advantage of the interest in foreign cigarettes, SEITA has launched several blonde brands with English names, such as Gauloises "Blue Way" and "News" (Figs 2, 3). Perhaps the only eyebrows that have been raised are those of the Association Générale des Usagers de la Langue Française, which has strenuously objected to the English names.

The image of the French smoker is often that of the intellectual at the café reading or writing and drinking alcohol or coffee.[8] These images are frequently encouraged on television, where debates and lectures often feature intellectuals who light up one cigarette after another. Children surveyed about smoking frequently say that a positive motivation to use tobacco happens when thought processes are needed and intellectual efforts must be made.[2] That young people think smoking is a natural social behavior is reflected in the numbers that are regular smokers: 45% of girls and 43% of boys under 18 years of age.

The use of alcohol in conjunction with tobacco is known to increase the risk of cancer of the mouth, larynx, and esophagus—the predominant forms of cancer in France.[6] Thus, the stereotype of the wine-drinking smoker is confirmed. Older French men also "droop" their cigarettes—holding the lit cigarette in the mouth without puffing—a custom that is facilitated by the use of corn paper around "black" cigarettes. Black tobacco is high in nicotine,[6] most of which is absorbed through the buccal mucosa, making deep inhalation less necessary. Today, however, most young smokers buy filtered cigarettes and inhale the smoke.[6]

Although only tobacconists and licensed cafés may sell and advertise cigarettes at their shops, covert advertising has been widespread, using loopholes in the laws to promote lighters and matches of specific well-known brands (Figs 4–6). Condemnation of these manipulations of the laws has led to litigation.

Smoking in France seems a socially and culturally acceptable behavior entrenched in tradition. The medical profession has shown little interest in providing the essential impetus needed to counteract the use of cigarettes.

Ms Bittoun is a respiratory physiologist who was awarded a research fellowship in France in 1984. In 1982, the Philip Morris tobacco company failed in legal maneuvers to prevent publication of an article by Ms Bittoun entitled "A tracheostomy for the Marlboro man" (*Med J Austr* 1982; 2:69–70).

Address correspondence to Ms Bittoun, Director, Smokers' Clinic, St Vincent's Hospital, Sydney, Australia 2010.

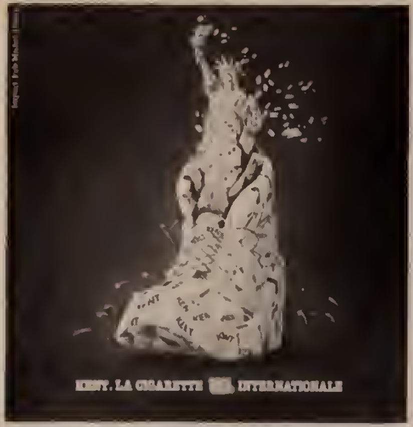
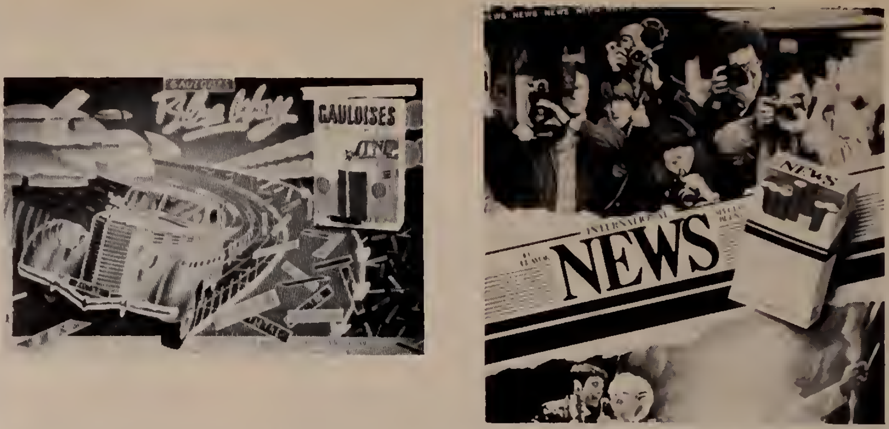
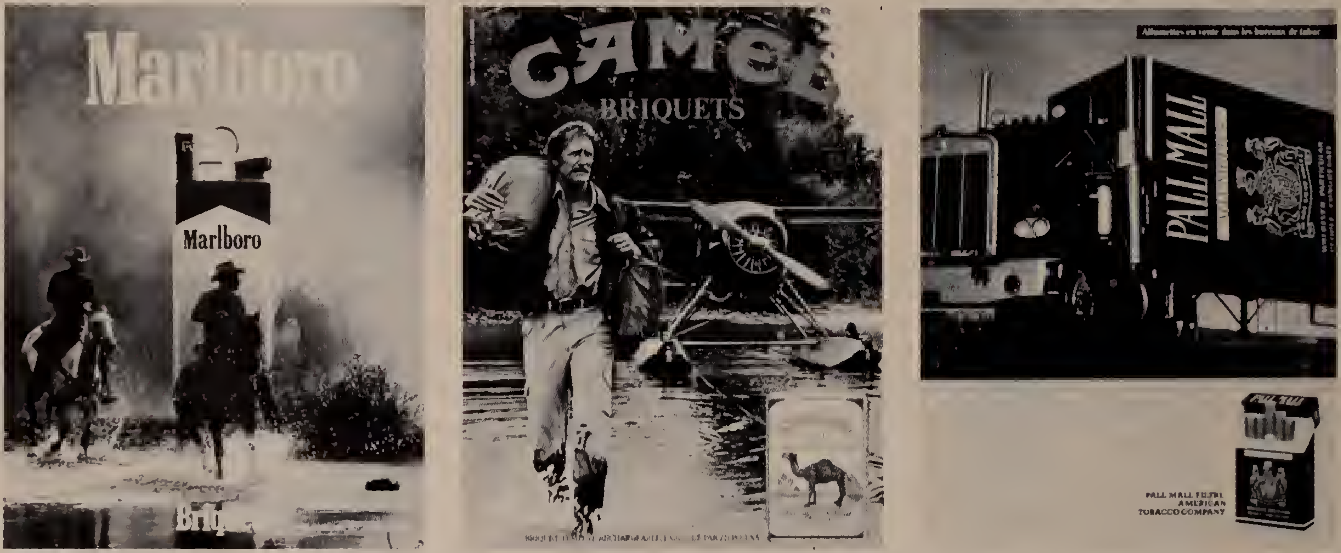


FIGURE 1. The image of the Statue of Liberty emerging from a sneaker is invoked to sell KENT cigarettes in France.



FIGURES 2, 3. Chic selling vehicle: English names on French-made cigarettes. The glorified image of photojournalists in advertisement for NEWS may also have been designed to enhance the social acceptability of cigarette smoking by members of the press.



FIGURES 4–6. End-run around the law: American cigarette brands advertised in the form of cigarette lighters (briquets) and matches (allumettes).

REFERENCES

1. de Givry Y: *International Digest of Health Legislation*, vol 35, no. 2, 1983.
2. "Monographies Européennes de Recherche en Éducation pour la Santé," no. 4, 1983.
3. Comité National Contre le Tabagisme: Le tabac vu par les futurs professionnels de la santé. *Tabac et Santé*, vol 40, October 1980.
4. Lagrue G, Poupy JL, Grillot JC, Ansouer JC: Acupuncture anti-tabaque. *La Nouvelle Presse Medicale*, vol 9, no. 13, 1980.
5. Lagrue G, Choppy-Jacolin M, Demaria C, Loufrani E, Dietling S: Comment lutter contre la dependance tabagique? *Medicine Generale*. September 1983, pp 72–74.
6. Wynder EL, Mushinski MH, Stellman SD, Choay P: Tobacco usage in France: an epidemiological study. *Prev Med* 1981: 10:301–315.
7. Comité National Contre le Tabagisme: Legere augmentation des ventres de cigarettes en 1983. *Tabac et Santé*, vol 55, no. 3, 1984.
8. Lang T, Degoulet P, Aime F, et al: Relationship between coffee drinking and blood pressure: analysis of 6,321 subjects in the Paris region. *Am J Cardiol* 1983: 52:1238–1242.

After reading about Billboard Utilizing Graffitists Against Unhealthy Promotions (BUGA-UP) (*N Y State J Med* 1983; 83:1333–1334), a group of health professionals and others called MUGA UP (Media Utilizing Graffitists, etc.) has been formed to exert pressure on magazines that accept advertising for tobacco products. MUGA UP's goal for 1985 is for 100,000 postage paid subscription cards to be returned to the publishers of these magazines, with a hand-written or rubber-stamped request to cease advertising cigarettes and other forms of tobacco. Such subscription cards can usually be obtained in quantity at supermarkets, in libraries, or, unfortunately, from the magazines in all too many waiting rooms of hospitals and medical offices.

JANET KAHLER
3412 West Rosewood
Phoenix, AZ 85029

Smoking and its promotion in Greece

A 20th century Trojan horse

ELIZABETH KEENAN

> *Greece is known in the whole world not only for her ancient monuments but for her tobacco as well.*
> —advertisement of the National Tobacco Board of Greece

The tobacco industry is regarded as one of the most productive sectors of the Greek economy. Tobacco is the main source of income for about 100,000 families, and more than 500,000 people (about 6% of the Greek population) are dependent on income derived from tobacco production and handling (Figs 1, 2) (*Tobacco International*, March 2, 1984). The government assists tobacco producers by fixing support prices for each crop, providing loans to enable growers to modernize their production methods, and subsidizing growers' incomes to encourage the cultivation of commercially desirable varieties.

In 1957 the government established the National Tobacco Board to assist tobacco farmers and promote tobacco production. The board oversees the tobacco industry and all aspects of the production, marketing, and export of Greek tobacco leaf and cigarettes. Greek tobacco is considered to be of high quality, with a relatively low nicotine content, characteristics which, according to the Tobacco Board, "give Greek tobacco an outstanding position in the international tobacco trade."

Greece exports about 80% of its tobacco crop each year, much of it to the United States. In 1983 tobacco exports earned $198 million in foreign exchange. But the other 20% of the crop, which supplied more than 90% of the domestic cigarette market, was even more valuable. Government taxes on cigarettes in 1983 totalled 34.5 billion drachmas (drs) (about $345 million)—about 5% of the national budget revenue.

Tobacco consumption and government revenue from the taxes thereon thus go hand-in-hand. Between 1982 and 1983, consumption of cigarettes in Greece rose by 5.1%. In the same period, largely as the result of an increase in retail prices (although at prices ranging from 34 drs (0.34¢) to 87 drs a pack, Greek-made cigarettes are still within reach of every pocket), the total value of cigarette sales, and consequently of tax revenue, rose by 30%.

In 1983, 26.6 billion cigarettes were smoked in Greece—roughly 10 cigarettes a day for every Greek over 15 years of age. (It should be noted, however, that in the same year Greece's population of 9.8 million was host to 5.3 million tourists.) During the last 10 years, cigarette consumption has increased by 50%.

The traditional *komboloi*, or worry beads, are increasingly giving way to cigarettes as a pacifier in the hands of Greek men. Women, few of whom smoked 20 years ago, are taking up the smoking habit in growing numbers. According to figures provided by the Department of Epidemiology at the University of Athens Medical School, 54% of men and 13% of women in urban areas are regular smokers. In rural areas, the proportions are 41% for men and 2% for women; these are probably comparable with urban figures of 20 years ago.

Among school children, smoking or experimentation with smoking has reached epidemic proportions. A small study conducted by the department at four high schools (two urban and two rural) revealed that among students in the third year (9th grade), 65% of boys and 50% of girls had smoked at least one cigarette, and by the 6th year (12th grade), 79% of boys and 82% of girls had tried smoking. (In another study the department found that of children who had smoked more than one cigarette, only 15% did not become regular smokers.)

FIGURE 1. A map from the National Tobacco Board of Greece highlights tobacco-growing regions.

FIGURE 2. A picture postcard depicts a young girl harvesting tobacco on a family-owned farm. Promotional literature of the Tobacco Board describes the crop as "the gold of sunny Greece."

EMULATING AMERICAN MODELS

Approximately 96% of the cigarettes consumed in Greece are manufactured locally. As well as their own brands, four of the six Greek cigarette manufacturers produce foreign brands under license from multinational tobacco giants such as Philip Morris and RJ Reynolds of the United States and Reemtsma of West Germany. Of the locally produced foreign brands, which account for almost a quarter of domestic output, by far the biggest seller is Philip Morris's Marlboro. Vigorously promoted in association with a rugged cowboy image that appeals strongly to the machismo ideal of the Greek male, the Marlboro

---

Ms Keenan, a former writer and editorial assistant with *The Medical Journal of Australia*, is an editor of the English-language monthly, *The Athenian*.

Address correspondence to Ms Keenan, c/- Worning, Flat B3, Arivou 18-20, Pangrati 116 33, Athens, Greece.

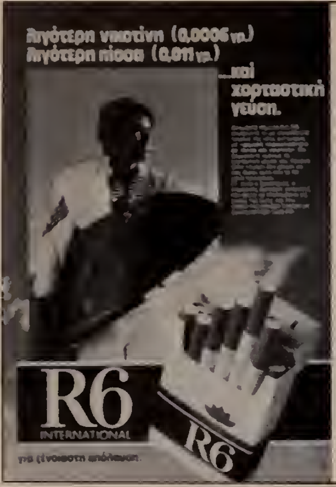
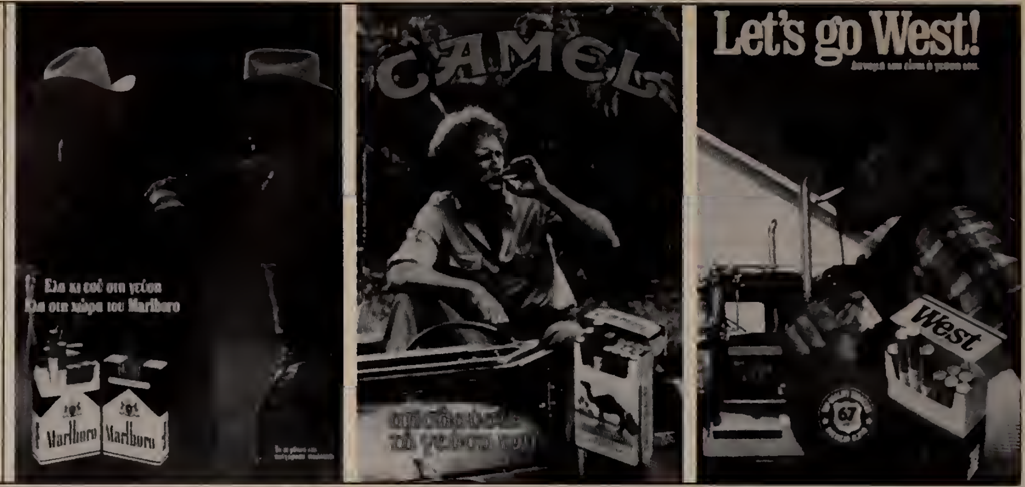


FIGURE 3. R6: less tar/nicotine.



FIGURES 4, 5, 6. Greek cigarette advertisements depict the American Adonis.

brand has captured 17% of the market since it was introduced in Greece in 1975.

Distant runners-up to Marlboro are Kent (British American Tobacco) and West (Reemtsma). West, introduced in 1982 with a truck driver he-man advertising image, boosted the sales of its Greek manufacturer SEKAP by 38% and has displaced its nearest rival, Camel (RJ Reynolds), from third position.

As a result of government-imposed restrictions, imports of foreign-made cigarettes declined by 20% between 1982 and 1983. The Greek government's concern to restrain imports indicates its determination to produce and support the local tobacco industry—not just the production of tobacco for export but the manufacture and sale of cigarettes—and disproves any claims that it is making a genuine effort to tackle the smoking problem.

Extensive advertising for Greek-made foreign "prestige" and "macho" brands in the "super-deluxe" (most expensive) category is wooing smokers away from cheaper domestic brands: in 1983 so-called "super-deluxe" filter cigarettes accounted for 87% of total cigarette consumption. High cost does not appear to be a deterrent in smokers' choice of brand—in fact, with its connotations of wealth, it may be a positive criterion.

Twenty years ago, advertisements for once-popular brands like Alpha Alpha and Ethnos featured ancient vase paintings and sculpture and slogans such as "Enjoy a Greek cigarette" and "A Greek cigarette of high quality." Today, cigarette advertisers have learned to appeal to smokers' insecurities rather than their patriotism or ethnic identity. The most common themes of cigarette advertisements—virility and elegance—play on the aspirations of ordinary Greeks to masculinity, wealth, and status. Astor, made under license from Reemtsma, and at 87 drs the most expensive locally made cigarette, takes a leaf from the book of Benson and Hedges (British American Tobacco) and offers "gold like the values that don't change . . . the golden taste of life." R6 (Reemtsma) (Fig 3) offers the youth-and-health conscious "less nicotine, less tar . . . the active modern smoker who knows how to enjoy life . . . now smokes R6." For men anxious to assert their virility (and women their independence) there are the Marlboro cowboys, the Camel explorer, the West trucker (Figs 4–6) and, most recently, the Paramont racing car driver (see attached story). All these brands present a masculine image using a rugged-looking male model, who, incidentally, is never Greek: advertisers believe Greeks prefer to identify with northern European or American stereotypes, who are seen as more "Western," modern, and successful.

GOVERNMENT FENCE-SITTING

In Greek society, there is no social stigma attached to cigarette smoking. On the contrary, smokers are seen as being almost as virile and vigorous as the advertising images they unconsciously imitate. According to Anna Kalandidi, a lecturer in the Department of Hygiene and Epidemiology at the University of Athens School of Medicine and a co-author of several studies on the effects of smoking, children and young adults begin smoking cigarettes mainly for social reasons. Social class, the example of parents and siblings, the desire to emulate sports and movie heroes (not to mention the hero-images supplied by the cigarette industry), and the age at which they leave school all influence young people to start smoking. The strongest influence of all, however, is peer pressure—which cigarette advertising plays no small part in generating. Cigarette advertisements screened in Athens cinemas invariably get a good laugh—often claps and cheers as well. Then, at intermission half the audience (the majority of whom are under 30 years of age) rush to the lobby to smoke.

In light of the widespread ignorance or indifference to the problems of smoking and the economic importance of the tobacco industry, it is perhaps not surprising that the Greek government's record in dealing with the cigarette epidemic is poor:

- There are no health warnings or product descriptions on cigarette packets.
- Although cigarette advertising is forbidden in state-owned media or on public buildings or property, it is almost impossible to walk down a major Athens street without seeing one or several cigarette advertisements—either on billboards or on

the ubiquitous street kiosks (whose main money-earners are cigarettes).

- There are few regulations governing the placement of advertising signs. At least one Athens hospital has billboards carrying cigarette advertisements within five meters of the gate, and the kiosk at its entrance is plastered with cigarette advertisements. An advertising executive readily admitted in a trade publication the "indirect advertising" value of posters placed in and around sports stadiums.

Legislation passed by the previous government bans smoking in public places, such as in cinemas and museums and on public transport, and stipulates that a special room be provided in all hospitals, public and private, where visitors, nurses, and patients may smoke.

No visible action is being taken against smoking or its promotion by either consumer or physicians' groups, and almost no assistance is available in the form of informa-

The Paramont man: the new cowboy on the block

In November 1984 the Greek cigarette manufacturer SEKAP began production of a new brand, Paramont, under license from the West German corporation Reemtsma. SEKAP already manufactures the Reemtsma brands Astor, R6, and West, which in 1983 accounted for 81% of the Greek company's total sales. (In 1983, after introducing West, SEKAP increased its sales by 38%.) Presumably, Paramont is the 1985 version of West.

Paramont cigarettes have been launched on the market with a 25-million-drachma ($250,000) advertising campaign divided into two phases. The "launching phase" consisted of "below-the-line" activity which aimed to create brand awareness by means of free giveaways to make people try the cigarettes; advertisements on billboards and in newspapers and magazines, which in this stage showed only the packet (Fig 8); Paramont stickers (Fig 9) and change-trays for "point-of-sale" advertising; a fleet of jeeps painted red and white (the Paramont colors) driven through central Athens towing white mini-trailers on which were mounted giant mock-ups of the Paramont pack.

The aim of the second, "position-building" phase was to create the "atmosphere" to go with the (by now well-recognized) packet. The vehicle for this was a series of advertisements—in newspapers, magazines, cinemas, and on billboards—featuring a masculine-looking model dressed as a racing car driver (Fig 10). The racing driver, it seems, is the third alternative to the Marlboro man (after the explorer and the truck driver)—the modern expression of the cowboy.

Paramont was first introduced in Switzerland, where it achieved only marginal success. In Greece, where the cigarette is having a second run, the advertising campaign will be much the same as the Swiss one, but will not mention tar and nicotine content (which in the Swiss variety were low but in the Greek version will be middle to high). No data are yet available on the campaign's impact on sales. It is calculated, however, to persuade people to associate Paramont cigarettes with the following:

- "Americanness." Paramont is marketed as a "true American-style cigarette." The line "Great American Taste" in newspaper and magazine advertisements is printed in English, and in cinema advertisements is spoken in English.
- Movies. The name's similarity to that of Paramount studios carries connotations of Hollywood, and in the cinema advertisements, a mini-"documentary" (showing divers feeding ravening sharks) is introduced by a frame reading "Paramont presents."
- Luxury and class. The cigarettes are labelled "Class A cigarettes."
- Masculinity. The cigarettes have a strong taste and the advertising model was carefully chosen as having a "masculine" image.

How many Greeks will fall for all this remains to be seen.

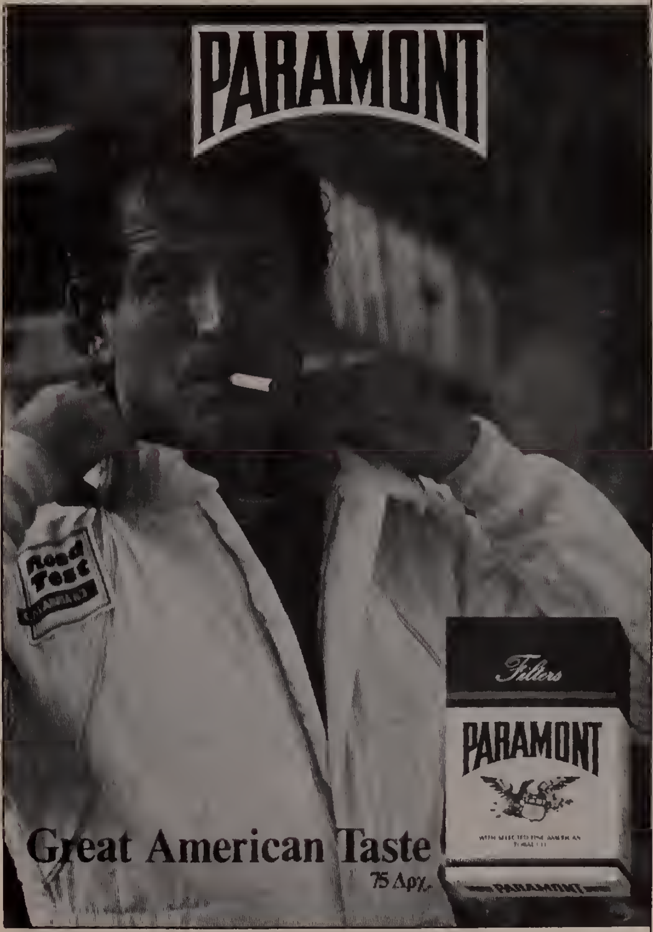
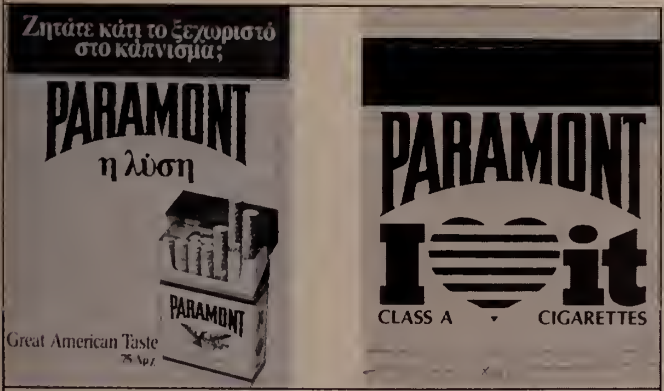


FIGURE 10. Racing car driver image for Paramont.



FIGURES 8, 9. Advertisements for Reemtsma brand Paramont.

tion, clinics, or counselling to smokers who wish to quit.

A campaign comprising a number of anti-smoking television and radio commercials, posters (Fig 7), and information pamphlets was launched by the Ministry of Health under the previous government in 1979 and 1980. This campaign was shelved in 1981 when the present socialist government came to power and the Department of Health Information was reorganized. The posters and pamphlets produced for the campaign were kept and were handed out to doctors, nurses, and teachers who asked for them. However, the supply of these resource materials has now run out, and although the department claims to have a budget of 50 million drs ($500,000) a year for health eduction, no more are being planned.

FIGURE 7. A 5-year-old health poster created under the previous government. The supply has run out, and no new campaigns have been developed.

According to the Ministry of Health, the previous government's anti-smoking campaign was not entirely successful: instead of reducing total cigarette consumption, it produced a shift from Greek to foreign brands, which advertisers had succeeded in presenting as having lower tar and nicotine content and, therefore, according to the logic peculiar to advertisers, as being more "healthy." (In fact, most foreign brands sold in Greece are made from the same tobacco as Greek brands.)

Whether by design or default, the campaign was interrupted indefinitely in 1981. The new government stopped telling people to quit smoking in order, it says, to concentrate on preventing them from starting in the first place.

A committee comprising representatives of the Ministries of Health, Youth, and Education was formed. After studying smoking prevention and education programs in other European countries, it worked out a two-stage plan of action. The first stage is to comprise teacher education. A series of seminars is now being conducted throughout Greece to inform teachers of kindergarten and elementary school children about the problems of smoking and to encourage them to pass this knowledge on to their students. Later, the Ministries of Education and Health intend to conduct a joint program in high schools, again using teachers as primary resources, to catalyze interest and to educate.

The second stage, still on the drawing board, will be a publicity campaign using the state-owned television and radio networks, the print media, and health education programs in schools of adult education.

Under pressure from the World Health Organization, the government has also held a few interdepartmental discussions on the possibility of introducing alternative crops to tobacco farmers, but no action has been, or is likely to be, taken.

The Greek government says it prefers to tackle the problem of cigarette smoking through education rather than legislation. However, in light of the slow and halting steps it has taken in its four years in office, this looks all too like another way of saying it prefers to sit on the fence for as long as possible.

CONFERENCES ON SMOKING

An international Conference on Smoking and Reproductive Health will be held in San Francisco on October 15–17, 1985. The conference will review current knowledge about the effects of smoking on fertility, gestation, and the female and male reproductive systems. Strategies for preventing smoking during pregnancy will be explored, and public policy implications of this work will be discussed. Information: Michael Rosenberg, MD, Family Health International, Research Triangle Park, NC 27709. Tel: (919) 549-0517.

The First World Conference on Nonsmokers' Rights will be held on October 5, 1985, in Washington, DC. ASH (Action on Smoking and Health) is coordinating the conference, the purpose of which is to teach and exchange strategies aimed at advancing nonsmokers' rights. Information: ASH, 2013 H St, NW, Washington, DC 20006. Tel: (202) 659-4310.

Smoking and health in the Soviet Union and Eastern Europe

RICHARD COOPER, MD

Born and raised to maturity in the United States, the epidemic of cigarette-related disease has spread to almost all other countries in the world. Few other public health problems weave together so completely the social and biological factors that produce the plagues of modern man. Despite its obvious political importance, relatively little has been written about smoking and health in the Soviet Union and Eastern Europe, and even less effort has been made to explain the social and economic roots of the problem. Much of the reason for this silence can be blamed on two major difficulties: lack of adequate information and a poorly formulated political perspective on the nature of social development in those countries. In recent years modest advances have been made against both these obstacles. Renewed interest in Soviet health was sparked in part by the reported rise in infant mortality, a controversy which also demonstrated the prevalent misconceptions about the nature of Soviet society.[1-3] Additional work has drawn attention to the similar public health trends experienced in the adult populations of the East and West bloc countries.[4-7] Central to the adult mortality trends is cigarette smoking.

RISING ADULT MORTALITY

Comprehensive mortality data from the USSR and Eastern Europe are not always available. Soviet vital statistics have not been published since the mid-1970s and cause-specific data from Eastern Europe are often difficult to interpret because of trends in the method of coding death certificates. Overall trends for the region do present a consistent pattern, however, and gaps in knowledge from one country can be supplemented by findings in another. The purpose of this article is to present trends for all causes of mortality for the region and to focus on specific causes from two countries: the USSR and Poland.

Data from the Annual Statistical Yearbook of the World Health Organization (WHO) show that crude mortality rates began to rise in the USSR in the late 1960s to early 1970s. (Crude rates are the only vital statistics from the USSR available from WHO.) Crude death rates do not take into account the increasing average age of the population and, therefore, must be subjected to age-adjustment to reflect trends over time. Rarely, however, do the trends in crude and age-adjusted mortality move in opposite directions. On further analysis of vital statistics from the USSR, it became apparent that a sharp increase had occurred in mortality rates after adolescence for men, with a slight increase for women.[8-9] In the rest of Eastern Europe, with the exception of East Germany, where data are not available, a similar upturn in death rates for men has been observed (Table I). The slope of the mortality curve for men aged 40 to 69 years from 1960 through the late 1970s was positive and significantly different from zero in all cases ($P < .05$). The average increase in mortality for all six countries was 0.8% yearly, or almost 15% over the 18-year period studied. Every country except Romania ended the period with a rate higher than reported for 1960, and Czechoslovakia alone experienced a decline in the last years reviewed, although rates remained well above the 1960 level. Only in the Soviet Union did rates rise for women.

As noted, cause specific data are not always available in the optimal form. In Table II, mortality from the two major cigarette related diseases—coronary heart disease (CHD) and lung cancer—are reported for both sexes combined, ages 45 to 64 years. (Since men have taken up smoking at a faster rate than women, just as in other parts of the world, rates that combine men and women understate the adverse trends; unfortunately, comparable data by sex are not available.) Large percentage increases are apparent in most countries, with the exception of Romania where only lung cancer was reported to increase.

The USSR and Poland can serve as examples for this region and can be looked at in further detail. In the Soviet Union death rates from CHD rose up to 40% among middle-aged men and women from the 1960s to the 1970s.[10] For lung cancer the increase was seen primarily among men over this period. Since CHD and lung cancer represent two of the three most important causes of death, their marked increase can account for the vast majority of the upturn in

TABLE I. Death Rates from All Causes for Men, Ages 40 to 69 years, in Eastern Europe and the Soviet Union, Selected Years, 1960–1978.* (rate per 100,000)

Year	Bulgaria	Czechoslovakia	Hungary	Poland	Romania	USSR†
1960	1173	1438	1367	—	—	1445
1961	1136	1420	1294	1442	—	—
1962	—	1507	1406	1479	1434	1489
1974	1228	1654	1497	1416	1274	1779
1975	1267	1611	1540	1478	1281	—
1976	1241	—	1550	1536	1298	—
1977	1336	—	1584	1579	1338	—
1978	1316	—	1710	1616	1394	—
Yearly change in rate	9.4	14.9	16.2	6.4	−2.5	26.4
Yearly percentage change	0.8%	1.0%	1.2%	0.4%	−0.2%	1.8%

* German Democratic Republic not available, rates age-standardized.
† Two-year average of year as designated and preceding year.

Address correspondence to Dr Cooper, Associate Chairman, Division of Cardiology, Department of Medicine, Cook County Hospital, 1835 W Harrison St, Chicago, IL 60612.

TABLE II. Death Rates from All Causes, Coronary Heart Disease, and Lung Cancer, Both Sexes, Ages 45–64, Eastern Europe,* 1964–1965 and 1974–1975.† (per 100,000)

Country	Cause	1964–1965	1974–1975	Percent Change
Bulgaria				
	Coronary heart disease (CHD)	81	161	+100
	Lung cancer	41	57	+39
Czechoslovakia				
	CHD	183	201	+11
	Lung cancer	80	83	+4
Hungary				
	CHD	216	219	+1
	Lung cancer	52	64	+22
Poland				
	CHD	126	132	+4
	Lung cancer	42	63	+22
Romania				
	CHD	129	101	−22
	Lung cancer	47	50	+6

* East Germany not available.

† Age adjusted by averaging of 10-year age groups.

all causes mortality.[10] Other factors that play a role in the etiology of CHD and lung cancer have been discussed in detail elsewhere.[4–6] Consistent trends in the major risk factors for CHD have been well documented in the Soviet Union.[5–6] Despite the emphasis given to alcohol consumption in the USSR[2] (and there is reason to believe this problem is overstated), the vital statistics support a more important role for the primary cigarette-related illnesses.

The Soviet Union is now a modern urban nation with an enormous industrial output. Most of Eastern Europe lags behind in economic development, and divergent health patterns could be anticipated. Poland was a country of small farmers until the last three decades and still has a large peasant population despite rapid urbanization. As in the USSR, marked upturns in all causes of mortality for men have been noted.[7] In addition, the causes of death that have led to this mortality rise are the same as in the USSR. Studying the period from 1970 to 1979, for which the best data base exists, a 20% increase in cardiovascular disease and a 40% increase in lung cancer have been observed.[7]

CIGARETTE PRODUCTION AND CONSUMPTION

Trend data on production and consumption are available only for the three major tobacco countries (Table III). While the Soviet Union is by far both the largest producer and consumer, the rate of increase has been more marked in Poland and Bulgaria. The most detailed cigarette consumption data are available from the Soviet Union. The trends in per capita cigarette use have been consistently upward, beginning at 1,059 in 1962 and reaching 1,786 in 1978.[6] (These figures include the "papyrosa," a rough cigarette-like product smoked primarily by peasants, now almost out of use.) A gradual steady increase is noted, with a 69% higher rate in 1980 compared to 1962. In Poland, smoking rates are even higher, with 2,633 cigarettes per adult reported for 1980.[7]

TABLE III. Tobacco Production (Prod) and Consumption (Cons) in the Soviet Union, Poland and Bulgaria, 1960–1977.*

	1960		1977		% Increase 1960–1977	
Country	Prod	Cons	Prod	Cons	Prod	Cons
Soviet Union	160	226	270	361	69%	58%
Poland	37	44	85	91	130%	107%
Bulgaria	51	14	135	78	165%	457%

* In thousand metric tons dry weight.

Smoking rates in the Soviet Union for men have been reported in the range of 44% to 69%, while among women only about 10% use cigarettes regularly.[5] Among men ages 20 to 29 years the rates are higher than average (about 60%), and women in that age range also smoke above average (17%).[11] These higher figures among young adults do not bode well for future trends. Average daily cigarette consumption per smoker was 18.1 for men and 14.7 for women,[11] similar to the United States and other western countries. In Moscow, boys 10 to 15 years of age already reported regular cigarette use at a 4% rate. An inverse relationship to education was noted in a survey in six major Soviet cities: 37% of those with a higher education smoked, compared to 60% with a secondary education, and 65% with a primary education only.[12] In Czechoslovakia it was reported in a factory survey that 42% of manual workers smoked, whereas only 24% of those in research and development did so.[13] A population-based cardiovascular survey in East Germany reported smoking rates of 39% among men and 14% among women between the ages of 30 and 49 years.[14] Bulgaria is a major tobacco producer in the Eastern bloc, and smoking rates there are even higher than in other countries. In Hungary, for reasons that are unclear, women have taken up smoking almost as much as men. Tobacco represents an important product for trade, and the Soviet Union imports raw tobacco from over a half a dozen other producing countries.[15]

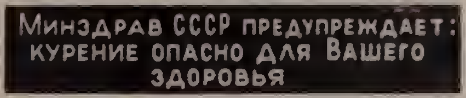
COMMENT

The cigarette has been a uniquely American contribution to modern world culture. It should be noted that in this presentation of cigarette consumption and related disease trends in the East bloc countries, the focal point is the similarity to the situation in the United States, rather than any special condemnation of the health record of the so-called socialist countries. Indeed, the US produces twice as many cigarettes as does the USSR, and per capita yearly consumption is 4,000 in the US and 2,000 in the USSR.[16] Although some of this imbalance reflects lower smoking rates among women in the USSR, the US remains the unchallenged world leader in cigarette consumption. Mortality data further demonstrate remarkably similar rates of CHD and lung cancer in Soviet cities and the Slavic regions as found in the US.[4,10]

Periods of rising mortality among adults are also not unique to the Soviet Union and Eastern Europe. In the first 15 years after World War II, a group of countries, including the US, Japan, Chile, and Czechoslovakia, experienced rising adult mortality for at least one major sex-race group.[10] In general the causes have been the same: an increase in cigarette use and more fat in the diet. The pattern in the East bloc countries is different only by virtue of a steeper slope of the mortality curves; this phenomenon may represent the much quicker tempo of economic development and the more abrupt introduction of cigarettes and a high fat diet in those countries. It is also possible that the hardships of World War II may still be exacting a toll from this generation, although this effect is hard to measure.

One important East-West difference cannot go unnoticed: the Soviet Union and Eastern Europe have centrally planned economies in which the intermediary of regulation and all the obstacles that private industry is able to bring to bear on that process are absent. One wonders why policy makers in those countries do not use the levers available to them through state control of tobacco production and advertising. On the one hand tobacco is recognized to be an enormous social menace, yet on the other hand production continues to rise. It can only be assumed that on balance the government derives substantial benefit from the tobacco crop. Tobacco is a cash crop without equal, and, contrary to some opinion, all Soviet industry operates on a profit basis.[17] Further, as with alcohol, tobacco products absorb an important portion of the purchasing power in an economy in which the consumer usually has more cash than items he or she wants to buy. As described in detail by Millar,[18] many of the social and economic processes at work in these countries lead to a similar market economy as found in the US. In effect, the economy now designated as socialist can in fact be regarded as a distinct sub-species of capitalism.[10] In recent years it has become apparent that China has led the way in introducing overt capitalist methods and has further demonstrated how the socialist economy incorporates all the basic elements of capitalism.[19,20] Viewed in this way it is not difficult to understand how the disease patterns have come to approximate each other. Economic factors have been paramount in the development and maintenance of the cigarette epidemic; one could argue that the identical outcomes in the US and USSR reflect the working out of similar economic laws. Further crucial evidence on the attitude of the government toward smoking policy lies in the social class gradient of cigarette use. The privileged classes generally introduce the new diseases, since they have the opportunity to experience the first exposure. Thus CHD was originally more common among the rich and middle class. As the hazards associated with social practices are better understood, the social class relationships are reversed, and the unhealthy products are "dumped" on the working class and poor. Sales—and by extension profits—are maintained, yet the educated and affluent groups are protected, while the government appears to promote "free choice." The limits of choice when different classes occupy distinct social universes have never been thoroughly examined. The US cigarette industry not only encourages smoking among minority and white working class groups in the US, but it also aggressively "dumps" its product on other countries around the world. The USSR appears to have adopted the same double standard: protecting the educated while exacting a tribute in health from the working class.

The Soviet cigarette package carries a subscript warning which says, "Smoking will harm your health. The Ministry of Health":



Минздрав СССР предупреждает:
курение опасно для Вашего
здоровья

This hypocritical effort to divorce one branch of government from the actions of another carries no more weight in the USSR than it does in the US. As long as powerful profit-motivated interests hold sway within either government, the population will continue to be poisoned with tobacco and a host of other health-destroying products.

REFERENCES

1. Davis C, Feshbach M: *Rising Infant Mortality in the USSR in the 1970s.* Foreign Demographic Analysis Division, Department of Commerce, September 1980 (Series P-95, No. 74). Washington, DC, Government Printing Office, 1980.
2. Eberstadt N: The health crisis in the USSR. *New York Review of Books.* February 1981; 28:22-31.
3. Syzmanski A: On the uses of disinformation to legitimize the revival of the cold war. Health in the USSR. *Int J Health Serv* 1982; 12:481-496.
4. Cooper R: Rising death rates in the Soviet Union: the impact of coronary heart disease. *N Engl J Med* 1981; 304:1259-1265.
5. Cooper R, Schatzkin A: Recent trends in coronary risk factors in the USSR. *Am J Public Health* 1982; 72:431-440.
6. Cooper R: Smoking in the Soviet Union. *Br Med J* 1982; 285:549-551.
7. Cooper R, Schatzin A: Rising death rates among Polish men. *Int J Health Serv* 1984; 14:289-302.
8. Naseleniye SSSR 1973, Moskva, *Statistika*, 1975. (In Russian, "Population of the USSR," 1973, Moscow, *Statistica*, 1975).
9. *Vestnik Statisiki*, Selected Years (In Russian, *Statistical Herald*).
10. Cooper R, Schatzkin A: The pattern of mass disease in the USSR: a product of socialist or capitalist development? *Int J Health Serv* 1982; 12:459-480.
11. Olenikov SP, Glazunov IS, Chiazova LV: Smoking among men and women of various age groups. *Ther Arch* 1981; 14:111-115 (Russian).
12. Chazov LV, Okeinikov SP, Shishova AM: Study of multifactor prevention of coronary heart disease in a free population. *Cardiovascular Epidemiology Newsletter*, Council on Epidemiology, American Heart Association, Dallas, TX, 1981; 80:140-141.
13. Simon J, Cajzl L, Kraus J: Longitudinal primary preventive study of coronary heart disease in Skoda Works, Pilsen, Czechoslovakia. *CVD Epidemiology Newsletter*, Council on Epidemiology, American Heart Association, Dallas, TX, 1983; 34:75-77.
14. Gunther KH, Bahre D, Oertel R, et al: The Cottbus general practitioners preventive study, Substudy Senftenberg: Smoking habits. *CVD Epidemiology Newsletter*, Council on Epidemiology, American Heart Association, Dallas, TX, 1983; 34:51-55.
15. Market prospects for tobacco in the Soviet Union. *Foreign Agriculture Circular*, Washington, DC, US Department of Agriculture, FT4-4, 1978.
16. Tobacco—Hazards to health and human reproduction. *Population Reports*, Series L, No. 1. Baltimore, Population Information Program, Johns Hopkins University, 1979.
17. Gregory PR, Stuart RC: *Soviet Economic Structure and Performance.* New York, Harper and Row Publishers, 1974.
18. Millar J: *The ABCs of Soviet Socialism.* Champaign, University of Illinois Press, 1981.
19. China retools urban economy with capitalism. *Chicago Tribune*, October 21, 1984, section 1, p 1.
20. China's new face: what Reagan will see. *Time*, April 30, 1984, p 24.

Israel: gaining momentum against smoking and cigarette advertising

CHERI M. PAPIER, MPH, MS

Cigarette smoking in Israel (population 4 million) is a problem of tremendous proportions. In 1984, a report prepared by the Israel Society for the Prevention of Smoking estimated the total annual cost of smoking to Israel's gross national product to be in excess of $388 million (or nearly 2%). This figure is based on related health-care expenditures, work absenteeism, premature death, and loss of daily productive work time. It does not take into account smoking-related fires or traffic accidents, which would increase this estimate.

PREVALENCE

The prevalence of smoking in Israel has not changed considerably during the past decade, with the exception of increases among women over 40 years of age and adolescent girls. Epstein and Biger[1] pointed out that although there are marked variations in prevalence from one survey to the next, in general children are taking up the habit at younger ages, and from age 13 there is a rapid rise in smoking rates. Adolescent smoking is much more common among those with parents, siblings, and friends who smoke. In addition, there is a close correlation between adolescent smoking behavior and other health-related behaviors, including drug use, drinking of alcoholic beverages, and unprotected sexual intercourse—all known risk factors for subsequent school drop-out. One study conducted in Jerusalem found a higher smoking prevalence among those of North African origin than those of European origin.[2] The overall prevalence rate for 18-year-olds averages about one third of the population (slightly higher among men than women—36% of men and 30% of women).

Among adults, the prevalence rate among men rises to about 50% by age 25 and remains at that level until age 50, after which it decreases (perhaps due to a survivor effect). Somewhat lower rates are observed among women.

The major intervening variable from age 18 is army service. Men generally serve for three years, whereas women serve for two years. Although many youths begin smoking before their army service begins, there is no doubt that the army provides a socializing influence conducive to smoking. Studies of adults also revealed higher rates of smoking among persons of North African descent compared to European origin. Approximately 20% of men smoke more than a pack per day, compared to 6% to 7% of women.

Surveys conducted by the Israel Institute for Applied Social Research[3] of Jewish adults over age 20 (not including kibbutz members), show that since 1970 little change in smoking patterns has occurred. In the United States, however, there has been a greater than 25% decline in smoking during the period 1963–1980. Both countries have experienced a parallel decline in mortality from coronary heart disease, which in Israel does not appear to have resulted from declining rates of smoking. Unfortunately, there are no good statistics available in Israel for morbidity or mortality from smoking-related diseases.

Regarding the smoking patterns of Israeli physicians, Yaron and Lerer[4] reported that 27% of doctors smoked in 1971, a lower figure than that for the general population at that time. By 1980, this figure was 16%.[5] A study by Luz[6] on smoking among nursing students showed that about one third of the students in each class were smokers in 1984.

MANUFACTURING

The Dubek Company has had a monopoly on cigarette production and tobacco processing in Israel since 1971. More than 20 million cigarettes are manufactured daily at Dubek's five plants. Despite rising cigarette sales at home and abroad, Dubek's monopoly status of Israeli-made cigarettes brings with it governmental restrictions. A three-month price freeze instituted in 1984 to aid Israel's sagging economy resulted in Dubek's temporary halting of production due to their refusal to operate at a loss. The government soon agreed to allow the company a 10% price hike despite the ongoing price freeze. The threat to cease production led to soaring cigarette sales.

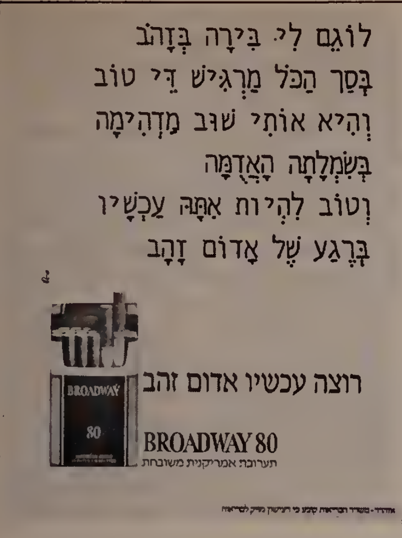
Since 1980 the Dubek Company has been diversifying. The company has acquired 75% of Bar-Kat Food Industries, Ltd and 80% of Technological Systems Ltd (for marketing microcomputers and software). Dubek has also adopted a leading Israeli soccer team and paid $110,000 for two star players. Such activities, plus a large-scale advertising campaign, suggest that Dubek's financial difficulties are not as bad as the company implies.

The price of cigarettes varies by brand, although during the price freeze, prices of Israeli-made brands ranged from about $0.13 for a pack of 20 to about $0.60 for the more "expensive" brands. Imported brands cost about $1.94. These prices include the 15% VAT (value added tax), and imported brands include a 9% customs tax (recently reduced from 12%). The government receives 60% in taxes (recently raised from 50%) on every pack sold, amounting to an estimated $76 million per year (estimate provided by the Israeli Treasury Ministry, based on the 50% tax rate). There is little mystery, then, why the government was so anxious for Dubek to resume production of cigarettes. As in other countries, shortsightedness prevents legislators and politicians from recognizing the hidden costs of smoking to the economy.

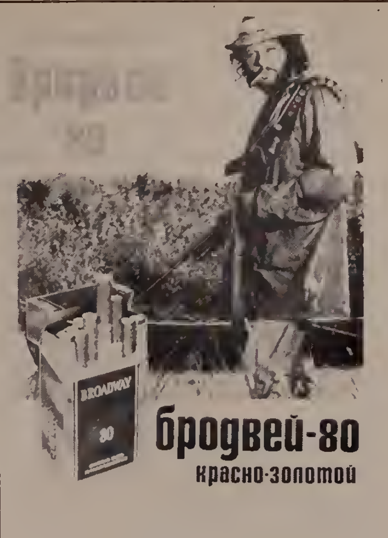
With the exception of the three-month price freeze,

Ms Papier is a member of the Executive Board of the Israel Society for the Prevention of Smoking, a member of the Council on Smoking Prevention of the US Association for Public Health Policy, and the author of *"Be A Quitter! Stop Smoking the Way that Works Best for* You," (Wilton, CT, Guide Books, 1984).

Address correspondence to Ms Papier, Department of Preventive and Social Medicine, Sackler School of Medicine, Tel Aviv University, Tel Aviv 69978, Israel.


לוֹגֵם לִי· בִּירָה בְּזָהֹב
בְּסַךְ הַכֹּל מַרְגִּישׁ דֵּי טוֹב
וְהִיא אוֹתִי שׁוּב מַדְהִימָה
בְּשִׂמְלָתָהּ הָאֲדֻמָּה
וְטוֹב לִהְיוֹת אִתָּהּ עַכְשָׁיו
בְּרֶגַע שֶׁל אָדוֹם זָהָב
רוצה עכשיו אדום זהב
BROADWAY 80
תערובת אמריקנית משובחת

Left, this advertisement from the front page of a leading Hebrew weekend newspaper would appear to be aimed at minors, despite the legal restrictions against such advertising. The extra-large typeface, printed with vowel dots, tends to catch the eye of children because this is the way Hebrew is printed in their grade-school primers. The translation is "Pour me golden beer. All in all I feel very well and Broadway again impresses me with her red dress. Good to be with her again. In this moment of red gold. I want red gold right now. Broadway 80. Quality American Blend." In smaller type: " Warning—The Ministry of Health has established that smoking is harmful to health."
Right, Advertisement in Russian aimed at Russian immigrants from an Israeli magazine. As legal restrictions prohibit depicting men in uniform, this advertisement depicts a hunter with his rifle. The translation is "I smoked all kinds of cigarettes—then I opened Broadway 80."



prices of basic commodities, including cigarettes, generally increase monthly. This translates into a cost-of-living index which is a measure of inflation. But unique to the Israeli system is the subsequent cost-of-living increment added automatically to monthly salaries. This leads to a situation of spiralling inflation, where purchasing power remains linked to costs. Thus, although cigarette prices continue to rise at regular intervals, those who smoke are able to purchase cigarettes at no greater financial burden, due to their automatic salary increments.

LAWS

All cigarette packs and advertisements must contain the following warning: "*Warning*—The Ministry of Health has established that smoking is harmful to health." This warning is generally located on the side of the packs, in small letters underneath the name and address of the company's main office, so that it looks as if it is part of the address. No mention of tar or nicotine levels is required.

A new law came into being in July 1983 restricting the advertising of tobacco products. Although the draft of the law called for a total ban on advertising, the law as passed by the Knesset (Israeli parliament) was a compromise. It prohibits oral, written, or printed advertising in praise of smoking. It also prohibits advertising on radio, television, Israeli broadcasts and public screenings, in newspapers or other printed materials designated primarily for youths up to age 18, and inside public transportation. Public "sign-boards" may be used for advertising if they are authorized and if they include the health warnings, visible from a reasonable distance. Sign-boards, it should be noted, are not the same as billboards. Unlike the United States, no billboard advertising of smoking is permitted in Israel, only sign-boards at places of business where cigarettes are sold. Advertisements may not be placed in other public areas such as stadiums.

Newspaper or other printed advertisements may not include the name, nickname, picture, or likeness of well-known persons, persons under age 40, or those wearing uniforms, sports-related clothing, or bathing costumes. The same brand may not be advertised twice in one issue

"It suits me."

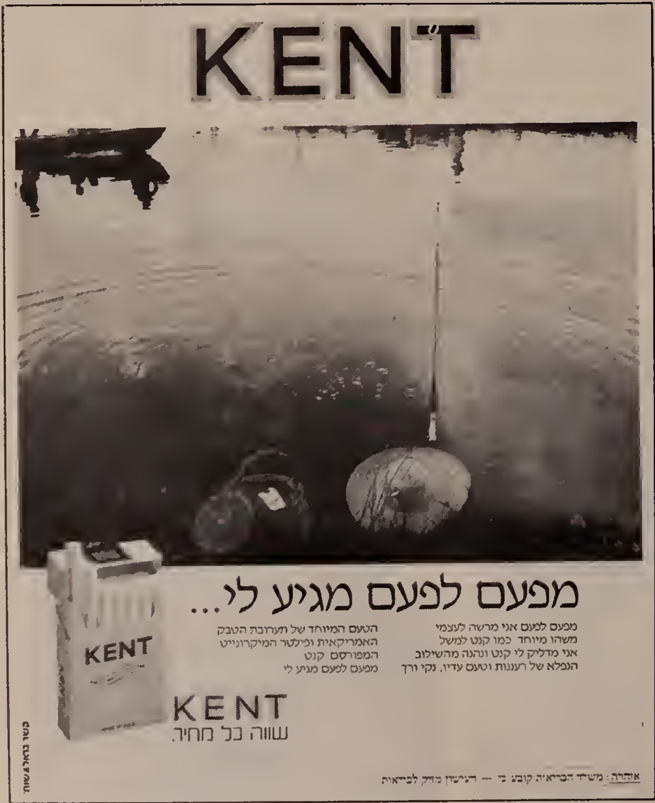
"I want red-gold now."

of a newspaper or magazine. Tobacco products may not be distributed free of charge, and they may not be associated with lotteries, prizes, or contests. Fines levied against violators are currently 150,000 IS (Israeli shekels) for individuals and 400,000 IS for corporations, with the Minister of Health responsible for implementing this law. Although these fines are increased periodically according to the cost of living index, their dollar equivalent now is much less than when the law first went into effect, due to devaluation of the Israeli currency. When translated into dollars, the original fines were about $1,500 for individuals and about $4,000 for corporations. In present-day terms (less than two years since passage of the law), the equivalent fine in dollars is $140 for individuals and $380 for corporations. No regulations are in force with regard to the sale of cigarettes to minors, and enforcement of the law restricting advertising is virtually nonexistent.

In February 1984 a new law against smoking in public places went into effect. Smoking is prohibited in public auditoriums, hospitals, clinics, public areas of pharmacies, reading areas of public libraries, all educational institutions, buses, taxis, and passenger elevators. While "no-smoking" signs are required to be placed by the management of all the above (except for elevators in residential buildings), some areas where smoking is permitted may also be designated. These areas may include, for example, corridors and lobbies of movie houses, theaters, concert halls, opera houses or lecture halls; special areas set aside by the management of public libraries; and residential school dormitories. Fines levied for disobeyance of this law are now 25,000 IS for individuals and 50,000 IS for management of the above-mentioned institutions for improperly displayed signs (approximately $25 and $50 at present-day exchange rates).

A major victory for the growing anti-smoking movement, the law has been beneficial in making people aware of the problems and hazards of smoking in public places, but it has not been effective in terms of compliance. A great deal of publicity was given to the law around the time of its passage. Non-smokers are now more inclined to request those who smoke to refrain since the law is technically behind them. Those who smoke are beginning to acquiesce, although reluctantly, at least in some of the public areas listed (particularly in public buses, where there are no longer any "smoking permitted" areas). Dubek responded to the law with advertisements in the newspapers suggesting that smokers' rights were being violated and by lobbying against any further additions to the list of public places where smoking is prohibited.

The law does not pertain to government buildings (although a recent regulation forbids government employees from smoking if they have contact with the public), banks, supermarkets, restaurants and places where food is handled, workplaces, airplanes, or trains; and it is open to much misunderstanding and misinterpretation. It might be compared to other "incomplete" laws that the Israeli Knesset has passed in regard to health and safety: for example, the law designating the wearing of automobile safety belts in intercity travel only, but not in urban areas where the majority of traffic accidents occur. As in the

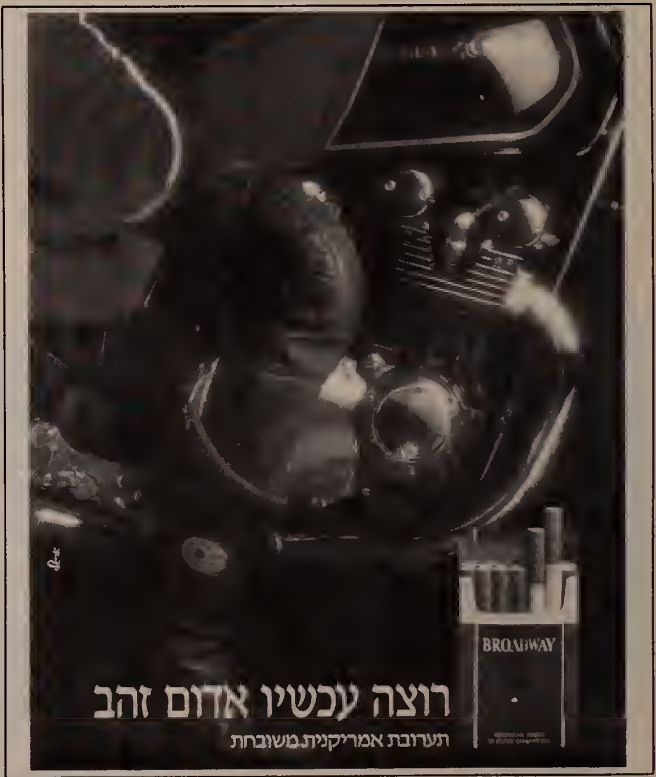


מפעם לפעם אני מרשה לעצמי
משהו מיוחד כמו קנט למשל
אני מדליק לי קנט ונהנה מהשילוב
הנפלא של רעננות וטעם עדין, נקי ורך

הטעם המיוחד של תערובת הטבק
האמריקאית ופילטר המיקרונייט
המפורסם קנט
מפעם לפעם מגיע לי

אזהרה: משרד הבריאות קובע כי — העישון מזיק לבריאות

"From time to time I deserve it. From time to time I allow myself something good like Kent. I light up and enjoy the marvelous combination—the enjoyment of freshness, delicate taste, clean and pure. The special taste of the American tobacco mixture and famous Micronite Filter. Kent. From time to time I deserve it. It's worth any price."



It is hard to tell whether this leg belongs to a person over the age of 40 or not. However, this advertisement is directed towards young men with its masculine images of motorcycle and rider—dressed in the red-gold colors of Broadway cigarettes. It is also questionable whether this advertisement goes against the legal admonition of not showing persons in sports uniforms.

case of the law restricting advertising of tobacco products, the Ministry of Health is responsible for execution of this law. As opposed to the lack of enforcement of the law restricting advertising, however, this law has been enforced to some extent. The Israel Society for the Prevention of Smoking, founded in 1972, is a consumer group active in promotion of the rights of non-smokers as well as other educational and legislative anti-smoking efforts, which has been pushing for enforcement of the law. The government, although reluctant to act, does so in some situations.

RELIGION

Leading authorities on Halacha (the body of Jewish law) including American Rabbi Moshe Feinstein, have come out strongly against smoking. Doing something that endangers life, such as smoking, goes against basic Jewish laws and principles. That is the interpretation given to Deuteronomy 4:15: "Take ye therefore good heed unto yourselves." Life is precious, a gift from God, to be protected and nurtured. Another law prohibits placing one's life in potential danger (Yore Dea, 116), emphasizing that drinking uncovered water or eating potentially dangerous foods should be avoided. Certainly smoking cigarettes fits these injunctions. Observant Jews do refrain from smoking on the Sabbath for about 26 hours. One tragic irony is that smoking has been encouraged to some extent among observant Jews by the common practice of giving the 13-year-old "Bar Mitzvah boy" his first pack of cigarettes—a symbol of having attained manhood.

THE FUTURE

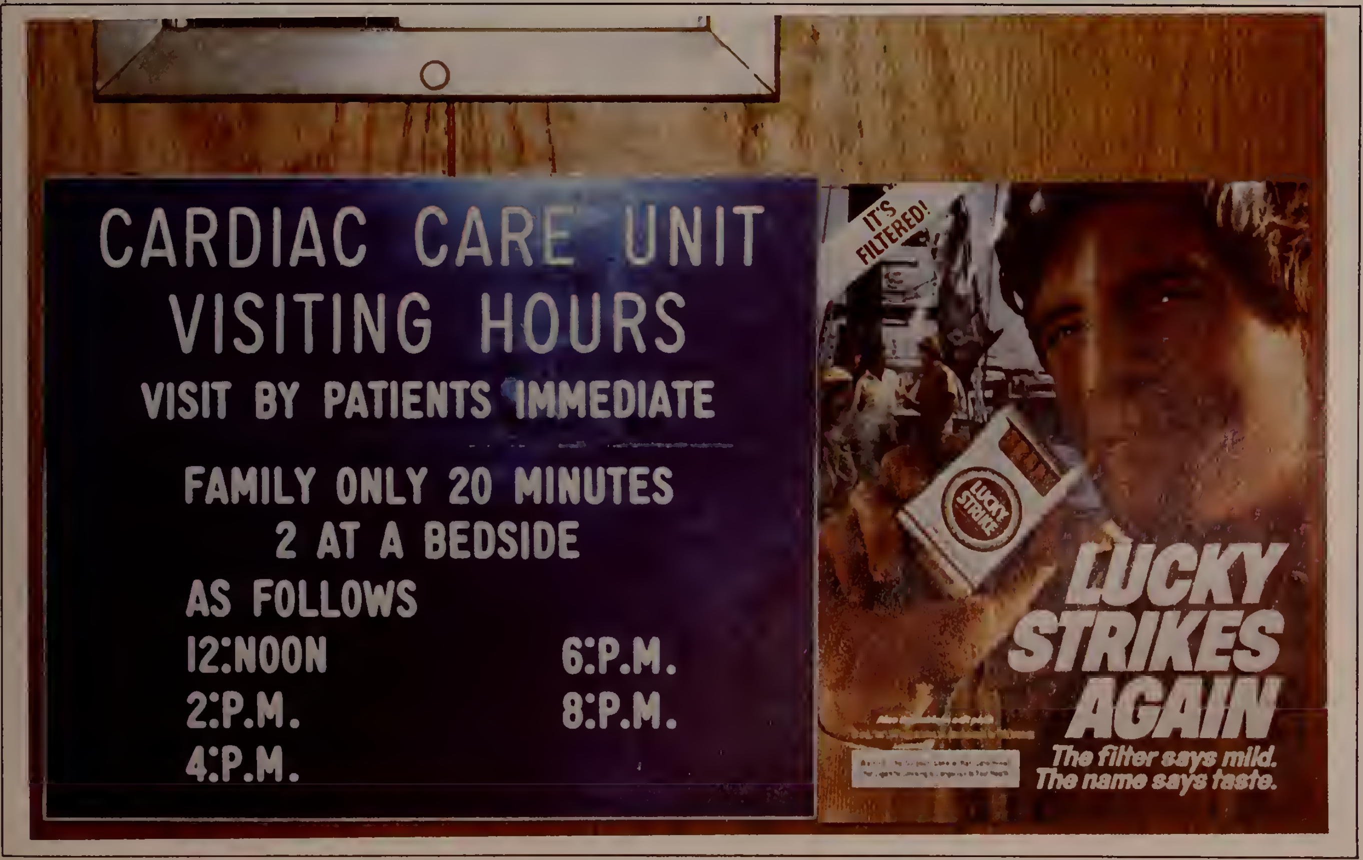
In 1984, Israel's anti-smoking movement received a boost with the visit of United States Surgeon General Everett Koop. Dr Koop's well-known and respected anti-smoking stand and successes in the United States presented a challenge to the various organizations and individuals working on the different aspects of the problem in Israel. His enthusiasm and support sparked a desire to work together on a comprehensive program.

The Ministry of Health, under the direction of Health Minister Mordechai Gur, has recently appointed a central steering committee composed of representatives of the various organizations. Their initial operational plan places great emphasis on educational efforts among children and youths and on large-scale mass communications programs for increasing awareness of smoking-related issues among the general population.

Acknowledgments. The following people involved in Israel's anti-smoking campaign provided information for this article: attorney Amos Hausner, Dr Tuvia Lerer, Dr John Goldsmith, Dr Jeremy Kark, Mrs Ada Kimhe, Dr Amiram Olenik, Mrs Aviva Karev, Dr Jerry Westin, and Dr Leon Epstein. Thanks also to Mr Yoram Gabai from the Israel Treasury Ministry for Economic Information. Ms Shoni Hanft and Ms Lisa Geiger provided typing and technical assistance.

REFERENCES

1. Epstein L, Biger C: Cigarette smoking in Israel: prevalence and control. Paper presented to the meeting on Primary Prevention of Heart Disease, Jerusalem, October, 1984.
2. Halfon ST, Kark JD, Bonar M, Friedlander Y, Eisenberg S: Smoking, lipids and lipoproteins in Jersusalem 17 year olds. *Isr J Med Sci* 1982; 18:1150-1157.
3. Ben Sira Z: *Smoking Among Adults and Youths.* Israel Institute for Applied Social Research, Jerusalem, 1983.
4. Yaron D, Lerer T: A study of smoking habits of physicians in Israel. *Harefuah* 1971; 80:150-151.
5. Lerer T: Smoking habits among Israeli physicians. *Dape Meda le Rofe* 1980:271-272.
6. Luz N: *Health-Related Behavior of Nursing Students.* MPH thesis, School of Public Health and Community Medicine, Jerusalem, 1984.



"L.S.M.F.T.C.C.U." (Lord Save Me From The Coronary Care Unit). Photograph by vascular surgeon George Gitlitz, MD, 33 Mitchell Ave, Binghamton, NY 13903.

Cigarette smoking in Papua New Guinea

A model for keeping a people down

EUAN M. SCRIMGEOUR, MB, BS

Papua New Guinea (PNG) is emerging as one of the most vigorous and influential countries in the South Pacific region. This independent nation of 3.1 million Melanesians was administered until 1976 by Australia. Until the advent of colonization, which began with the settlement of New Britain by Germany in 1880, the culture of PNG was essentially neolithic. It is understandable that the Papua New Guineans were strongly influenced by the European colonists and later administrators and advisors, and that they adopted many aspects of their lifestyle, including cigarette smoking.

Tobacco was already extensively cultivated in PNG when the first European colonists arrived. It was probably introduced in the early 17th century from Malaysia by way of Java and the Philippines, following its earlier introduction to Malaysia from South America.[1] The colonists brought twist tobacco with them, which soon became an important trading item, and inevitably the consumption of tobacco increased.[2]

The climate of PNG, especially in the highlands, is eminently suitable for growing tobacco, and as most people are subsistence farmers, nearly everyone has access to home-grown, air-cured tobacco. Until about 1960, most commercially available tobacco was also cured naturally by enzymatic fermentation and was marketed as "twist and coarse-cut" tobacco, which was smoked in pipes or rolled into cigarettes or cigars using dried leaves or newsprint. The latter practice was so widespread that the lone national English newspaper was once said to be "the most widely smoked newspaper in the Southern hemisphere."[3]

Since 1960 there has been a rapid increase in the demand for flue-cured cigarettes, which has encouraged various international tobacco companies to extend their operations in the country.[4] The annual consumption of commercial cigarettes first exceeded the consumption of twist and coarse-cut tobacco in 1969.[3] Between 1960 and 1979 the total annual tobacco consumption per head increased by 121%, and by 1979, 71% of tobacco consumed was in the form of commercial cigarettes.[4,5] Cambridge, produced by Rothmans, was the most popular brand, accounting for some 74% of cigarette sales.[4] This company also markets Dunhill, Winfield, and Peter Stuyvesant, while W. D. & H. O. Wills (connected to BAT industries, formerly known as British American Tobacco), the other major international company operating in PNG, produces Benson & Hedges and Kool.[4]

Most of the tobacco required for cigarette manufacture has been imported, and domestic tobacco production has failed to keep pace with the demand. But with the expansion of tobacco farming in the highlands it was expected that 50% self-sufficiency would be achieved by 1982.[4] The development of these farms deprives PNG of some of its most valuable food-producing land.

Surveys conducted in 1968[6] and 1981 (E. M. Scrimgeour, Annual Symposium, Medical Society of Papua New Guinea, 1982) have shown that the patterns of smoking and betel nut chewing have changed in recent years (Table). In the highlands, where home-grown tobacco is freely available and the impact of modern society and advertising is less evident, most people continue to smoke their own tobacco, but there is an increasing trend to commercial cigarettes. On the coast of Papua smoking used to be largely restricted to men, but now more women are smoking, and, especially in urban areas, as a rule only commercial cigarettes are smoked by both men and women.[3,4] In highland and coastal regions there is a trend among both sexes to start smoking at an earlier age.

Changing Tobacco Smoking and Betel Nut Chewing Habits in Papua New Guinea.

	1968		1981	
	Men %	Women %	Men %	Women %
New Guinea highlands*				
Tobacco smokers	74.5	78.3	77	80
Betel nut chewers	46	1.9	26.7	1
Papuan south coast				
Tobacco smokers	76.5	33.8	85	76
Betel nut chewers	79	83.8	71.4	66.7

* The 1968 survey was conducted in Mount Hagen[8] and the 1981 survey in Goroka.

This table is reproduced by permission from the *Br Med J* (1983; 86:1414–1416).

The reduction in betel nut chewing in all areas of PNG in recent years (Table) is probably related to the increase in cigarette smoking. The betel nut is produced by the areca palm (*Areca catechu* L), which grows best on the coast and is chewed with betel pepper (*Piper betel*) and slaked lime to produce a feeling of relaxation and mild euphoria. Betel nut chewing is a widespread custom in PNG, has important social connotations,[7] and does not appear to have any significant adverse effects on health. There is no evidence of an intrinsic oncogenic effect.[5,7] However, it is easier to smoke a cigarette than to chew a betel nut and, among educated young Papua New Guineans, especially in urban areas, betel nut chewing is declining in popularity. Government employees are not permitted to chew betel nut during working hours.

Energetic advertising encourages cigarette smoking in

Dr Scrimgeour was formerly a senior lecturer in the Department of Medicine at the University of Papua New Guinea and the editor of the *Papua New Guinea Medical Journal*.

Address correspondence to Dr Scrimgeour, c/o Cowan, 738 Doncaster Rd, Doncaster, Victoria 3108 Australia.

PNG. This follows the usual strategies, with billboard advertisements in public places depicting happy, healthy, successful young men and women enjoying the good life and puffing on cigarettes. The models for these advertisements are often white and may be engaged in activities unfamiliar to the average Papua New Guinean—for example, rounding up cattle on a ranch. Sporting activities are prominently supported by the tobacco companies. In 1982, at the time of the Commonwealth Games (and less than two years prior to the Olympic Games), several new brands of cigarettes were introduced which had in common the motif of a victorious athlete.

At present, health warnings are not required on cigarette packets or advertisements in PNG (Fig). In view of the many adverse effects on health caused by smoking and the significant association between tobacco consumption and the development of cancer of the oral cavity, esophagus, larynx, lung, bladder, kidney, and to some extent the pancreas,[8] it would seem to be the responsibility of the tobacco producers to warn PNG consumers of the potential hazards of cigarette smoking.

As yet, the only significant increase in the incidence of tobacco-associated neoplasms in PNG has been confined to cancer of the oral cavity in women.[5] This may reflect the marked rise in cigarette smoking among female coastal Papuans who habitually chew betel nut.[5] The incidence of lung cancer has not risen yet but may do so in the future if cigarette smoking by increasingly affluent young Papua New Guineans is unrestricted. For many years the medical profession in PNG has been urging that cigarette advertising be banned, and that health warnings should be displayed on cigarette packets.[3,4] There is now growing evidence of government support for these measures.

REFERENCES

1. Ryan P: *Encyclopaedia of Papua New Guinea.* Melbourne, Melbourne University Press, 1972, pp 224, 734.

2. Parsons NJ: Tobacco. Agriculture in the community: A series of review papers. Port Moresby, Dept of Primary Industry, 1977. (mimeograph)

3. Wigley SC: Lung cancer and the Melanesian; an impending problem? *Papua New Guinea Med J* 1974; 17:296–303.

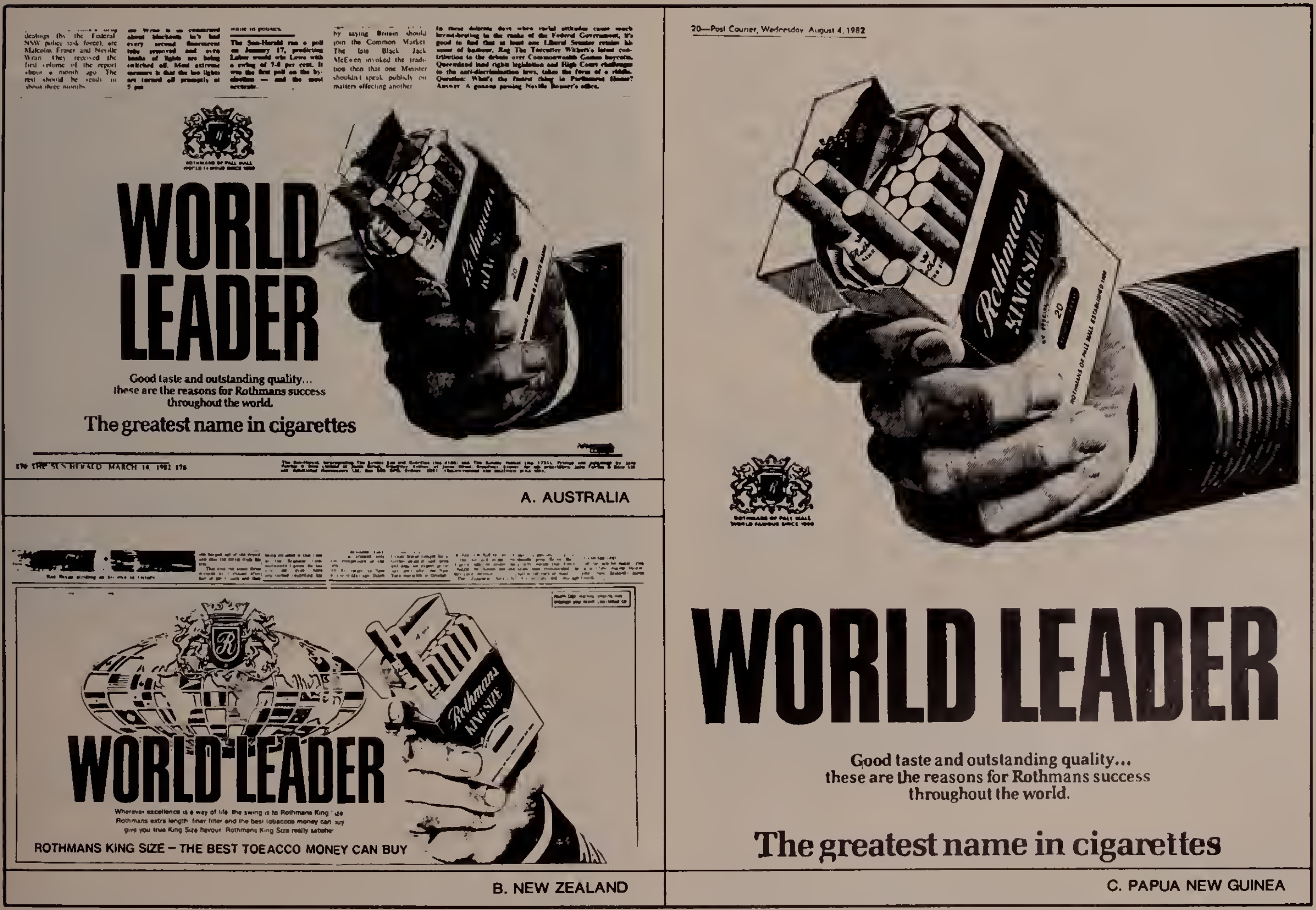
4. Brott K: Tobacco smoking in Papua New Guinea. *Papua New Guinea Med J* 1981; 24:229–236.

5. Scrimgeour EM, Jolley D: Trends in tobacco consumption and incidences of associated neoplasms in Papua New Guinea. *Br Med J* 1983; 286:1414–1416.

6. Burton-Bradley B: Arecaidinism: betel chewing in transcultural perspective. *Can J Psychiatry* 1979; 24:481–488.

7. Pindborg JJ, Barmes D, Roed-Petersen B: Epidemiology and histology of oral leukoplakia and leukoedema among Papuans and New Guineans. *Cancer* 1968; 22:379–384.

8. US Department of Health and Human Services: The health consequences of smoking. The changing cigarette. A report to the Surgeon General. Washington, DC, Public Health Service, 1981.


WORLD LEADER
Good taste and outstanding quality... these are the reasons for Rothmans success throughout the world.
The greatest name in cigarettes
WORLD LEADER
ROTHMANS KING SIZE – THE BEST TOBACCO MONEY CAN BUY
20—Post Courier, Wednesday August 4, 1982
WORLD LEADER
Good taste and outstanding quality... these are the reasons for Rothmans success throughout the world.
The greatest name in cigarettes

A. AUSTRALIA

B. NEW ZEALAND

C. PAPUA NEW GUINEA

Three South Pacific nations take different approaches to cautioning their citizens about the hazards of Rothmans King Size cigarettes. Can you find the warning label in each advertisement? (Answers below)

ANSWERS: A. At the bottom of the pack: "WARNING—SMOKING IS A HEALTH HAZARD." B. Box at upper right corner: "Health Dept. warning: smoking may endanger your health. Low/middle tar." C. No warning.

Ending the social acceptability of smoking and tobacco advertising in New Zealand

DEIRDRE KENT, BSc

As in most Western countries, cigarette smoking in New Zealand (population 3.2 million) is the greatest preventable cause of death and disease, causing 3,600 deaths annually or 15% of all deaths.[1]

Smoking prevalence in New Zealand was monitored in 1976 and 1981 by a question in the population census. In those five years, the prevalence dropped from 36% to 32%.[2] All age groups except women between 15 and 24 years showed a decline in smoking. More teenagers aged 15 to 19 years smoked in 1981 than in 1976, an equivalent new market of 40 new smokers a day. Political pressure from the tobacco industry may prevent the smoking question from being included in the 1986 census.

The Maori population of 280,000 comprises the largest ethnic group. Seventy percent of Maori women between 20 and 24 years of age smoke; 55% of Maori girls between 15 and 19 years smoke.[2] In contrast, only 24% of all other Pacific Island women smoke. The standardized death rate from lung cancer of Maori women (1976–1980) was 44 per 100,000 population, compared with 12 per 100,000 for all women.[3] The Maori Women's Welfare League conducted its own study and found that urbanization and the subsequent loss of spiritual identification with the tribal land have been major factors.[4] Urban Maori women perceive themselves as less healthy than their rural sisters. An organization called MASH (Maori Action on Smoking and Health) was launched in 1984.

BLACK PACK ATTACK

Only two companies manufacture cigarettes in New Zealand. Rothmans (namesake of the South African cigarette giant, with connections to Philip Morris, New York) has over 70% of the market and W D & H O Wills (the New Zealand cousin of BAT—British American Tobacco, parent of Brown and Williamson Tobacco Company, Louisville) has the rest. Best selling brands are Winfield, Rothmans, Sportsman, Pall Mall (all made by Rothmans), and Benson and Hedges (BAT). Advertisements for a new brand, John Brandon Black (Rothmans), identify the product with rugby, the national sport (Fig 1). The All Blacks, the national rugby team, are known for their powerful scrums or packs. John Brandon Black is advertised as the "Black Pack" on the sports pages of newspapers.

Approximately 3,700 people are employed growing, manufacturing, or distributing cigarettes in New Zealand. The government gave incentives four years ago for tobacco growers to change to other crops, but this was reversed recently, and planting is now on the increase. During the 1970s the conservative government helped establish a cigarette factory in Samoa, and New Zealand is a major exporter of cigarettes to other islands in the South Pacific.

Advertisements for tobacco products were withdrawn from radio and television in 1963, and in 1973 the first voluntary agreement between the Minister of Health and the tobacco industry brought about the banning of cigarette advertising on billboards and in cinemas. In addition, the size of print media advertising was restricted to half a newspaper page. These parts of the voluntary agreement are well enforced.

Efforts to restrict the creative content of advertisements have not been effective, however. In 1984, a complaint was lodged with the Committee on Advertising Practice (the self-regulatory body of industry) against an advertisement for Drum tobacco—used in hand-rolled cigarettes—on the grounds that the young woman depicted getting the "satisfaction of doing it yourself" appeared to be under 25 years, in violation of the voluntary agreement (Fig 2). The company stated in reply that she was 27, and no appeal was possible. A complaint about the slogan "Wherever excellence is a way of life the swing is to Rothmans King Size" on the grounds of inaccuracy (smoking is more prevalent among lower socioeconomic groups) met

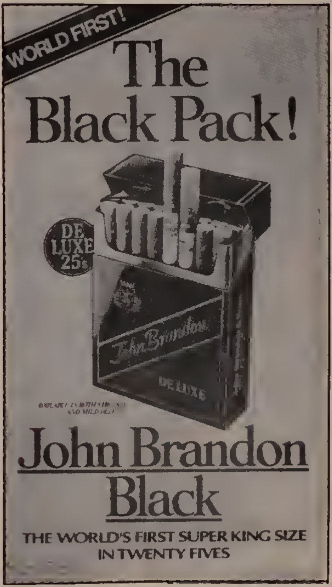


FIGURE 1. Preying on youthful sports enthusiasts; playing on the name of the All Blacks, New Zealand's national rugby team.

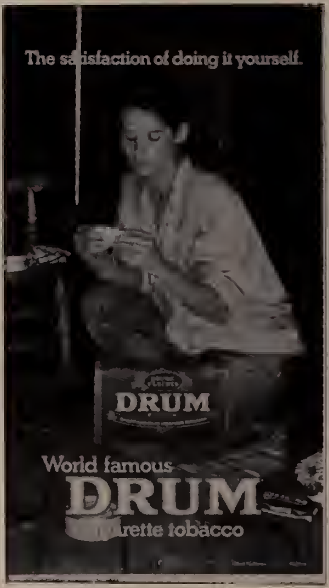


FIGURE 2. World's youngest-looking 25-year-old?

Address correspondence to Ms Kent, Research and Education Officer, Action on Smoking and Health (ASH), PO Box 8667, Symonds Street, Auckland, New Zealand.

Ms Kent edited *Smoking Control in New Zealand*, a comprehensive resource manual published in 1984. Copies are available for $5 (plus postage) from ASH.

with the reply from the Committee on Advertising Practice that "The word 'excellence' is related to the qualities of the product and not to any lifestyle."

Cigarette advertising has crept back onto television through sports events sponsored by cigarette companies, including cricket, tennis, motor racing, and horse racing. In January 1984 New Zealanders could view over 100 hours of Rothmans International Cricket on television, with a dominating Rothmans backdrop. In poor economic times, cultural and charitable organizations fearful of decreased donations increasingly seek tobacco money. The Foundation for the Blind and the Outward Bound movement both made pleas to Parliament opposing a ban on cigarette advertising.

AIRBORNE ADVERTISING

One of the most insidious new developments has been the introduction of the Winfield Regional Rescue Helicopter (Fig 3). Winfield is New Zealand's leading cigarette brand and is marketed by Rothmans. In October 1983 the Governor General (the Queen's representative in New Zealand) launched the helicopter in Auckland's main public square. A team of young surf lifesavers, wearing caps, shirts, and slacks in the red and white Winfield colors and the cigarette brand logo over the vest pocket and on the cap, paraded beside a helicopter painted in Winfield colors with the cigarette logo. A small group of protesters from MOP-UP (Movement Opposed to the Promotion of Unhealthy Products) carried placards reading "Kids need Winfield like a growth on the lung" and "Rothmans gives help like a snake gives venom." One protester sat in a wheelchair to symbolize the disabling effects of tobacco.

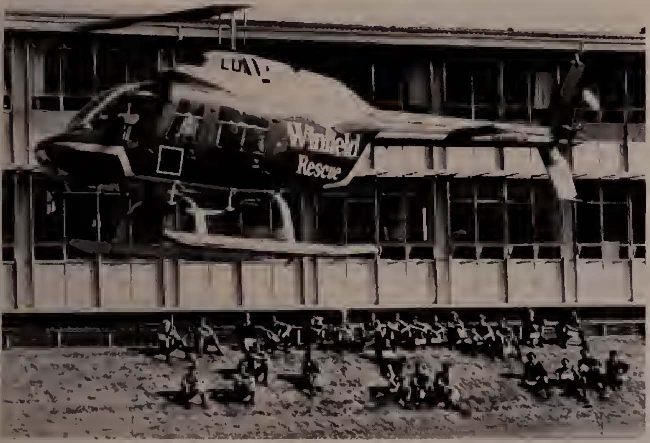


FIGURE 3. Cigarette pack as hero: Rothmans attempts to win over the public with flying colors. The Winfield Regional Rescue Helicopter is shown landing at Mt Albert Grammar School, Auckland, to teach children about lifesaving. (Photograph reprinted by permission of *The Auckland Star*)

The rescue helicopter flies over Auckland beaches, islands, and yachts, providing a full-time air ambulance service for the region; it lands in the park beside Auckland Hospital. Headlines such as "Cigarette copter proves its worth" appear regularly. The helicopter is taken to schools to teach adolescents about surf lifesaving.

One of the persons behind this venture is advertising executive Malcolm Beattie, who also heads the subcommittee of the Auckland 1990 Commonwealth Games Committee, which is responsible for arranging sponsorships. Action on Smoking and Health (ASH), founded in 1980 with the aim of reducing smoking, is working hard to ensure that the games are not used as a vehicle for cigarette advertising, as they were in 1982 in Brisbane, Australia, where victorious athletes were interviewed in the Rothmans-sponsored media center in front of a curtain emblazoned with Rothmans' insignia. Three representatives of ASH have visited the mayor of Auckland and have asked to send a delegation to the games' organizing committee. In December 1984 it was announced that there will be no tobacco company sponsorship of the 1990 Commonwealth Games. ASH will continue to press for an end to tobacco company involvement in sports.

Smoking control efforts in New Zealand have been led by the National Heart Foundation and its medical director, Dr David Hay; an epidemiologist, Dr Robert Beaglehole of the University of Auckland; several Health Department officials during the 1960s and 1970s; the New Zealand Cancer Society; and the Government Advisory Committee on Smoking and Health (founded in 1976). The momentum has increased dramatically in the 1980s. Dr Beaglehole established ASH in 1980; in 1982 ASH employed its first part-time worker, and by 1984 it had a full-time worker, two part-time workers, and four regional branches. Funding comes from the National Heart Foundation, the New Zealand Cancer Society, and the New Zealand Asthma Foundation. The patron is Sir Edmund Hillary, the first man to ascend Mount Everest. Each branch of ASH has a mix of conservatives, environmentalists, and doctors. Many campaigners have had experience with local and national issues in the New Zealand Values Party, which thrived in the 1970s by emphasizing social and environmental issues.

SOUTH AFRICAN CONNECTION

The major shareholding of Rothmans by South African interests is providing another source of public disapproval of the cigarette industry. At Rothmans' annual meeting in November 1984 company executives were repeatedly challenged by a group of 46 opponents of apartheid (who had purchased the minimum shareholding of 200 shares) to explain the company's connection with South Africa.

During the last 20 years little effort has been devoted to public information and education programs, except by voluntary agencies. During 1983 the government spent only $50,000 on such programs—compared with $2 million a year to be spent by Western Australia, a state with one-third of New Zealand's population. One possible source of such funding would be cigarette taxes, which currently account for 58% of the price of a pack of cigarettes.

In July 1984 the Labour Party came to power after nine years of pro-industry National Party rule. Very little on smoking has been discussed at Labour Party conferences, but the new Health Minister has increased spending on television advertisements to stop children from smoking. No law exists to ban the sale of cigarettes to children; the old law was repealed in 1981 on the grounds that, since the advent of vending machines, the ban was unenforceable.

A petition to ban the advertising of tobacco products is currently before Parliament. It was presented by a private citizen, Cliff Turner. Its existence provided the excuse for the former Minister of Health to defer negotiating the voluntary agreement during the election year. ASH has presented a submission to Parliament's Select Committee on Health and Welfare in support of this petition. The Tobacco Institute has argued that a “medical controversy” still exists. Another petition to ban smoking in public places was submitted to the Select Committee by a group of citizens with asthma.

It is now clear that a great deal of education of politicians is indicated, and that the medical profession will have to participate fully in this process. New Zealand has led the way in many social changes: it was the first country to allow women to vote and the first western country with a welfare state. New Zealand could well provide the model for ending the single most important preventable cause of illness—cigarette smoking.

REFERENCES

1. King DR, Smith AH, Salter DM: Mortality attributable to smoking in New Zealand. *NZ Med J* 1983; 96:195-199.
2. Hay DR, Foster FH: Intercensal trends in cigarette smoking in New Zealand: age, sex, and ethnic status. *NZ Med J* 1984; 97:283-285.
3. *Cancer Data: New Registrations and Deaths, 1980.* New Zealand, National Health Statistics Centre, Department of Health, 1984.
4. Murchie E: *Rapuora—Health and Maori Women.* Wellington, Maori Women's Welfare League, New Zealand Dept of Health, 1984.

IN CHINA, CIGARETTES ARE BIG BUSINESS

China (population one billion) is the world's largest producer and consumer of tobacco products; one trillion cigarettes a year are manufactured in 80 factories (*Tobacco Reporter* 1985; 112(3):46-50).

The annual increase in cigarette consumption has remained at 10% in recent years. Approximately 300 million Chinese smoke regularly, and smoking is almost exclusively a male habit; perhaps as few as 1% of women smoke (*Chicago Tribune* Aug 23, 1981).

Tobacco was introduced from the Philippines in the late 16th century and, until about 100 years ago was used primarily among the upper class. Smoking gradually grew in popularity, aided by Western-style advertising, until the communist revolution. The late Chairman Mao Zedong, who led China from 1949 to 1976 and whose face on billboards across China was even more ubiquitous than that of Marlboro cowboys in the United States, smoked heavily, as did many of the founders of the communist state.

As in the United States and most other countries, the Chinese government's actions toward the smoking and health issue are paradoxical. One agency continues to expand tobacco growing (China is a net exporter of tobacco, with sales to Eastern Bloc, Southeast Asian and Western European countries), while another denounces smoking. In 1982, the government took steps to modernize the cigarette industry by forming the China National Tobacco Corporation, (CNTC) which controls all phases of tobacco production and distribution. The CNTC has closed more than 300 small, inefficient factories in its effort.

The government launched its first anti-smoking campaign in 1978, consisting of warnings of the health hazards of smoking in newspapers, broadcasts, and films (*Los Angeles Times*, Aug 28, 1978:4).

In the past year, the government announced the formation of a quasi-government group, the Smoking and Health Association, to create a new anti-smoking campaign (*Tobacco Reporter* 1985; 112(2):9).

In 1982, in one of the few publications on the subject of smoking in China, two researchers in the School of Public Health at the Beijing Medical College found that smoking among high school students at eight schools in Beijing has increased among the ordinary high schools, while smoking in the “key schools,” where students are brighter and more likely to continue their studies, has remained at a negligible level (*World Smoking & Health* 1984; 9(1):15-18). In the key schools, less than 1% of the boys smoked and no girls smoked, while in the ordinary schools 19.7% of the boys smoked and only 0.4% of the girls smoked. Overall, by the last year of high school 34% of the boys surveyed said they smoked. The most common reason given by students for smoking was curiosity, but a “lack of ideals and dedication” and boredom were also cited.

60分
提倡戒烟
1980.4.7.
中国人民邮政
J.56(2-2)
1980

Chinese postage stamp

China's courtship of western technology further illustrates the contradiction between promoting health on the one hand and encouraging tobacco production on the other. RJ Reynolds Tobacco Company was one of the first foreign companies to take advantage of the special economic zone created in Xiamen, a port city about 300 miles northeast of Hong Kong. (*New York Times*, Dec 25, 1983:6F). The zone offers easier terms of land rents and wages, and local authorities are empowered to deal with foreign investors—which increases the speed of setting up a business venture. Reynolds has constructed a modern cigarette plant; one third of its initial production will be exported, one third will be sold in tourist areas in China, and one third will be sold in the domestic market. Philip Morris will have a plant commissioned before the end of 1985 in the city of Shenzhen in the Guandong province. Advertising for the American companies' brands has begun in China.

MICHAEL WROBLEWSKI

Japan: land of the rise in lung cancer

US government aids cigarette sales

The following article was written by Alan Blum, MD, *based on extensive materials supplied by Peter Evans, a teacher of English at Keio University, Tokyo.*

In no other country in the world is the two pack (or more) a day cigarette habit more prevalent among men than in Japan. Seventy percent of Japanese men admit to being regular cigarette smokers, more than double the American percentage.[1] In contrast to the trend in American business toward executive health and fitness programs, smoking is still very much a part of the Japanese businessman's lifestyle.

Notwithstanding the fact that lung cancer is the fastest growing cause of death in Japan (accounting for 5,171 deaths in 1960 and 25,647 in 1983, according to Japan's National Cancer Center), the Japanese government has shown little interest in the problem of smoking. Indeed, in 1983 the newly appointed minister of health and welfare said as he assumed office that smoking was the secret of his good health. In 1982 in the Diet (Japanese parliament) the minister of finance boasted of his heavy smoking and drinking,[2] and Prime Minister Yashuhiro Nakasone, responding to an opposition politician's objection to cigarette advertising on television, has said that cigarettes are not harmful as long as smokers adhere to the warning printed on each pack.[3] The Japanese warning reads, "For the sake of health, let's be careful not to smoke too much."

For 80 years the manufacture, distribution, marketing, and advertising of all domestic cigarettes were controlled by the Japan Tobacco and Salt Public Corporation, a government monopoly. The monopoly long protected its position (and 80,000 domestic tobacco farmers) by restricting the importation of tobacco and the distribution and advertising of foreign cigarettes.[4] Foreign brands accounted for only 1.8% of the 306 billion cigarettes sold in Japan in 1983. Under pressure from American cigarette companies, the government recently transformed the tobacco monopoly into a semi-private company, with two thirds of its shares still held by the Ministry of Finance. The new company, Japan Tobacco, Inc, made its public debut on April 1, 1985 with advertisements in daily newspapers that promised "to continue to be effectively managed and contribute to society by introducing ease and relaxation into daily life." The company offers miniature flowering tobacco plants, a "symbol of peace of mind and tranquility."

A second quasi-private corporation, Japan Tobacco International Corp, also has been formed to export cigarettes to countries in Southeast Asia, as a hedge against declining sales in Japan.

Although advertising is limited to some extent (to about $50 million a year) by mutual agreement of the tobacco companies,[5] cigarette advertising is still ubiquitous. Logos for such domestic brands as Cabin 85 and Mild Seven appear throughout the country on shopping bags, storefronts, trash cans, and trucks. Cigarettes are sold in more than 500,000 tobacco shops, coffee shops, bars, and restaurants, as well as from approximately 330,000 vending machines available around the clock. In 1973 there were 141,500 such machines (data obtained by Japan Action for Nonsmokers' Rights).

Cigarette advertising is still permitted on television. One recent advertisement featured a popular Japanese novelist conducting an orchestra using a Cabin 85 cigarette as a baton. Another in the series showed a popular young movie star sitting in a dune buggy, in violation of the monopoly's own guidelines against using youthful role models. Television advertisements for another domestic brand, Cosmos, employ the latest in computer graphics. At the end of each commercial is a statement that smoking by minors is prohibited, an admonition, observes Peter Evans, that could hardly be calculated to glamorize smoking among young people. Advertisements for Cosmos using the same images also appear in numerous magazines read by teenagers.

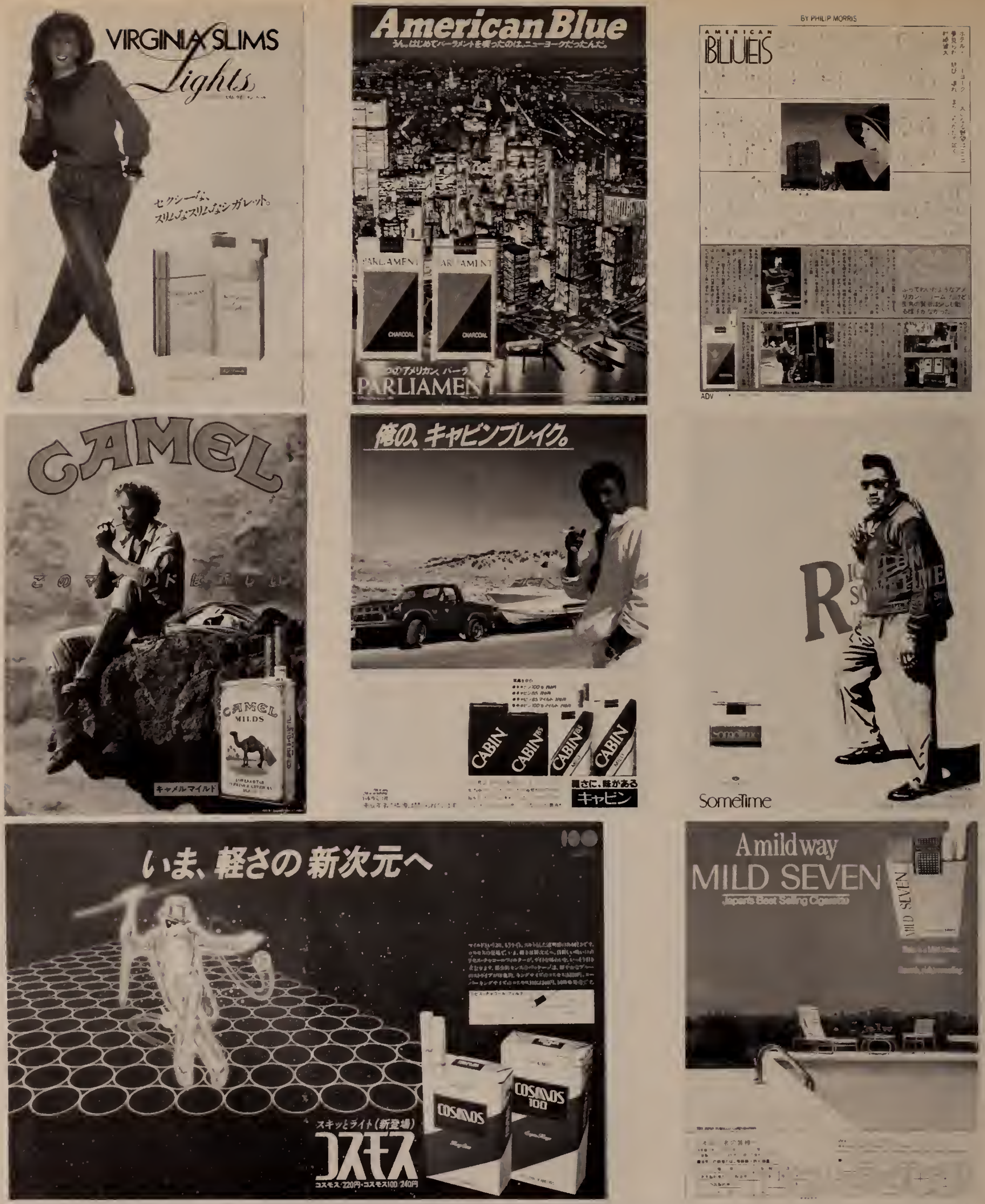
Foreign cigarette companies are stepping up their campaigns on billboards and in retail shops—advertising by Philip Morris is up by 50% in 1985.[5] The company's Lark brand (Japanese trademark rights for which were purchased from "rival" multinational tobacco company Grand Metropolitan) has become known through its advertising spokesman, American actor James Coburn, who advises viewers to "Speak Lark."

RJ Reynolds' Camel is the fastest growing brand. A new company, RJ Reynolds/Mitsubishi Corporation Tobacco Company Ltd, controls sales for Reynolds' cigarettes and merchandise bearing its cigarette brand logos.[6,7] (Mitsubishi Corporation, closely allied to the giant automobile maker that recently announced a joint venture with Chrysler in the American automobile market, has served as RJ Reynolds' import agent since 1971.) Reynolds-Mitsubishi will try to increase sales by targeting its advertisements to young smokers.

In 1984 Brown & Williamson Tobacco Corporation (BAT) announced it would link with Japan's leading confectionery company, Meiji Seika Kaisha Ltd, which is also a manufacturer of pharmaceutical products, to sell its cigarettes in 4,900 kiosks, 12,000 pinball arcades, and 175,000 confectionery and baked goods outlets.[8,9] However, vigorous protests and threatened boycotts by the Japanese Lawyers' Organization for Nonsmokers' Rights and the nearly 40 other groups belonging to the All-Japan Anti-smoking Liaison Council succeeded in delaying implementation of the marketing agreement. The lawyers' group made it clear that it would seek enforcement of laws that forbid persons under 20 years of age to smoke or buy

---

For information on the non-smokers' rights movement in Japan, address correspondence to Bungaku Watanabe, Director, Japan Action for Non-smokers' Rights (Ken'en-ken Kakuritsu o mezasu hitobito no kai), 1-7-3 Hirakawa-cho, Chiyoda-ku, Tokyo 102, Japan. For additional information on smoking, cigarette advertising, and efforts to counter smoking, address correspondence to Takashi Shiraishi, The All-Japan Anti-smoking Liaison Council, 846 Kami Kawai, Asahi-ku, Yokohama 241, Japan.



Japan's $11 billion cigarette market is double the size of the country's famed telecommunications market. Although foreign imports have accounted for only 2% of cigarettes smoked, the recent relaxation of trade barriers and advertising restrictions has given American cigarette makers hope of nearly tripling their sales by 1988. Most aggressive in its promotions is Philip Morris. Its advertising for Virginia Slims features an American model and the slogan, "Sexy, slim, slim cigarette" (top left). Parliament advertisements in magazines and on billboards feature the slogan, "Yeah. When I first smoked Parliament, it was New York" (top center). In magazine advertisements, impressionistic essays with English titles like "American Blues" and "Downtown" describe how it feels to be in New York (top right). RJ Reynolds uses its caucasian "everyman" to appeal to impressionable young Japanese (middle left). Japan Tobacco, Inc (JTI) advertisements for Cabin 85, Sometime, and Cosmos are imitative of American advertisements (middle center, middle right, bottom left). The advertisement for another JTI brand, Mild Seven, appeared in the March 1985 issue of the United Airlines flight magazine on all US flights, notwithstanding the absence of the Surgeon General's warning (bottom right).

cigarettes and that prohibit anyone from selling to a minor.

Such protests have not deterred the Reagan administration from working on behalf of tobacco interests to press the Japanese government to permit increased imports of American tobacco. The All-Japan Anti-smoking Liaison Council has pointed out the hypocrisy of America's trying to expand exports of goods that are labeled as dangerous within the United States.

Seven members of the Diet have formed a non-partisan anti-smoking group known as the Parliamentarians' Council on Smoking Problems.[10] But so deeply involved in the tobacco business is the Japanese government that it has never produced an anti-smoking poster and has not supported restrictions on smoking in public places. A request by the Education Ministry for 6 million yen ($25,000) from the 1985 budget for the development of a national anti-smoking program was rejected, as was a request by the Ministry for Health and Welfare for 7 million yen ($29,000) for courses for health officials to curb smoking.[11] Unlike most western countries, which are at least talking about laying the groundwork for a non-smoking generation, Japan gives no systematic information on the dangers of smoking at any stage in school education. The situation is just beginning to change. A handful of junior high schools have introduced anti-smoking literature into the curriculum and a workshop on anti-smoking education held in Tokyo in 1984 drew 70 teachers from across the country.[12] The chairman of the Japan Teachers' Union has also endorsed stepped-up educational efforts.

The impetus for the increasing attention to smoking has come from the All-Japan Anti-smoking Liaison Council, which helps coordinate the activities of nearly 40 organizations around the nation. An annual anti-smoking week, begun on April 7 to 13, 1984 to coincide with World Health Organization Day, earned an editorial salute from *The Japan Times*, an English-language newspaper.[13] The week features parades, exhibits, and smoking cessation clinics throughout Japan, united by graphic posters. In 1985 the council scheduled its campaign to coincide with the end of the government monopoly, and its posters carried the slogan, "The state quits, I also quit."[12]

Vigorous opposition by the council has been launched against the exporting of cigarettes to other Asian countries. A recent appeal has been made to the Japanese emperor to end the practice of giving cigarettes as gifts to guests and volunteers in the Imperial household.[14] In January 1985, the council sponsored a concert whose title satirized the tobacco monopoly's "Nice day, nice smoking" slogan: "Clean air, clean music." The event, featuring a leading Japanese composer, singers, and other musicians, raised funds for the council.[15] The council has established a hotline for complaints about smoking in the workplace, on transportation systems, and in other public places.[1]

The effort to assert the rights of non-smokers began in earnest in Japan in 1978 with the establishment of *Ken'en-ken*—a catchy phrase meaning "the right to dislike smoking"—as a national movement to ban smoking in public places. *Ken'en-ken,* headed by Bungaku Watanabe, is more formally titled Japan Action for Nonsmokers' Rights (JANR). At the Fifth World Conference on Smoking and Health in Winnipeg in 1983, Watanabe called attention to the targeting by Japanese cigarette advertisers of women and teenagers. Whereas only 8% of women aged 20–29 years smoked in 1968, 17% reported doing so in 1981. Watanabe pointed to an advertisement featuring a popular Japanese actor lighting up a cigarette after exercising with a jump rope as "fraudulent, criminal advertising."

As the result of growing public complaints and the efforts of JANR and other groups, in 1984 the Japanese National Railways added a second non-smoking car to each of its famed bullet trains. Still, two thirds of Japanese express trains lack such accommodations.

JANR has found that only two of 75 major general hospitals in Tokyo prohibit smoking and only 18 others provide smoking rooms that are separated from non-smoking areas.[1]

Doubtless because of the contributions of Watanabe, the council, and Dr Takeshi Hirayama (whose research [*Br Med J,* Jan 17, 1981] indicates that the spouses of persons who smoke also have a high risk of lung cancer), Japan has been chosen as the site of the Sixth World Conference on Smoking and Health in 1987.

REFERENCES

1. Fujimoto M: Non-smoking movement gaining strength. *Japan Times,* Jan 7, 1984.
2. Heavy-smoking minister tells Diet panel tobacco not so bad. *Japan Times,* March 26, 1982.
3. Nakasone defends smoking. *Japan Times,* Feb 21, 1984.
4. Reform of tobacco monopoly. (editorial) *Japan Times,* April 3, 1984.
5. Slavin B: Lifting the lid on cigarettes. *Newsday,* April 21, 1985.
6. RJR-Mitsubishi venture to be on 70-30% basis. *Tobacco International,* Nov 16, 1984.
7. RJR International enters joint venture with Mitsubishi. *Tobacco Reporter,* Dec 1984.
8. World news roundup. *Advertising Age,* Oct 1, 1984.
9. B&W goods to be sold in Japanese pinball parlors. *Tobacco International,* Nov 2, 1984.
10. Diet anti-smoking group sees uphill fight in coming drive. *Japan Times,* March 14, 1984.
11. Jackson M: Government rakes in as Japanese smoke themselves to death. *Asahi Evening News,* Feb 11, 1985.
12. Uchiyama Y: Schools cry out against lighting up. *Asahi Evening News,* March 29, 1985.
13. An anti-smoking movement. (editorial) *Japan Times,* April 15, 1984.
14. Imperial household urged to stop cigarette gift giving. *Japan Times,* April 7, 1985.
15. "Clean air, clean music" concert. *Asahi Evening News,* Jan 16, 1985.

American writer and translator Sandy Heinlein, who resides in Japan, observed the union of the ancient performing arts and the modern cigarette pack when he visited the remote village of Kushibiki, which has a 450-year old tradition of amateur Noh performance. In one of the two farmhouse-shrine-theaters of this community, many of the village elders and leading religious figures wearing traditional dress, smoked cigarettes at the most important Buddhist festival. To Heinlein this seemed akin to a priest and choir lighting up during midnight mass.

Maverick doctor-politician puts pillory on tobacco and alcohol companies

Tobacco companies are easy targets in Australia. Their ubiquitous and slick propaganda combined with astronomical profits and 16,000 smoking-related deaths annually provide plenty of ammunition for opponents of the mass consumption of cigarettes. However, to hear a senator in the conservative Liberal Party unequivocally blast the tobacco companies as "bad corporate citizens and unscrupulous traders" is without precedent.

Dr Peter Baume, formerly a surgeon and now in the federal Parliament, lashed out at tobacco and alcohol interests during an invited oration at the Royal Women's Hospital in Melbourne in May 1984. Known for his strong opposition to the advertising techniques employed by the companies, Dr Baume used slides to demonstrate the modus operandi of the companies and their sponsorships. During the speech, he said that cigarette and alcohol companies represent "the unacceptable face of capitalism." He went on to discuss the strategies of such companies:

> The corporations stand condemned of the most cynical, reprehensible, irresponsible, and blatant disregard for the public interest where this conflicts with their corporate goals of profitable consumption. And no amount of sponsorship of sport, culture, or of medical research can ever expiate this sin. I am forced to conclude that, in the case of alcohol and tobacco, any corporate strategy which favors profit and consumption—in spite of massive evidence of crippling adverse effects—is an indictment on the honor of the corporations involved. Sponsorship *per se* is not a bad thing. It is certain sponsorships—those promoting illness and death—which are bad things. The distinction is important.

The companies were outraged. Protests began flowing into Canberra, the seat of the government, and flak is still peppering Dr Baume. The leader of the Liberal Party, Mr Andrew Peacock, quickly distanced himself from his parliamentary colleague, saying, "Senator Baume's strong personal views on this issue are well known to us, but they are not shared by me."

CORPORATIONS COMPLAIN

That corporate storm was not Dr Baume's first baptism with hot oil. In 1978 he called the tobacco companies "drug pushers," a common enough charge, but unheard of for a senator who was then a minister in the ruling government. "Oh boy, calling them 'drug pushers' caused no end of trouble for me, but let's face it, that's really the situation," Dr Baume told *NYJ News Features*. "The corporations complained to the [former] prime minister, and I understand that they are still complaining to my Liberal colleagues, although I haven't seen any written complaints. But they [the companies] appear to be totally displeased with me."

After the May speech, Dr Baume received vigorous protests from alcohol company executives who resented being tarred in the same breath with cigarette companies. They felt that the physiologic, medical, and social aspects of smoking tobacco and drinking alcohol were so totally different that a comparison was unfair. "I could understand their point of view, and we ironed out some of our differences," Dr Baume continued, acknowledging that he appreciates good wine and calling himself a moderate drinker.

Dr Baume has yet to hear of any actions that could replace him as a Liberal candidate. Indeed, he was reelected in December 1984.

For several years Baume wrote a popular column, "From the Benches," for the *AMA Gazette*, the official publication of the Australian Medical Association, in which he frequently called for tougher national strategies against smoking, cigarette advertising, and tobacco companies. While he has gained support among health professionals, his political colleagues have not accepted his views, and they have made efforts to dissociate themselves from him. Bipartisan support from the Labor Party has been equally sparse.

He feels that the major short-term objectives are to get stronger limitations on advertising and to close loopholes in the existing restrictions on advertising. For example, sponsors of cricket can easily circumvent the banning of cigarette commercials on television: in large, bold lettering, a cigarette company's name is painted around the field's perimeter fencing and is continually displayed by the television camera covering the sporting event. "That kind of sponsorship is designed to increase consumption. I'm not out to ban the right to smoke, as some companies like to portray," says Baume. "I just want to stop the recruitment policies which I see as seductive and dishonest. Considering that so many people are venal and will settle for money, the odds are stacked in favor of the tobacco companies. They have tremendous political and economic power. The only way to break that power is to build a large constituency of people and organizations who oppose their products."

INADEQUATE SELF-REGULATION

Dr Baume claims that the self-regulatory advertising codes are inadequate and unfairly stacked in favor of the tobacco companies. Complaints about tobacco advertisements are dealt with slowly, but complaints from tobacco companies about anti-smoking advertisements receive prompt attention. "The self-regulatory codes must be strengthened and administered impartially, which I don't believe is being done. If there was a real commitment to proper advertising, the code could be tightened up by voluntary agreement. But the most vital interest—money—might be affected, so the companies have no intention of improving the code."

In 1977 Baume was the chairman of a senate committee that recommended strong sanctions against tobacco and alcohol companies, but the government largely ignored the report entitled *The Intoxicated Society*.

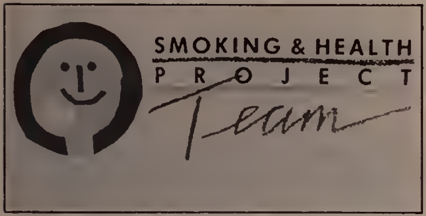
"We did get some wins," Baume states. "The government accepted a policy recommendation that smoking at any level is harmful to health, and a policy that public programs should be started to reduce smoking. Those were important milestones, but the government then proceeded to forget all about them. Politicians may be reluctant to see the overall public health issue because other interests are clouding their vision. But as a doctor, I find the issue clear cut."

Calvin Miller, PhD

Calvin Miller is a medical and science writer based in Taree, New South Wales.

Western Australia gives health promotion the nod

Using funds earmarked from increased tobacco taxes, the Western Australia government has started the world's wealthiest anti-smoking program per capita. The Smoking and Health Project has been granted $6 million over the next three years to deliver its messages to the state's population of 1.2 million. The Labor Government intended the Smoking and Health Project to complement its proposed laws to ban tobacco advertising, but the bills were narrowly defeated in the state Parliament following intense pressure from the Tobacco Institute and print media dependent on cigarette advertising revenue. Despite that setback, the Smoking and Health Project formally began its operations in the state's capital, Perth, in April 1984, after nearly a year of groundwork.



Logo for Western Australia's innovative health-promotion project.

One focal point of the project will be to discourage smoking in children 10 to 14 years of age who are experimenting and are not yet regular smokers. Although social and commercial pressure to smoke is intense, smoking has developed a distinctly negative image in Australia, and this is valuable in discouraging impressionable adolescents from adopting the habit. In an Australian survey, about 95% of the respondents associated smoking with the terms "expensive, obnoxious, harmful, and bad breath," whereas about 10% associated smoking with "sophisticated, clean, or feminine."

Paid Counter-Advertising

The project's campaign will include paid advertising on television, radio, cinema, and billboards; community promotions to highlight the program; training and support groups to assist people wanting to stop smoking; and literature, counselling, and public talks in schools, businesses, and hospitals. To coordinate the program statewide (an area about 25% of the continental United States), the project's team of 13 has enlisted the help of regional health education officers in the Department of Health. Other groups important to the program's success—such as general practitioners and other health professionals, businesses, sporting organizations, health clubs, and entertainment venues—have been recruited to help maintain a high public profile for the project. The Australian Bureau of Statistics will conduct a variety of epidemiologic surveys to assess the effectiveness of the campaign.

To help implement the scheme, Mike Daube, a health activist and lecturer at the University of Edinburgh, was recruited as the Department of Health's executive director of health promotions and educational services. Following a recent shake-up in the system of management of the Department of Health, preventive medicine and health promotion have assumed a greater importance. Daube is responsible for all aspects of health promotion—for example, issues on alcohol abuse, dental health in school children, and community education about sexually transmitted diseases, but his initial thrust is focused on the Smoking and Health Project.

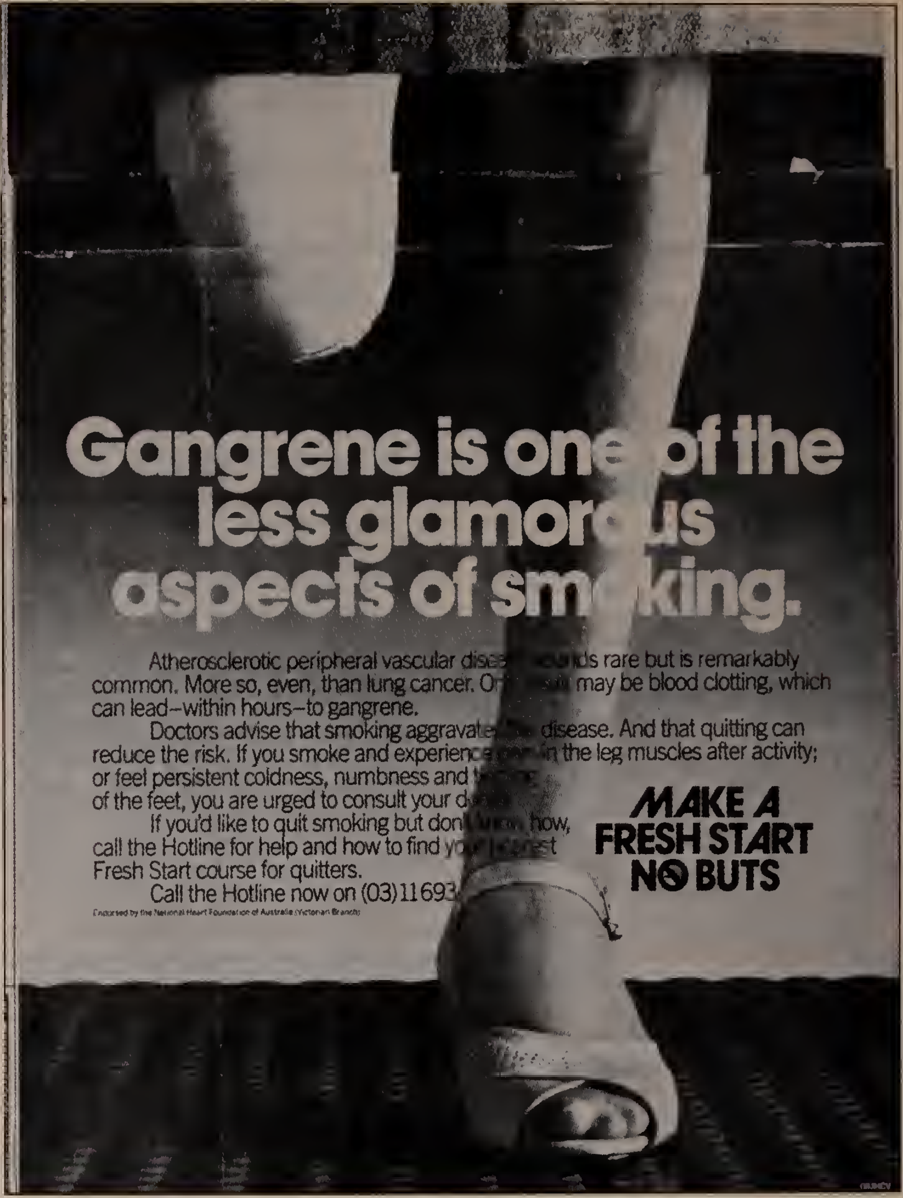
Daube, 36, has not been closely allied with the bureaucracy before, and the appointment is very much a case of "the poacher turning game-keeper." Despite that, Daube said that the state government's firm commitment to reducing smoking was a unique instance of a government living up to its rhetoric on the smoking and health issue. Because the government has placed him on the executive committee of the Health Department, his political vantage point is commanding. "This is the first time in any country that the person in charge of health promotions has been given a knife and fork at the policy table," Daube told the *Journal*. "In Western Australia, health promotion now has a prominent government role and political clout, and will have funding to match that." The Minister of Health for Western Australia, the Hon. Barry Hodge, has taken a determined stand on smoking.

A non-smoking Australia—logo of ASH.

"I gave up smoking and gained 8 pounds"

NSW Cancer Council.

With half-page newspaper ads aimed at pregnant smokers, the New South Wales Cancer Council pointed out the risks for the unborn in a year-long campaign.


Gangrene is one of the less glamorous aspects of smoking.

Atherosclerotic peripheral vascular dise[illegible] [illegible]ds rare but is remarkably common. More so, even, than lung cancer. O[illegible] [illegible] may be blood clotting, which can lead–within hours–to gangrene.

Doctors advise that smoking aggravate[illegible] [illegible] disease. And that quitting can reduce the risk. If you smoke and experienc[illegible] [illegible] in the leg muscles after activity; or feel persistent coldness, numbness and [illegible] of the feet, you are urged to consult your d[illegible]

If you'd like to quit smoking but don[illegible] [illegible] how, call the Hotline for help and how to find y[illegible] [illegible]est Fresh Start course for quitters.

Call the Hotline now on (03)11693

Endorsed by the National Heart Foundation of Australia (Victorian Branch)

MAKE A FRESH START NO BUTS

The Victorian government's no-holds-barred campaign against smoking has featured large advertisements in daily newspapers.

Although the project is well-heeled, budgets for the cigarette companies still leave health-promotion dollars far behind. While the tobacco moguls allocate $5 per Australian for advertising, the government's project has mustered $1.30, with only 80 cents of that being used directly on advertising costs. Despite the disparity, the budget for anti-smoking propaganda is still far ahead of similar programs in other countries. For example, Britain's equivalent program gets only 20 cents per head.

CIGARETTE SALES DOWN

Already, there is evidence that the project's propaganda is altering the public's smoking habits. In June and July 1984, during the project's first "Quit" campaign, government revenue from tobacco taxes was down by 21%. The state—perhaps the first government in history to welcome a loss of $2 million in revenue—applauded the trend. "That's the hardest, firmest evidence of all: sales," Daube said. "For the first time, I think we really have a chance to put health promotion on the map in a way that hasn't happened anywhere else."

Daube's colleagues concur. Deborah Fisher, the team's state coordinator, said that the concept of the program and its implementation had been well received by health professionals, institutions, the general public, and even the media. "When the government was trying to pass the bans on tobacco advertising, the print media were very biased toward the tobacco industry. So I've been surprised at the excellent support we've had from them when our campaign was launched," she said. "The support from all quarters has been so good that I suspect the Tobacco Institute is worried. They've just appointed a spokesman to represent their interests in Western Australia."

Largely educational, the Smoking and Health Project will be joining efforts with the political and lobbying group, Action on Smoking and Health (ASH Australia), based in Melbourne, Victoria, some 2,000 miles to the east on the island continent's south coast. Also born in April 1984, ASH is Australia's first national anti-smoking coordinating organization.

Although funding has come primarily from the Australian Cancer Foundation and the National Heart Foundation, the state governments have pledged once-only funds for setting-up expenses—except Queensland in the north which produces nearly 70% of Australia's tobacco crop. "The Queensland government and its health minister will not do anything that conflicts with the interests of the tobacco industry," said Steve Woodward, the chief executive officer of ASH. "Considering that the tobacco industry is subsidized by the taxpayer to the tune of $30 million every year, Queensland could afford to contribute to these programs. Instead, it's too concerned about catering to the tobacco lobby."

Because the three-year Smoking and Health Project is dependent on government money, its long-term survival is vulnerable to a change in government in Western Australia. However, as a national body supported by non-government organizations, ASH plans to avoid political pressures commonly attached to government funding by becoming financially independent during the next three years.

Calvin Miller, PhD

Medical students take action outside children's hospital

For medical students Tim Read and Karl Horsburgh, the environs of a Melbourne children's hospital was a dubious venue for an enormous cigarette billboard featuring a popular television comedian. In dwindling twilight on a winter afternoon last July, Read and Horsburgh parked next to the billboard—directly opposite the Royal Children's Hospital—and readied their weapons: cans of paint, rollers, and paint brushes.

Veterans of the Australian BUGA-UP campaign to stop unhealthy promotions (*NY State J Med* 1983; 1333–1334), the fifth-year medical students had "redecorated" about 350 billboards during the past two years, and this was simply going to be another notch on the BUGA-UP belt.

As Horsburgh prepared to paint "I am a drug pusher" in a speech bubble next to the head of comedian Graham Kennedy (Figs), a passing motorist, Petty Officer R. Broniman of the Australian Navy, spotted the action and, to the students' surprise, made a rather forceful citizen's arrest, as he allegedly became angry and pushed Horsburgh. Two policemen arrived on the scene and made the arrest official, charging the men with causing willful damage. At first the police and the students mistakenly thought that Broniman was an employee of the Australian Poster Company who had kept watch on the often-defaced billboard.

Sense of Priorities

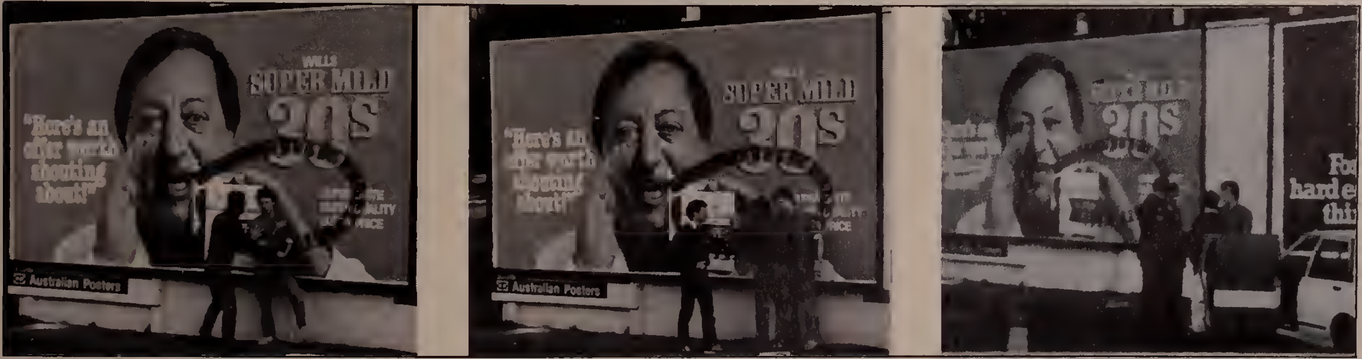
During the past two years, the billboard has attracted the attention and wrath of BUGA-UP spray painters, particularly because of its location outside a children's hospital. Medical personnel have felt that the billboard is offensive and cynical, especially in view of the tobacco industry's claim that its advertising is not directed at children.

Horsburgh told the *Journal* why they felt compelled to attack the advertisement: "We feel that it is justified to break the law which protects property at the expense of community health. This issue has to do with a sense of priorities. If someone's priority is to uphold the law at all times, then we disagree. We believe that the number-one priority is to take measures that will help to reduce the smoking epidemic that has been going on for so many years."

Only minutes prior to their arrest, they had been discussing the possibility of arrest as they walked up to the billboard. "We were well aware of the risks," said Horsburgh. "We've been caught before by the police. They took our names and addresses, but we never heard anything from them subsequently. I suppose an arrest had to happen sooner or later. Actually, after we were arrested, one of the policemen at the station was quite sympathetic to what we're trying to do."

The students were ambivalent about pleading not guilty. If they were convicted, the medical registration authorities would be unlikely to act against the men on the basis of a misdemeanor. However, unemployment among doctors is high in Australia, and the convictions could lead to a decision by conservative authorities to deny the students a hospital position after they qualify. No matter how altruistic the intent, defacing a billboard carries a risk that could rebound on the graffitist in later years.

Because of a last minute mix-up in their lawyers' schedules that threatened to stretch the trial well into 1985, interfering with their final year of studies, Horsburgh and Read decided to plead guilty. Said Horsburgh, "We could have pleaded 'sense of necessity' [an argument where breaking the law becomes more important than observing the law, for example, ignoring a 'no trespassing' sign to save occupants in a burning house]. However, this would have left the case hanging over our heads for some months, and exams are still coming up. Also, Tim is doing a medical elective in New Guinea, and I'm going to Eritrea to do



Medical students Tim Read and Karl Horsburgh are interrupted during their billboard refacing by a concerned citizen. Police officers arrive and arrest the medical students for willful damage to the cigarette advertisement, located opposite the Royal Children's Hospital in Melbourne. (Photographs courtesy *The Melbourne Times*). A photographer from *The Melbourne Times* had accompanied the students to the site because of BUGA-UP's increasing popularity during the past year. He recorded the incident on film, and a front-page story appeared in the 28-page community newspaper.

nutrition surveys for three months in 1985. Having a looming trial could interfere with getting visas from these countries."

Vandals or Heroes?

Hearing the case on October 3 and the students' pleas of guilty, the magistrate put Read and Horsburgh on a one-year good behavior bond of $50 and ordered them to compensate the poster company for $215 in damages to the sign. Although Horsburgh and Read were satisfied with the penalties and the publicity, Petty Officer Broniman apparently was not, feeling that the men had been let off much too lightly. His letter on the matter appeared in *The Australian* newspaper the following week: "As I took some risk in apprehending the youths (I am 43 years old), I did expect some acknowledgement of this and expected much harsher punishment for what was after all an act of vandalism. . . . If the courts expect the law-abiding citizens of Australia to support the police and the law, then I for one will expect to see punishments fitting the crime—and potential danger involved in a citizen's arrest taken into account—when passing judgement before I again do my civic duty."

In a protest attended by about 100 demonstrators during the trial, Karl Horsburgh (center left) and Tim Read (center right) are flanked by a skeleton mannequin with a Graham Kennedy mask (arrow) and posters.

Contacted by the *Journal*, Broniman said that *The Australian* had omitted an important paragraph in his letter. "I had written that the beneficiaries of the arrest were the Australian Poster Company and the Wills Tobacco Company (W.D. & H.O. Wills Australia Ltd), and that neither had bothered to write me or thank me for bothering to help save their property. It would have been a nice gesture, considering my action."

But Horsburgh had the last word. In a polite but frank letter to Broniman he wrote, "The main reason that I became acquainted with the cigarette advertising issue was through my experience of seeing scores of patients, particularly at Heidelberg Repatriation Hospital (in Melbourne), dying prematurely and horribly because of their cigarette smoking. Given your commitment to the public interest, I would be very interested to hear any suggestions you may have for tackling this scourge of our nation."

Calvin Miller, PhD

Medical journal editor stuns and angers health campaigners

Australian health professionals and activists have learned from overseas visitors that this country is foremost in national action to combat the smoking epidemic. For those who have fought a long and difficult battle which is far from over, such praise is hard-won encouragement. The many and varied health campaigners—doctors, physiotherapists, anti-cancer and heart foundations, non-smokers' organizations—have become acclimated to strident opposition, but they were unprepared for an attack from a most unlikely quarter: the editor of the *Medical Journal of Australia (MJA).*

Writing in the April 14, 1984 issue of the *MJA*, Dr Alister Brass warned that some anti-tobacco reformers were tinged with "self-righteous wowserism" and claimed that the use of tobacco as a means of easy, non-verbal communication was a basic human instinct. He wrote that the cigarette is a universal token of acceptance, a form of relaxation for workers on lower rungs of the social ladder, and similar to the peace pipe of North American Indians. Referring to the movement against smoking as a "jihad" (holy war), Brass noted that life itself is a terminal illness. His final words were "Anyone for a smoke?"

Among internationally recognized medical journals, the editorial is the only one of its kind since the first scientific papers linking smoking to cancer and cardiovascular disease appeared more than 30 years ago. Although several guest editorials in various medical and scientific journals have questioned the validity of some scientific studies, none has been written in such a devil's-advocate style.

The reaction from the press and health groups was swift but not as extensive or prolonged as might have been expected. Individuals and groups protested to the *Journal's* parent, the Australian Medical Association (AusMA) and wrote angry letters to various AusMA officials, but the furor died down within weeks. The editorial was an acute embarrassment to the AusMA which has endorsed policies on smoking and public health that were in conflict with Dr Brass' views. Sources within the AusMA have said that the editorial provoked confrontations between Brass and the *Journal's* Board of Directors, but that the board was loathe to interfere publicly, lest it be seen as stifling editorial independence.

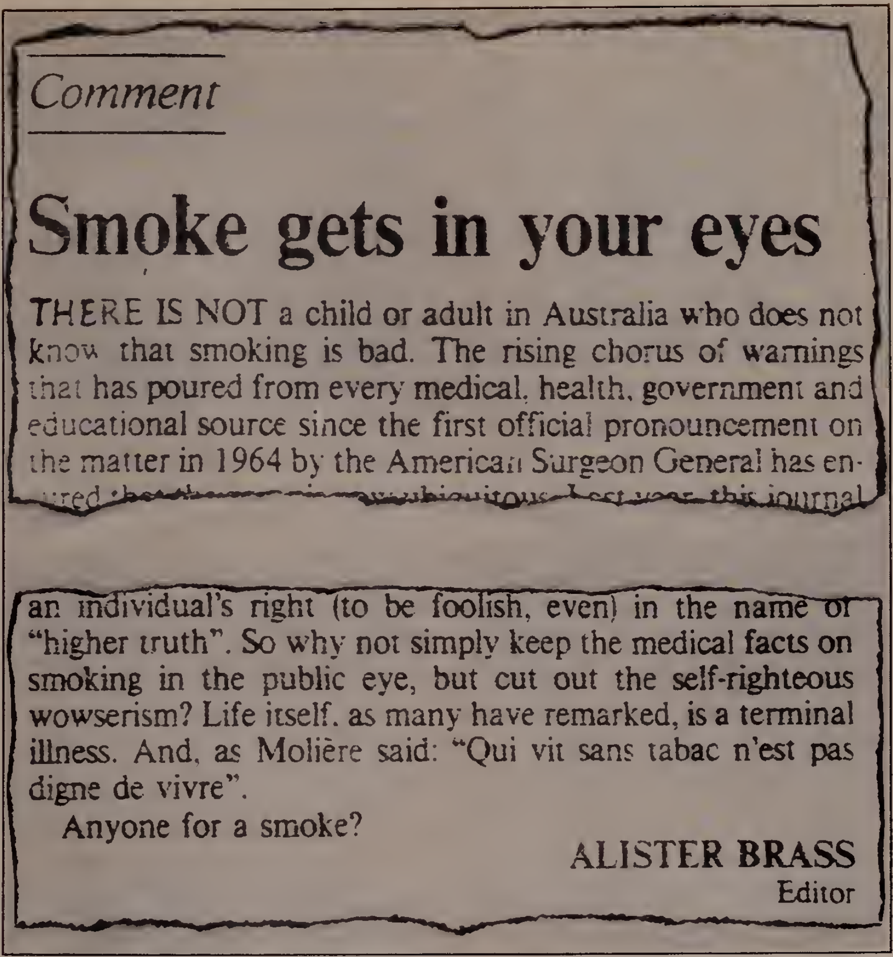
Peter Taylor, British author of *Smoke Ring—The Politics of Tobacco* (London, Bodley-Head, 1984), drew attention to the editorial during a talk in Sydney attended by 100 health educators, activists, and journalists. Taylor told his audience that when he first read the editorial, he stared at it in disbelief, thinking "it must have been some kind of joke." Asked about his reaction to the editorial, Dr Paul Magnus, medical associate of the National Heart Foundation, commented simply, "Unbelievable."

At least two senior officials of national health bodies were so incensed by Dr Brass' reasoning that they privately called for his resignation or a retraction. On the long-term effect, one Melbourne educator noted the following: "The editorial is going to provide plenty of live ammunition for the continuing onslaught of obfuscation by the tobacco industry—and they won't even have to quote him out of context. This editorial is going to be regurgitated ad nauseam by the tobacco lobby."

Those fearing that Brass' article would provide solace to the cigarette industry had their concerns confirmed quickly. Before a Senate Select Committee examining legislation to ban tobacco advertising in the state of Tasmania, the head of the industry's public relations arm, the Tobacco Institute, used the editorial in defense of cigarette advertising.

To its credit, the *MJA* published 10 strongly worded letters to the editor in its edition of May 26, 1984: "the most contemptible piece of commentary ever to have disgraced the pages of a medical journal with pretentions to responsibility. . .feather-brained nonsense"; "his [Brass'] own use of loaded words. . .sets a note of superficiality and insensitivity that seriously calls into question his own judgement and credibility"; "[Brass'] comments will be construed by the tobacco industry and a large segment of the public as an official endorsement of smoking"; "Dr Brass' blinkered view. . .is in harmony with the tobacco industry's dinosaurian view of social responsibility and science."

Brass replied to his critics: "I am disappointed and surprised at the hurt over-reactions of several of those correspondents representing prominent medical bodies whose long-held concern over smoking is generally quite unwowserish. A wise woman once told me that the reason angels can fly is that they take themselves so lightly. Would that we doctors could learn the art," he concluded.



Comment

Smoke gets in your eyes

THERE IS NOT a child or adult in Australia who does not know that smoking is bad. The rising chorus of warnings that has poured from every medical, health, government and educational source since the first official pronouncement on the matter in 1964 by the American Surgeon General has en-
[illegible]

an individual's right (to be foolish, even) in the name of "higher truth". So why not simply keep the medical facts on smoking in the public eye, but cut out the self-righteous wowserism? Life itself, as many have remarked, is a terminal illness. And, as Molière said: "Qui vit sans tabac n'est pas digne de vivre".

Anyone for a smoke?

ALISTER BRASS
Editor

In the late 1960s Dr Alistair Brass, then an assistant editor at the *Medical Journal of Australia*, contributed a series of moving and vivid essays on the tragic war in Vietnam (republished under the title *Bleeding Earth*) that has been hailed as a landmark of frontline medical journalism. In 1984 Brass' cynical editorial commentary deriding the antismoking movement caused dismay among health professionals throughout Australia.

Steve Woodward, a spokesman for the national Action on Smoking and Health (ASH Australia), said that many people in cancer societies and heart foundations were questioning Brass' motives. "Common public knowledge is that the *Medical Journal of Australia* is having serious financial problems. One of Brass' roles would be to stimulate a wider readership, and that could have been a reason for such an inflammatory message. And his article certainly did that," Woodward said. "That issue of the *MJA* was probably more widely read and more commented upon that any other issue during his editorship. But if he got more subscriptions, they would have only come from the tobacco industry," he continued. "His article has done a lot to damage the reputation of the *MJA*. So rather than increase the circulation of the journal, the editorial is likely to have done exactly the reverse."

Contacted by the *Journal* eight months after the initial storm, Dr Brass defended his editorial and said he was disappointed that smoking had become such an emotive issue in Australia, with doctors here apparently unwilling to get as deeply involved with other important issues. "For example, I'd like to see more debate on the oversupply of medical students and medical schools which has become a major threat to the profession," Brass said. "The journal must be a meeting ground for airing philosophies, ideas, angst, and concern, and for initiating debate. With that in mind, I've tried to be provocative. The smoking debate has become full of knee-jerk responses. I've seen doctors condemn smoking and then light up five minutes later, so there's a lot of hypocritical, two-faced behavior. Many anti-smokers have invested in the tobacco issue a degree of ego involvement that would normally be seen in a religious crusade."

About 35 protests concerning the editorial have appeared in the *MJA*'s correspondence column during the past year, and letters continue to trickle in. Brass felt that most of the letters were "excessively emotional, self-serving and self-righteous, and intemperate in language." He felt bound to publish the correspondence, except for some that had legal implications. Some doctors called him and supported his views, but did not want their names to become involved in the debate. "Other doctors said they were sick of anti-tobacco preaching but refused to be quoted. That worried me. People can become intimidated by a dogma and then afraid to question the conventional wisdom."

Brass was disappointed that the tobacco industry had seized the opportunity to misconstrue his editorial as support for its views. "The tobacco people twisted my editorial for their own ends. If they had chosen to read it properly, they would have seen that I noted that the dangers of tobacco should be kept very much in the public's eye—children and adults. But the key issue is the way in which those warnings on smoking are promoted; that is what I was writing about."

In consideration of the heat and friction provoked by his article, Brass appeared ready to let the issue rest, at least temporarily. "I think this journal should play possum for awhile on the tobacco issue."

Calvin Miller, PhD

BUGA-UPdate

Since the *Journal*'s first story on Australia's BUGA-UP movement (Billboard Utilising Graffitists Against Unhealthy Promotions) in the December 1983 issue, the activists have stepped up their efforts.

BUGA-UP has mixed a cocktail of civil disobedience, humor, and disregard for convention that would have urged an approving smile from Henry David Thoreau. One suspects that Thoreau's scorn of materialism and fondness for unperverted truth would have provoked more volumes had he seen around his beloved pond giant billboards promising wealth, vitality, social acceptance, sexual success, and glamor in the consumption of cigarettes, fast food, liquor, and similar products.

Civil disobedience is the keystone of BUGA-UP. In the five years of declared war on advertising propaganda and psychology, the activists (known as BUGs) have sprayed their war paint on between 7,000 and 10,000 billboards—the majority being in Sydney, although other capital cities (most notably Melbourne) have been catching on.

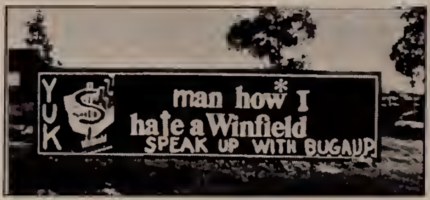
The acronym plays on an inoffensive Australian idiom "bugger up," meaning to cause frustration or inconvenience to something. In spraying their humorous artwork over the crafty subliminal messages, the BUGs have indeed caused headaches among advertising executives, who claim that BUGA-UP has defaced billboard posters worth millions of dollars in rental fees.

The Bogie Awards

To celebrate BUGA-UP's fifth birthday in October 1984, 250 sympathizers attended the First Advertising Bogies, a madcap dinner and ceremony (created by businessman and BUGA-UP supporter Peter Vogel) that was essentially a Bronx cheer for the advertising industry.

The Bogies presentation night was preceded by afternoon seminars on advertising psychology, law, industry self-regulation (or the lack of it) and consumerism. Other topics included nutrition, the pharmaceutical industry, sexual images and stereotypes, and gambling.

Although speakers consisted of professionals such as physicians, educators, lawyers, civil servants, and journalists, the Advertising Federation of Australia (AFA) was displeased with the event and protested to the owners of the hall, the New South Wales Institute of Technology. "The institute was in a bit of a panic and wanted confirmation that BUGA-UP was not going to commit any criminal activities, so we reassured them," said Brian Robson, a communications engineer and one of the originators of the BUGA-UP concept. "The AFA's letter was mischievous and misleading, to say the least."



Refaced billboard for most-advertised cigarette brand.

Dr Arthur Chesterfield-Evans, a surgeon, told the *Journal* that the dinner and the seminar received good publicity from print and broadcast journalists. "The seminar was the first other-side-of-advertising convention that's ever been held in Australia, and we'd like to make it an annual event. Our speakers came from all over the country," he said.

A veteran of BUGA-UP, Dr Chesterfield-Evans has had battle experience in the front lines. His arrest in September 1983 for "willfully marking a premises with paint" (the fifth Australian doctor to be charged with defacing a cigarette billboard) was one of the most celebrated and publicized of all BUGA-UP cases. Pleading innocent, he was found guilty and fined $20, with $160 court costs. He appealed and in the District Court was acquitted in May 1984. Paradoxically, the state prosecutor provided the surgeon's defense by finding an obscure loophole: the poster that Chesterfield-Evans was alleged to have defaced was not actually part of the billboard (or "premises") because it was a movable structure. "I think the government didn't want a conviction because that could have provoked the situation. They were faced with the possibility that I would choose to go to jail rather than pay the fine, and that would have created sensational publicity," he said. "The press would have had a field day with the courts, the police, and the poster companies over a doctor sent to jail for painting a cigarette billboard—for fighting an unhealthy promotion."

Turning the Tables

Because of the precedent, no one in BUGA-UP has since been charged under that section of the law, but a graffitist could still be charged with malicious damage. "However, the prosecution would have to prove malice, and that can be extremely difficult," Chesterfield-Evans said. "If the police want to charge any of us with malicious damage, then that's going to be the best publicity bonanza we could have. Publicity is such a crucial part of BUGA-UP's civil disobedience. The sad reality is that if you don't have a gimmick, then

Sydney, Christmas eve, 1981: From Marlboro to "it's a bore."

you don't get the publicity. If you're strictly law abiding, no one takes any notice. The point we're trying to emphasize is this: advertisers and the advertising industry have unfettered powers, they are using those powers irresponsibly, and the time has come for a challenge from consumer groups. BUGA-UP would not claim to be more than publicists for this idea."

Since his acquittal, the doctor has helped start Australia's first radio program devoted solely to the battle against the promotion of unhealthy products. Aired in Sydney, "Puff-Off" features BUGA-UP news and interviews with doctors and politicians.

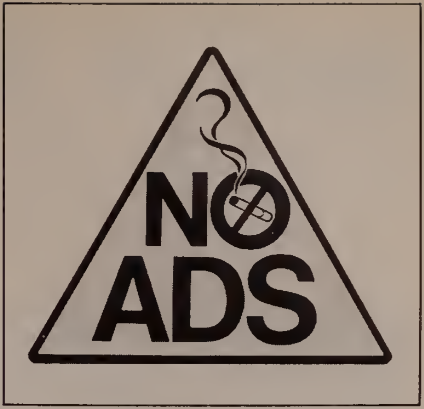
In the past year, the courts have continued to wrestle with BUGA-UP's own brand of civil disobedience. During July in Adelaide, Fred Cole, 58, an environmentalist and a decorated veteran of World War II, was arrested after he used an extension ladder to reach a cigarette billboard 30 feet above the pavement. Below the slogan "Come to Marlboro Country," Cole painted "And meet BUGA-UP." Driving in rush-hour traffic, police spotted the graffitist from about one mile away, but by the time they arrived, he had emptied several spray cans.

He refused demands to come down the ladder and then perched resolutely on a ledge, forcing police into the delicate maneuver of urging him down without precipitating a fall. He was charged with causing $2,600 of damage to the billboard, but in an apparent lapse of concentration, the poster company and the prosecution neglected to produce receipts to the court proving the damage. The judge refused to commit Cole to trial and dismissed the case.

Although surviving that charge, Cole is awaiting the outcome of another case in which he attempted to interview hostesses who were passing out free cigarettes at a Sydney shopping arcade. He was asking the young women whether they felt any remorse at "pushing addictive drugs on children," when he was arrested by Sydney police (summoned by the arcade manager) and charged with "remaining on enclosed lands." The court case became complex because of two statutes that contradicted each other, one exonerating Cole and the other incriminating him. When the magistrate had to adjourn the case for two weeks to deliberate over the inconsistency, Cole (conducting his own defense) argued that if a magistrate needed two weeks to decide the dilemma, then an ordinary citizen could not be expected to make a decision on the spur of the moment about the law. The case has yet to be resolved, with both sides claiming that they will appeal if the decision is against them.

EMBASSY BESIEGED

In May 1984 legal troubles befell the BUGA-UP embassy, a mobile van set up outside the Sydney offices of Leo Burnett, the Chicago-based international advertising agency that promotes Philip Morris' Marlboro cigarettes. Next to a large poster of an advertising executive grasping handfuls of dollars with the caption "Greed Breeds Mean Deeds," the embassy set up a confessional and encouraged advertising executives to admit "their nefarious deeds." After three weeks of media events, distributing propaganda to the public, and verbally skirmishing with the advertising agency, the embassy became the victim of behind the scenes political pressure. The "ambassador," foodstore owner Bill Snow, was arrested for trespassing, despite his claim of diplomatic immunity. His case is pending.



New international symbol created by BUGA-UP.

Now that more than 40 BUGs have been arrested during the past five years, a pattern is beginning to emerge about their fate and treatment by the courts: typically lenient, as exemplified by a case reported in the *Journal* (1983; 83:1333-1334). Unresolved when that issue went to press, the case was finally decided in October 1984. Two BUGs, Rick Bolzan and Lord Bloody Wog Rolo, were charged with damaging a Marlboro racing car (on display at an art gallery during a Philip Morris-sponsored exhibition) by spraying paint and sprinkling cigarette butts over it. The prosecution claimed that the men caused $1,800 damage to the high-tech Alfa Romeo. Bolzan was acquitted by the jury, but Rolo was found guilty and fined $75, which he declared he would refuse to pay, opting for three days in jail. No costs were awarded to the Alfa Romeo owners because the extent of damage was not proven.

"Civil disobedience is the best way of fighting the advertising establishment, but to be successful, you have to know how to get into the media," Bolzan told the *Journal* after his acquittal. "It's important to get publicity at the time of the event and during the trial, and then you can measure the impact. Since we've had so much publicity, the Art Gallery of New South Wales has not had any similar displays. Our action was clearly effective—if we had simply written protest letters, that car would probably still be sitting there."

The poet Walt Whitman admired Thoreau's "lawlessness, his dissent, his going his own absolute road let hell blaze all it chooses"—words that today still find particular relevance for BUGA-UP men and women resisting the onslaught of dubious advertising tactics. Thoreau and BUGA-UP are separated by 10,000 miles and 123 years, but their common intent and civil disobedience stand clear and firm.

Calvin Miller

MOP-UP battles tobacco sponsorship—legally

In the beginning, there was MOP-UP (the Movement Opposing the Promotion of Unhealthy Products), formed in Sydney during June 1979—before BUGA-UP graffitists began their formal career. MOP-UP is the strictly legal alternative for consumer-protection advocates who are too circumspect to emulate the tactics of BUGA-UP and risk an arrest. The group's strategies consist of well-organized, humorous demonstrations at high-profile events such as the Australian Open tennis competition sponsored by Philip Morris' Marlboro and the Australian Ballet sponsored by Benson and Hedges (British American Tobacco).

Sharing prestige with the US Open, the French Open, and Wimbledon as one of the Grand Slam tournaments, the Australian Open is now known as the Marlboro Australian Open, to the chagrin of the nation's health organizations. But MOP-UP, at least, has been a thorn in the side of Philip Morris' publicity bonanza. For the past two years MOP-UP has staged street theater in the environs of the Melbourne tournament with displays including tombstones with satiric inscriptions; a "Marble Row" scoreboard tallying the number of cigarette-related deaths; cowboys and cowgirls handing out "Smokers are dying to bring you the tennis" badges to spectators; protesters in the stands dressed in skeleton costumes and wearing cowboy hats; and puppet shows with sports-star marionettes being manipulated by a "tobacco rep" sitting in an umpire's chair.

At the Australian Open, now renamed the Marlboro Australian Open, perimeter advertising for Marlboro was constantly in the viewfinders of the television cameras. (Photo courtesy *The Age*).

The Australian Ballet's production of "The Sleeping Beauty," sponsored by Benson and Hedges (British American Tobacco), attracted unwanted publicity when MOP-UP members dressed in skeleton suits and tutus distributed health-promotion literature outside the Victorian Arts Centre.

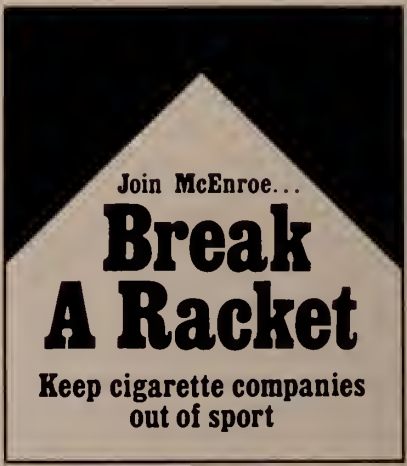
Marge White, a spokeswoman for MOP-UP, said that radio and television media were generally eager to cover the demonstrations, but that coverage from the print media (which are still permitted to carry tobacco advertisements and depend on tobacco products for about 10% of their advertising income) was slim. "Our goal is to produce humorous and mocking images, not frightening ones. The public enjoys the humor, and even smokers like to see the big companies being brought down a peg or two," she said.

MOP-UP's most notable achievement was a legal battle against Winfield cigarettes, marketed by Rothmans of Pall Mall Australia Ltd. Rothmans used Paul Hogan, a popular comedian who now appears throughout American television in tourist promotions for Australia, for Winfield's big commercial debut in 1972. The advertising gimmick snatched a startling 25% of the market for Winfield. In advertising and marketing circles, Rothmans' $25 million campaign was considered a brilliant coup, but MOP-UP claimed that Hogan, a hero to the nation's children, was responsible for inducing youngsters to smoke. The original complaint against the Hogan advertisements was filed in 1978 by John Carmichael, a health education officer with the New South Wales Department of Health, who formed MOP-UP in association with other health educators, and Simon Chapman, now at the Health Education Council in London.

After a frustrating delay of 18 months, the Advertising Standards Council (an industry-run body) decided in 1980 that the Winfield advertisements contravened the advertising code because of their appeal to children. Rothmans was ordered to exclude Hogan from its cigarette advertising (*Br Med J* 1980; 281: 1187–1190). Carmichael's methods were unorthodox for the Department of Health, and his superiors were unsure of how to handle his activities. "I put other projects on the back burner and concentrated full-time on MOP-UP and the action against Rothmans," Carmichael told the *Journal*. "My supervisors made formal requests that I not do that, but then they turned a blind eye. The funny thing was that administrators in the upper echelons were defending MOP-UP, but at the same time telling me not to do it. They were on-side but covering their tracks if anything went wrong."

MOP-UP's victory was only a minor setback for Rothmans' aggressive marketing strategies. In August 1984, Rothmans (Australia) posted a record profit of $38.5 million (up 22%) for the fiscal year and captured 35% of the tobacco market, edging out Amatil (the Australian branch of British American Tobacco) (32%) and Philip Morris (31%).

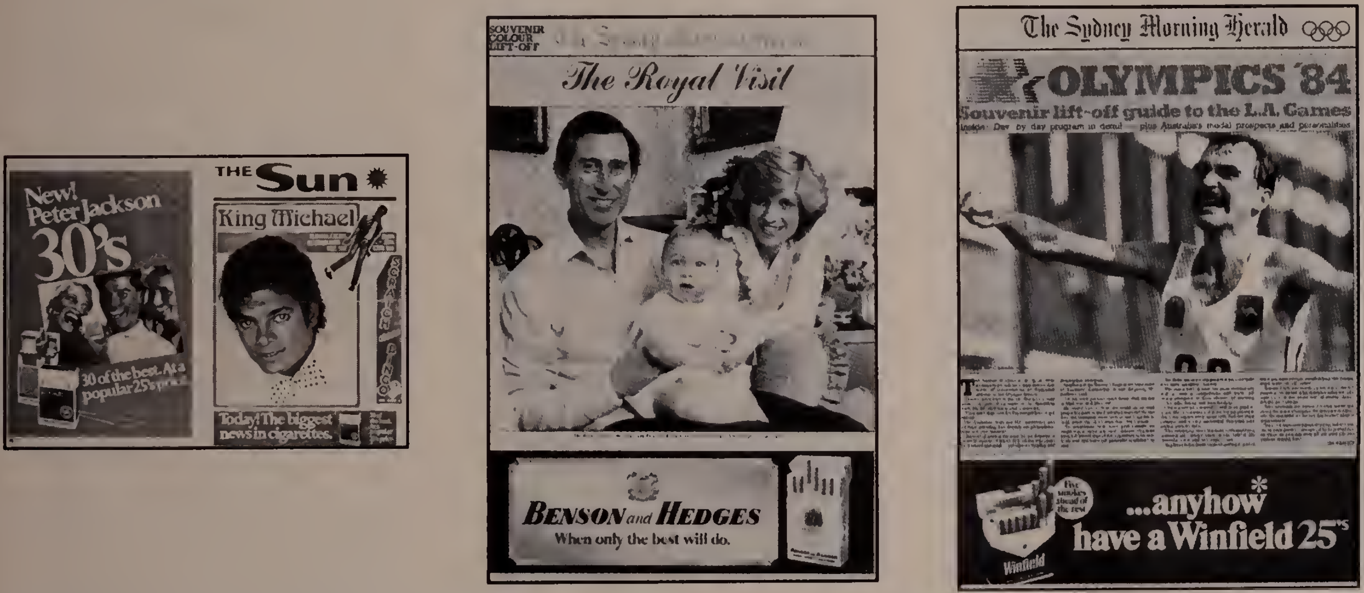
Calvin Miller, PhD



MOP-UP badge

Marlboro takes the wind out of tennis.

BENSON and HEDGES STUMPS YOUR GROWTH


New! Peter Jackson 30's
30 of the best. At a popular 25's price.
THE Sun
King Michael
Today! The biggest news in cigarettes.
SOUVENIR COLOUR LIFT-OFF
The Royal Visit
BENSON and HEDGES
When only the best will do.
The Sydney Morning Herald
OLYMPICS '84
Souvenir lift-off guide to the L.A. Games
...anyhow* have a Winfield 25's
Winfield

Examples of the way in which newspaper front pages and photographs of celebrities are used by cigarette advertisers.

NEW WAVE AMERICAN COUNTERADVERTISING

Photographs: Steven Furrow

"When you take a puff/you think it's cool/ I've had enough/I'm leaving you." Excerpt of lyrics from "Please Don't Smoke," one of the new wave rock video counteradvertisements created by Cole & Weber and Washington DOC (Doctors Ought to Care). Submitted by Robert Jaffe, MD, 1222 17th Ave East, Seattle WA 98112.

Canada's oldest daily newspaper is country's first to drop cigarette advertising

Why would a businessman turn down $50,000 in annual revenue that his colleagues readily accept? For newspaper publisher Michael Davies, it was a matter of putting social conscience ahead of commercial interests. On January 1, 1985 Davies' newspaper, *The Whig-Standard* (circulation 37,000), in Kingston, Ontario, became the first Canadian daily to turn down cigarette advertising.

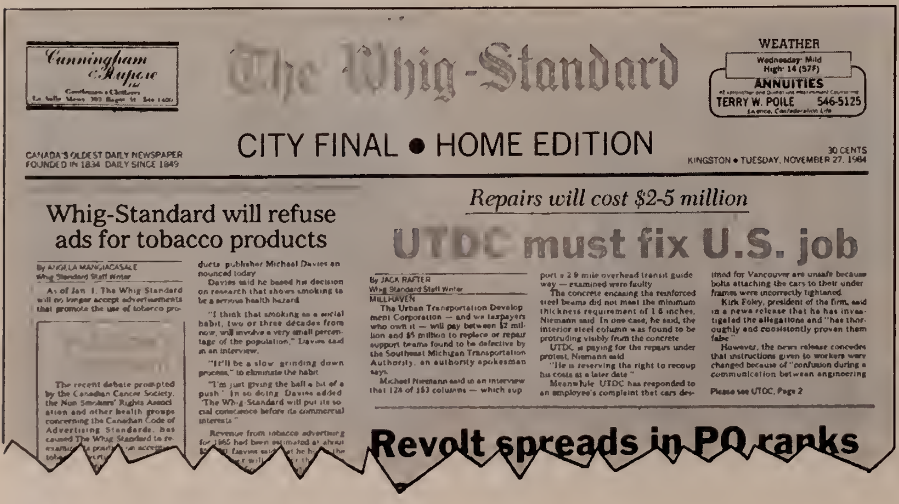
"Newspapers each day reflect social change in many areas; less frequently do newspapers champion and promote social change," Davies wrote in a front page publisher's statement on November 27, 1984 (Fig 1). Davies explained that he could no longer continue to accept advertising for a product that is a serious hazard to the health of Canadians. (Davies also banned smoking in the office.) "What I'm trying to do is stop these ads which equate smoking with a positive lifestyle," Davies told the *Journal.* "I think that two or three decades from now smoking as a social habit will involve a very small percentage of the population. Eliminating the habit is a slow grinding-down process. I'm just giving the ball a bit of a push."

Davies' action has been applauded by the Canadian Minister of Health, Jake Epp, who commended the publisher for "putting his money where his mouth is." John Last, MD, editor of the *Canadian Journal of Public Health,* called the move "very progressive. I hope it establishes a trend."

Other Canadian publishers were less laudatory, however. "Advertising is a form of freedom of speech, and I feel you interfere with that only when legal, moral laws or community standards are violated," said A. Roy Megarry, publisher of *The Globe and Mail* (Toronto), which has a national circulation of 328,000. Megarry has rejected a ban on tobacco advertising at his newspaper.

Gordon Fisher, president of a corporation (Southam) that publishes 15 daily newspapers in Canada, said the issue of refusing cigarette advertisements has never been formally discussed among his publishers. "My own view is that since tobacco is legally grown, manufactured into cigarettes, and sold with messages [about health dangers] required on all ads and packs, and since newspapers are involved in reporting the dangers of smoking, to deny access to our columns is probably not necessary," said Fisher. "I get nervous at the thought that we as newspaper publishers should decide what's good for the public and what isn't."

The tobacco industry was also upset by Davies' decision. Norman McDonald, executive director of the Canadian Tobacco Manufacturers' Council, called the ban "disturbing . . . [because] that is a legitimate product." To Jacques La Riviere, director of public affairs for the council, the banning of advertisements for a legal, highly taxed, widely used product raises the question, "What's next?" La Riviere also claims that there is "ample research that shows that advertising tobacco doesn't make people start smoking or continue."



Cunningham & Rupert

WEATHER
Wednesday: Mild
High: 14 (57F)

ANNUITIES
TERRY W. POILE 546-5125

The Whig-Standard

CITY FINAL • HOME EDITION

CANADA'S OLDEST DAILY NEWSPAPER
FOUNDED IN 1834 DAILY SINCE 1849

30 CENTS
KINGSTON • TUESDAY, NOVEMBER 27, 1984

Whig-Standard will refuse ads for tobacco products

By ANGELA MANGIACASALE
Whig-Standard Staff Writer

As of Jan. 1, The Whig-Standard will no longer accept advertisements that promote the use of tobacco products publisher Michael Davies announced today.

Davies said he based his decision on research that shows smoking to be a serious health hazard.

"I think that smoking as a social habit, two or three decades from now, will involve a very small percentage of the population," Davies said in an interview.

"It'll be a slow grinding down process," to eliminate the habit.

"I'm just giving the ball a bit of a push." In so doing, Davies added, "The Whig-Standard will put its social conscience before its commercial interests."

Revenue from tobacco advertising for 1985 had been estimated at about

The recent debate prompted by the Canadian Cancer Society, the Non-Smokers' Rights Association and other health groups concerning the Canadian Code of Advertising Standards, has caused The Whig-Standard to re-

Repairs will cost $2-5 million

UTDC must fix U.S. job

By JACK RAFTER
Whig-Standard Staff Writer

MILLHAVEN

The Urban Transportation Development Corporation — and we taxpayers who own it — will pay between $2 million and $5 million to replace or repair support beams found to be defective by the Southeast Michigan Transportation Authority, an authority spokesman says.

Michael Niemann said in an interview that 128 of 163 columns — which support a 2.9 mile overhead transit guideway — examined were faulty.

The concrete encasing the reinforced steel beams did not meet the minimum thickness requirement of 1.5 inches, Niemann said. In one case, he said, the interior steel column was found to be protruding visibly from the concrete.

UTDC is paying for the repairs under protest, Niemann said.

"He is reserving the right to recoup his costs at a later date."

Meanwhile UTDC has responded to an employee's complaint that cars destined for Vancouver are unsafe because bolts attaching the cars to their underframes were incorrectly tightened.

Kirk Foley, president of the firm, said in a news release that he has investigated the allegations and "has thoroughly and consistently proven them false."

However, the news release concedes that instructions given to workers were changed because of "confusion during a communication between engineering

Please see UTDC, Page 2

Revolt spreads in PQ ranks

FIGURE 1. Front-page publisher's statement.

Poor Sports

Through tobacco company sponsorship of such sporting events as the World Windsurfing Championships (held in the waters off Kingston in 1983), various cigarette brands receive extensive exposure in the mass media. As a result of *The Whig-Standard's* decision to refuse tobacco advertisements, Kingston was dropped as the site of the 1985 Windsurfer International Export A Cup by organizers fearing adverse publicity.

RJR Macdonald (Canadian subsidiary of RJ Reynolds), the tobacco company whose Export A brand sponsors the event, was not behind the decision, according to Michael Booth, program manager. "When we found out about the newspaper, even before we consulted RJR Macdonald, we decided to back out," said Booth. "We feel there shouldn't be any potential for conflict to arise for the area where we're going to hold the regatta. Any negative publicity is not good."

Booth added that he could not fault the newspaper's past coverage of sailing and sailboarding events. Publisher Davies said that *The Whig-Standard* has a tradition of extensive reporting of aquatic events and that advertising policy does not affect the news operation. Davies was public relations officer for the Canadian Olympic-training Regatta when it began in Kingston in 1969. The sponsor was Rothmans Pall Mall Canada Limited, another cigarette manufacturer (Fig 2). "Tobacco companies don't sponsor regattas because they think windsurfing is a marvelous thing," said Davies. "They want to sell cigarettes. My personal feeling is that tobacco companies and sports events are a poor mixture. A good athlete doesn't smoke."

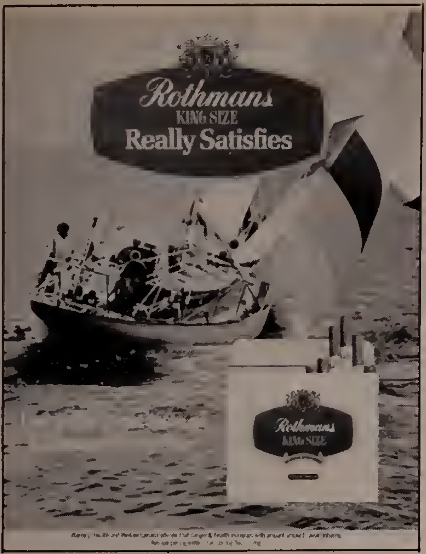


FIGURE 2. Sports motif of Rothmans advertisement.

PRECEDENTS IN THE UNITED STATES

In the United States a handful of large daily newspapers such as *The Deseret News* (Salt Lake City), (circ 64,426) and *The Christian Science Monitor* (circ 141,247) have never accepted tobacco advertising on religious grounds. But there are also a few smaller daily newspapers that have imposed a ban out of concern for the health of their readers.

In 1949 David B. Lindsay, Jr, publisher of the Sarasota (Florida) *Herald-Tribune and Journal,* stopped accepting cigarette advertisements. He had become convinced of the connection between smoking and disease after his father lost a lung to cancer. The policy remained in effect until 1982 when the newspaper was purchased by The New York Times Company, one of the nation's largest recipients of cigarette advertising revenue. Lindsay, who is no longer publisher, remains steadfast in his opposition to tobacco as a "totally poisonous substance."

At the *News-Banner* (circ 5,640) in Bluffton, Indiana, general manager James Barbieri said the newspaper stopped accepting cigarette advertising in 1964 following the release of the first Surgeon General's report on smoking and health. "We weren't telling people not to smoke," Barbieri said. "But we didn't want to take money for promotion of a product where there was such strong evidence that its use contributed to cancer, heart disease, and other illnesses. He estimates that the newspaper turns down between $1,000 and $2,000 in cigarette advertising per month, out of a total revenue of between $35,000 and $40,000.

The Daily Record (circ 59,037) in Morristown, New Jersey also does not accept cigarette advertisements. Nor does the *Daily Express and News* (circ 9,700) in Kirksville, Missouri. Publisher Ed Swain said his newspaper began refusing tobacco advertising shortly after the end of the World War II. "The cigarette companies came out with advertisements with professional players endorsing their products. My father, who had been captain of his college football team, found them pretty repulsive. He thought they were deceptive and contrary to health recommendations."

When Fred Vandegrift, publisher of the *Salina (Kansas) Journal* (circulation 30,000) announced that as of January 1, 1984 in the interest of the health of his 110 employees smoking would no longer be permitted in the newsroom, several letter writers and staff members pointed out the inconsistency of the newspaper's continuing to accept cigarette advertising. Upon reconsideration, Vandegrift dropped all cigarette advertising effective January 1, 1985.

Angela Mangiacasale

Ms Mangiacasale is a staff writer for the Whig-Standard.

TOBACCO COMPANY COULD NOT STAND HEALTHY COMPETITION

The reluctance of the mass media to challenge the tobacco industry would appear to be based on the perceived threat of loss of advertising revenue. Often overlooked is the importance tobacco companies place on advertising in the credible context of local media. This was well illustrated in June 1979 when the *Ottawa Citizen* launched an intensive stop-smoking campaign. In a series of daily front-page articles throughout the month, reporter Karin Moser conveyed comments on smoking by heart and lung specialists in a shocking but comprehensible way.

In editorials the newspaper urged readers to "start being a healthy Canadian" and offered free buttons that read, "I'm a non-smoking Canadian." Readers were encouraged to send in a photograph and fill out a pledge form to stop smoking permanently on June 30, 1979. On that day the *Citizen* published hundreds of photographs and the headline, "12 hours to go—then 1,052 butt out."

Photographs of lung tumors were featured along with statements by citizens who had been disabled by smoking. Among the headlines were the following: "More women getting throat cancer," "Heart surgery big convincer (for a 36-year old man)," and "Girl hooked at nine, quitting at 15."

In the midst of the series, Imperial Tobacco Ltd (affiliate of the British tobacco giant, Imperial Group) pulled the balance of its June and July advertising. The company's vice-president of marketing was quoted as expressing "surprise" that the *Citizen's* sales department "hadn't let us know you were running this type of campaign and ask us in advance if we wanted to run our ads during that time." The *Citizen's* headline over the story of the pull-out read, "They quit COLD TURKEY."

The advertisements resumed soon after the series ended.

—Editor

The Canadian Non-Smokers' Rights Association: shaming the establishment

Garfield Mahood's mission is to put himself out of a job. The 44-year-old executive director of the Canadian Non-Smokers' Rights Association (NSRA) would love to see his work result in the eventual demise of the $4 billion-a-year Canadian tobacco industry. There then would be no need for someone to spearhead a campaign to protect the rights of non-smokers.

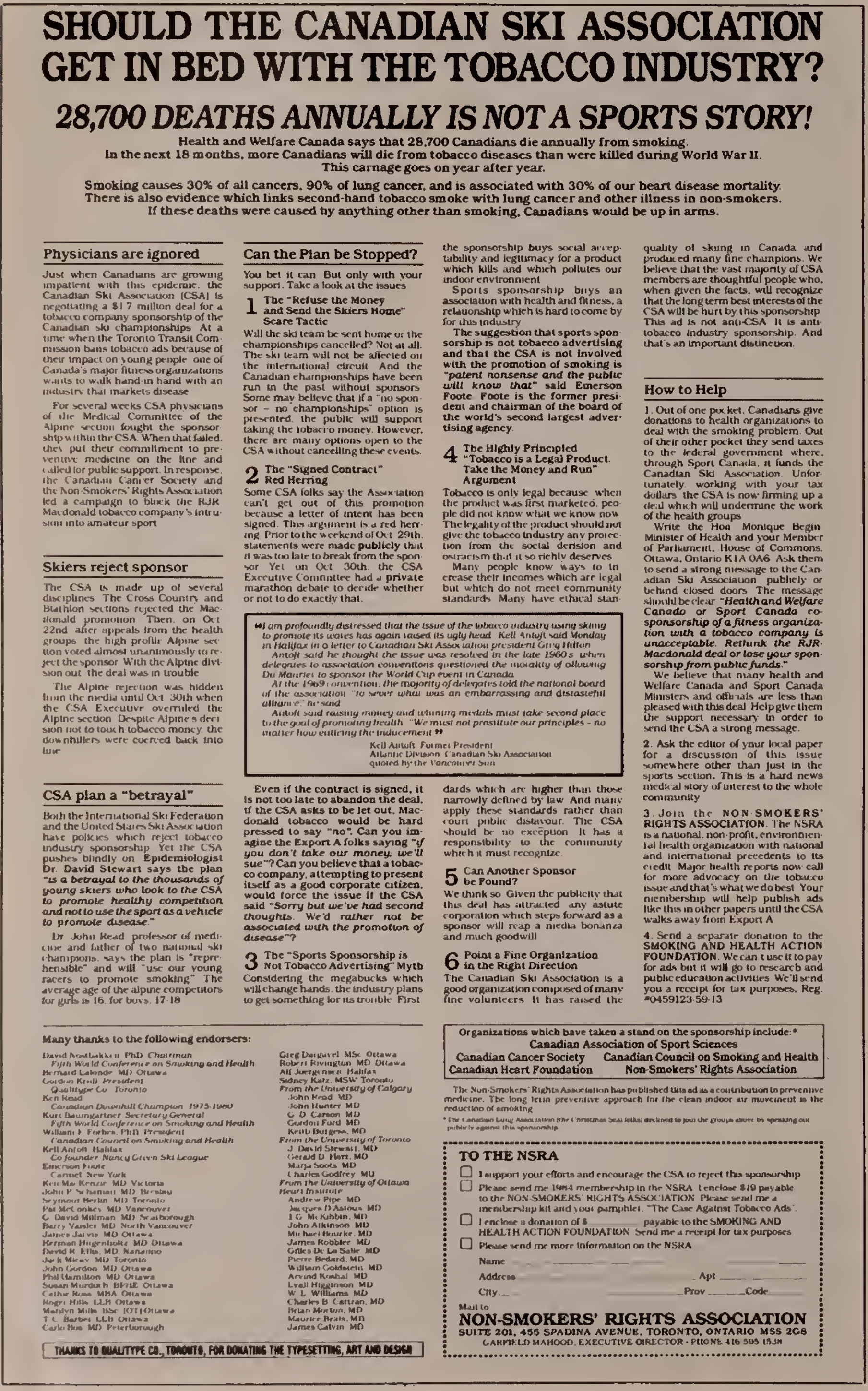
Since the group began, NSRA has played a major role in campaigns against the tobacco industry, especially in the areas of second-hand smoke and tobacco advertising. In an international precedent, the group successfully lobbied two of Canada's largest commuter transit systems (Toronto and Hamilton) to reject tobacco advertising. Lawyers for NSRA set a national precedent in winning an appeal before the Ontario Workmen's Compensation Board in which a worker received compensation for a disability attributed to second-hand smoke in the workplace. In 1984, NSRA received national attention when it released documentation of over 250 examples of cigarette signs and billboards near to or facing school playgrounds, in violation of the tobacco industry's self-regulatory advertising code that prohibits cigarette advertising within 200 meters of a school.

The battle continues against tobacco industry sponsorship of sports organizations, especially the government-subsidized Canadian Ski Association (Fig 1), and against advertising of tobacco products in Canadian newspapers (Fig 2). As the result of the former effort, several champion skiers refused to accept tobacco company-sponsored trophies (the Canadian Ski Association has made this the last year the Canadian Alpine championships will be sponsored by RJ Reynolds-Macdonald Tobacco Company), and the Minister for Recreation and Sport announced in January, 1985 that no new cigarette sponsorship of government-supported skiing will be condoned. In the latter campaign, NSRA's efforts helped convince the publisher of the Kingston, Ontario *Whig-Standard* to stop accepting tobacco advertising (see accompanying story).

Helping establish a ban on tobacco advertising in one newspaper, says Mahood, is the start of a significant phase in the battle against tobacco. "If we get the tobacco advertising ban in place in all media, I suspect that we will have won the war at that point. The issue will have won," Mahood said during an interview with the *Journal*. "Public opinion will have changed with respect to the tobacco industry. It will not be viewed as a good corporate citizen and the public will be ready to move with other legislative initiatives to curtail the industry. The ban on tobacco advertising is no panacea. However, it is probably the single most logical thing that can be done to bring some kind of regulation to the industry." NSRA has taken the lead in Canada in pressing newspaper publishers to stop running tobacco advertisements.

According to the Canadian Code of Advertising Standards, "Advertisements shall not display a disregard for public safety or depict situations which might encourage inappropriate, unsafe or dangerous practices." Newspaper publishers have voluntarily endorsed and applied the code, but they contravene it by publishing tobacco advertisements, says Mahood.

The argument from publishers across the country has been that such a ban would constitute "commercial censorship." Mahood claims that there is no



SHOULD THE CANADIAN SKI ASSOCIATION GET IN BED WITH THE TOBACCO INDUSTRY?

28,700 DEATHS ANNUALLY IS NOT A SPORTS STORY!

Health and Welfare Canada says that 28,700 Canadians die annually from smoking.
In the next 18 months, more Canadians will die from tobacco diseases than were killed during World War II.
This carnage goes on year after year.

Smoking causes 30% of all cancers, 90% of lung cancer, and is associated with 30% of our heart disease mortality.
There is also evidence which links second-hand tobacco smoke with lung cancer and other illness in non-smokers.
If these deaths were caused by anything other than smoking, Canadians would be up in arms.

Physicians are ignored

Just when Canadians are growing impatient with this epidemic, the Canadian Ski Association (CSA) is negotiating a $1.7 million deal for a tobacco company sponsorship of the Canadian ski championships. At a time when the Toronto Transit Commission bans tobacco ads because of their impact on young people, one of Canada's major fitness organizations wants to walk hand-in-hand with an industry that markets disease.

For several weeks CSA physicians of the Medical Committee of the Alpine section fought the sponsorship within the CSA. When that failed, they put their commitment to preventive medicine on the line and called for public support. In response, the Canadian Cancer Society and the Non-Smokers' Rights Association led a campaign to block the RJR-Macdonald tobacco company's intrusion into amateur sport.

Skiers reject sponsor

The CSA is made up of several disciplines. The Cross Country and Biathlon sections rejected the Macdonald promotion. Then, on Oct 22nd, after appeals from the health groups, the high profile Alpine section voted almost unanimously to reject the sponsor. With the Alpine division out, the deal was in trouble.

The Alpine rejection was hidden from the media until Oct 30th when the CSA Executive overruled the Alpine section. Despite Alpine's decision not to touch tobacco money, the downhillers were coerced back into line.

CSA plan a "betrayal"

Both the International Ski Federation and the United States Ski Association have policies which reject tobacco industry sponsorship. Yet the CSA pushes blindly on. **Epidemiologist Dr. David Stewart says the plan *"is a betrayal to the thousands of young skiers who look to the CSA to promote healthy competition and not to use the sport as a vehicle to promote disease."***

Dr John Read, professor of medicine and father of two national ski champions, says the plan is "reprehensible" and will "use our young racers to promote smoking." The average age of the alpine competitors for girls is 16, for boys, 17-18.

Can the Plan be Stopped?

You bet it can. But only with your support. Take a look at the issues.

1 The "Refuse the Money and Send the Skiers Home" Scare Tactic

Will the ski team be sent home or the championships cancelled? Not at all. The ski team will not be affected on the international circuit. And the Canadian championships have been run in the past without sponsors. Some may believe that if a "no sponsor – no championships" option is presented, the public will support taking the tobacco money. However, there are many options open to the CSA without cancelling these events.

2 The "Signed Contract" Red Herring

Some CSA folks say the Association can't get out of this promotion because a letter of intent has been signed. This argument is a red herring. Prior to the weekend of Oct 29th, statements were made **publicly** that it was too late to break from the sponsor. Yet on Oct 30th, the CSA Executive Committee had a **private** marathon debate to decide whether or not to do exactly that.

> *"I am profoundly distressed that the issue of the tobacco industry using skiing to promote its wares has again raised its ugly head,"* Kell Antoft said Monday in Halifax in a letter to Canadian Ski Association president Greg Hilton.
>
> *Antoft said he thought the issue was resolved in the late 1960s when delegates to association conventions questioned the morality of allowing Du Maurier to sponsor the World Cup event in Canada.*
>
> *At the 1969 convention, the majority of delegates told the national board of the association "to sever what was an embarrassing and distasteful alliance," he said.*
>
> *Antoft said raising money and winning medals must take second place to the goal of promoting health. "We must not prostitute our principles – no matter how enticing the inducement."*
>
> Kell Antoft, Former President
> Atlantic Division, Canadian Ski Association
> quoted by the *Vancouver Sun*

Even if the contract is signed, it is not too late to abandon the deal. If the CSA asks to be let out, Macdonald tobacco would be hard pressed to say "no". Can you imagine the Export A folks saying *"if you don't take our money, we'll sue"*? Can you believe that a tobacco company, attempting to present itself as a good corporate citizen, would force the issue if the CSA said *"Sorry but we've had second thoughts. We'd rather not be associated with the promotion of disease"*?

3 The "Sports Sponsorship is Not Tobacco Advertising" Myth

Considering the megabucks which will change hands, the industry plans to get something for its trouble. First the sponsorship buys social acceptability and legitimacy for a product which kills and which pollutes our indoor environment.

Sports sponsorship buys an association with health and fitness, a relationship which is hard to come by for this industry.

The suggestion that sports sponsorship is not tobacco advertising and that the CSA is not involved with the promotion of smoking is *"patent nonsense and the public will know that"* said Emerson Foote. Foote is the former president and chairman of the board of the world's second largest advertising agency.

4 The Highly Principled "Tobacco is a Legal Product. Take the Money and Run" Argument

Tobacco is only legal because when the product was first marketed, people did not know what we know now. The legality of the product should not give the tobacco industry any protection from the social derision and ostracism that it so richly deserves.

Many people know ways to increase their incomes which are legal but which do not meet community standards. Many have ethical standards which are higher than those narrowly defined by law. And many apply these standards rather than court public disfavour. The CSA should be no exception. It has a responsibility to the community which it must recognize.

5 Can Another Sponsor be Found?

We think so. Given the publicity that this deal has attracted, any astute corporation which steps forward as a sponsor will reap a media bonanza and much goodwill.

6 Point a Fine Organization in the Right Direction

The Canadian Ski Association is a good organization composed of many fine volunteers. It has raised the quality of skiing in Canada and produced many fine champions. We believe that the vast majority of CSA members are thoughtful people who, when given the facts, will recognize that the long term best interests of the CSA will be hurt by this sponsorship. This ad is not anti-CSA. It is anti-tobacco industry sponsorship. And that's an important distinction.

How to Help

1. Out of one pocket, Canadians give donations to health organizations to deal with the smoking problem. Out of their other pocket they send taxes to the federal government where, through Sport Canada, it funds the Canadian Ski Association. Unfortunately, working with your tax dollars, the CSA is now firming up a deal which will undermine the work of the health groups.

Write the Hon. Monique Begin, Minister of Health and your Member of Parliament, House of Commons, Ottawa, Ontario K1A 0A6. Ask them to send a strong message to the Canadian Ski Association, publicly or behind closed doors. The message should be clear: ***"Health and Welfare Canada or Sport Canada co-sponsorship of a fitness organization with a tobacco company is unacceptable. Rethink the RJR-Macdonald deal or lose your sponsorship from public funds."***

We believe that many Health and Welfare Canada and Sport Canada Ministers and officials are less than pleased with this deal. Help give them the support necessary in order to send the CSA a strong message.

2. Ask the editor of your local paper for a discussion of this issue somewhere other than just in the sports section. This is a hard news medical story of interest to the whole community.

3. Join the **NON-SMOKERS' RIGHTS ASSOCIATION**. The NSRA is a national, non-profit, environmental health organization with national and international precedents to its credit. Major health reports now call for more advocacy on the tobacco issue and that's what we do best. Your membership will help publish ads like this in other papers until the CSA walks away from Export A.

4. Send a separate donation to the **SMOKING AND HEALTH ACTION FOUNDATION**. We can't use it to pay for ads but it will go to research and public education activities. We'll send you a receipt for tax purposes. Reg. #0459123-59-13

Many thanks to the following endorsers:

David Nostbakken, PhD, *Chairman*
  Fifth World Conference on Smoking and Health
Bernard Lalonde, MD, Ottawa
Gordon Kroll, *President*
  Qualitype Co., Toronto
Ken Read
  Canadian Downhill Champion 1975-1980
Kurt Baumgartner, *Secretary General*
  Fifth World Conference on Smoking and Health
William F Forbes, PhD, *President*
  Canadian Council on Smoking and Health
Kell Antoft, Halifax
  Co-founder Nancy Green Ski League
Emerson Foote
  Carmel, New York
Ken MacKenzie, MD, Victoria
John P Schaman, MD, Breslau
Seymour Herlin, MD, Toronto
Pat McConkey, MD, Vancouver
G David Millman, MD, Scarborough
Barry Vasler, MD, North Vancouver
James Jarvis, MD, Ottawa
Herman Hugenholtz, MD, Ottawa
David R Ellis, MD, Nanaimo
Jack Micay, MD, Toronto
John Gordon, MD, Ottawa
Phil Hamilton, MD, Ottawa
Susan Murdoch, BPHE, Ottawa
Cathie Ross, MBA, Ottawa
Roger Hills, LLB, Ottawa
Marilyn Mills, BSc (OT), Ottawa
T C Barber, LLB, Ottawa
Carlo Bos, MD, Peterborough

Greg Dargavel, MSc, Ottawa
Robert Rivington, MD, Ottawa
Alf Juergensen, Halifax
Sidney Katz, MSW, Toronto
From the University of Calgary
  John Read, MD
  John Hunter, MD
  G D Carson, MD
  Gordon Ford, MD
  Keith Burgess, MD
From the University of Toronto
  J David Stewart, MD
  Gerald D Hart, MD
  Marja Soots, MD
  Charles Godfrey, MD
From the University of Ottawa Heart Institute
  Andrew Pipe, MD
  Jacques D Aslous, MD
  I G McKibbin, MD
  John Atkinson, MD
  Michael Bourke, MD
  James Robblee, MD
  Gilles De La Salle, MD
  Pierre Bedard, MD
  William Goldstein, MD
  Arvind Koshal, MD
  Lyall Higginson, MD
  W L Williams, MD
  Charles B Cattran, MD
  Brian Morton, MD
  Maurice Brais, MD
  James Calvin, MD

THANKS TO QUALITYPE CO., TORONTO, FOR DONATING THE TYPESETTING, ART AND DESIGN

Organizations which have taken a stand on the sponsorship include:*
Canadian Association of Sport Sciences
Canadian Cancer Society　**Canadian Council on Smoking and Health**
Canadian Heart Foundation　**Non-Smokers' Rights Association**

The Non-Smokers' Rights Association has published this ad as a contribution to preventive medicine. The long term preventive approach for the clean indoor air movement is the reduction of smoking.

* The Canadian Lung Association (the Christmas Seal folks) declined to join the groups above by speaking out publicly against this sponsorship.

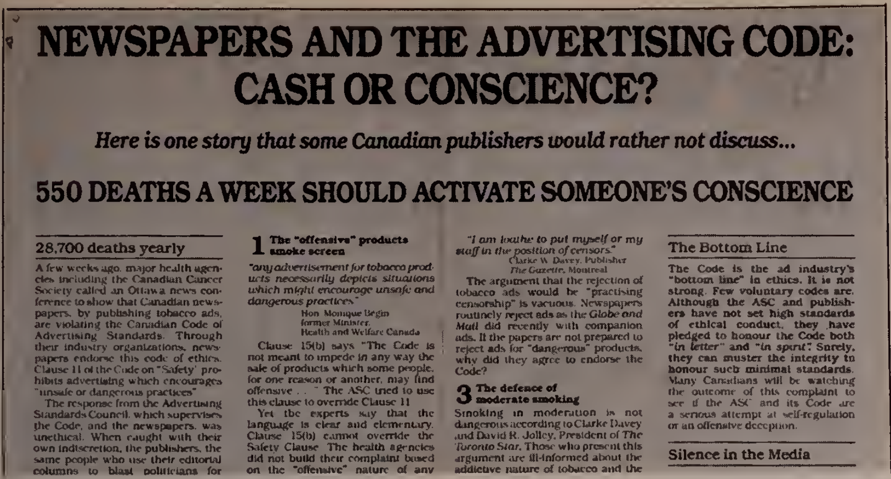
TO THE NSRA

☐ I support your efforts and encourage the CSA to reject this sponsorship.
☐ Please send me 1984 membership in the NSRA. I enclose $19 payable to the NON-SMOKERS' RIGHTS ASSOCIATION. Please send me a membership kit and your pamphlet, "The Case Against Tobacco Ads".
☐ I enclose a donation of $________ payable to the SMOKING AND HEALTH ACTION FOUNDATION. Send me a receipt for tax purposes.
☐ Please send me more information on the NSRA.

Name ____________________
Address ____________________ Apt ________
City ____________ Prov ________ Code ________

Mail to
NON-SMOKERS' RIGHTS ASSOCIATION
SUITE 201, 455 SPADINA AVENUE, TORONTO, ONTARIO M5S 2G8
GARFIELD MAHOOD, EXECUTIVE DIRECTOR · PHONE 416 595 1538

FIGURE 1. Advertisement purchased by the Canadian Non-Smokers' Rights Association, in *The Globe and Mail* (Toronto), Nov 12, 1983.



NEWSPAPERS AND THE ADVERTISING CODE: CASH OR CONSCIENCE?

Here is one story that some Canadian publishers would rather not discuss...

550 DEATHS A WEEK SHOULD ACTIVATE SOMEONE'S CONSCIENCE

28,700 deaths yearly

A few weeks ago, major health agencies including the Canadian Cancer Society called an Ottawa news conference to show that Canadian newspapers, by publishing tobacco ads, are violating the Canadian Code of Advertising Standards. Through their industry organizations, newspapers endorse this code of ethics. Clause 11 of the Code on "Safety" prohibits advertising which encourages "unsafe or dangerous practices"

The response from the Advertising Standards Council, which supervises the Code, and the newspapers, was unethical. When caught with their own indiscretion, the publishers, the same people who use their editorial columns to blast politicians for

1 The "offensive" products smoke screen

"any advertisement for tobacco products necessarily depicts situations which might encourage unsafe and dangerous practices"

Hon Monique Bégin
former Minister,
Health and Welfare Canada

Clause 15(b) says "The Code is not meant to impede in any way the sale of products which some people, for one reason or another, may find offensive . . ." The ASC tried to use this clause to override Clause 11

Yet the experts say that the language is clear and elementary. Clause 15(b) cannot override the Safety Clause. The health agencies did not build their complaint based on the "offensive" nature of any

"I am loathe to put myself or my staff in the position of censors."

Clarke W. Davey, Publisher
The Gazette, Montreal

The argument that the rejection of tobacco ads would be "practising censorship" is vacuous. Newspapers routinely reject ads as the *Globe and Mail* did recently with companion ads. If the papers are not prepared to reject ads for "dangerous" products, why did they agree to endorse the Code?

3 The defence of moderate smoking

Smoking in moderation is not dangerous according to Clarke Davey and David R. Jolley, President of *The Toronto Star*. Those who present this argument are ill-informed about the addictive nature of tobacco and the

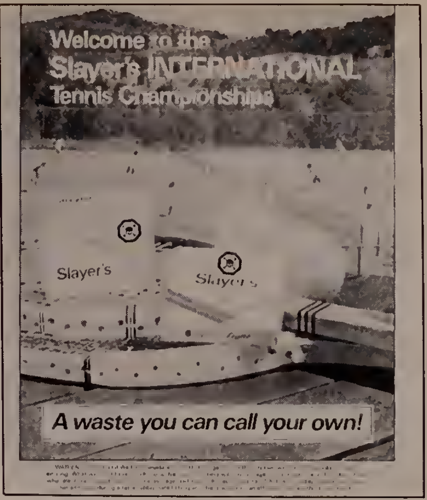
The Bottom Line

The Code is the ad industry's "bottom line" in ethics. It is not strong. Few voluntary codes are. Although the ASC and publishers have not set high standards of ethical conduct, they have pledged to honour the Code both "*in letter*" and "*in spirit*." Surely, they can muster the integrity to honour such minimal standards.

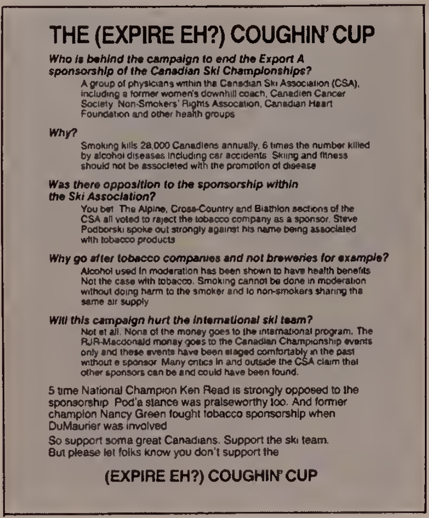
Many Canadians will be watching the outcome of this complaint to see if the ASC and its Code are a serious attempt at self-regulation or an offensive deception.

Silence in the Media

FIGURE 2. Advertisement purchased by the Canadian Non-Smokers' Rights Association, in *The Globe and Mail* (Toronto) Oct 10, 1984.



NSRA's protests of cigarette-sponsored sporting events that are covered by the mass media, such as the Player's (Imperial Tobacco) tennis matches (top) and the Export A (RJ Reynolds-Macdonald) ski championship (bottom), have included public demonstrations and the distribution of satirical literature pointing out that such sponsorships violate the tobacco industry's own advertising and promotion code.



THE (EXPIRE EH?) COUGHIN' CUP

Who is behind the campaign to end the Export A sponsorship of the Canadian Ski Championships?

A group of physicians within the Canadian Ski Association (CSA), including a former women's downhill coach, Canadien Cancer Society Non-Smokers' Rights Assocation, Canadian Heart Foundation and other health groups

Why?

Smoking kills 28,000 Canadiens annually, 6 times the number killed by alcohol diseases including car accidents Skiing and fitness should not be associeted with the promotion of disease

Was there opposition to the sponsorship within the Ski Association?

You bet The Alpine, Cross-Country and Biathlon sections of the CSA all voted to raject the tobacco company as a sponsor. Steve Podborski spoke out strongly against his name being associated with tobacco products

Why go after tobacco companies and not breweries for example?

Alcohol used in moderation has been shown to have health benefits Not the case with tobacco. Smoking cannot be done in moderation without doing harm to the smoker and to non-smokers sharing tha same air supply

Will this campaign hurt the international ski team?

Not et all. Nona of the money goes to the international program. The RJR-Macdonald monay goes to the Canadian Championship events only and these events have been staged comfortably in the past without e sponsor Many critics in and outside the CSA claim that other sponsors can be and could have been found.

5 time National Champion Ken Read is strongly opposed to the sponsorship Pod'a stance was praiseworthy too. And former champion Nancy Green fought tobacco sponsorship when DuMaurier was involved

So support soma great Canadians. Support the ski team.
But please let folks know you don't support the

(EXPIRE EH?) COUGHIN' CUP

Other publications drop cigarette ads

Citing the precedent of the *Whig-Standard*, publishers of a second Canadian daily newspaper, *The Recorder and Times*, Brockville, Ontario, announced on April 12, 1985, that they would no longer accept cigarette advertising. In a statement to readers, publishers Perry S. Beverley and Hunter S. Grant credited Garfield Mahood, director of the Non-Smokers' Rights Association, with providing the information and arguments that led to the decision to drop tobacco advertising:

> Mahood can drown you in statistics supporting his cause, a war against smokers that is relentless. Such is his persistence that Mahood is viewed by some publishers more as a burr beneath the saddle than a white knight on his charger.
>
> Burr or not, Mahood's volume of information on this topic confirms that smoking is indeed, as we said in the beginning, a deadly habit. It has killed thousands and cost millions.
>
> To continue to promote tobacco use, whether or not that use is legal, flies in the face of our conscience, more than whispers of hypocrisy, and is contrary to our own standards.
>
> Because of this, effective Monday, April 15, the pages of *The Recorder and Times* will no longer carry tobacco advertising.

In 1974 the publishers had banned smoking in the workplace. After evaluating time lost through illness by their 66 employees, the newspaper found that 40.4% of non-smokers lost time through illness, compared to 84.2% of smokers. In the April 12 statement the publishers extended the ban on smoking to cover the entire building. The statement concluded, "We appreciate that these decisions are but a small gesture; however we join a growing throng who agree smoking wastes health, time and money, and has absolutely no redeeming value."

The *Canadian Geographic*, a magazine with a paid circulation of 135,000, has also announced it will no longer accept tobacco advertisements.

such thing as commercial freedom of speech. "That argument about censorship is a red herring. It just sounds good when publishers put it out."

A pamphlet on the case against tobacco advertising prepared by the NSRA addresses the issue: Some media managers reject advertisements because they consider them to be "inappropriate," "offensive," or "of questionable taste." If this is true, advertisements that promote Canada's most preventable medical problem should top the list. The pamphlet is one of numerous slick-looking, research-based information packages distributed to 4,000 members of the association across Canada.

David Crombie, a former federal minister of health, called NSRA "the most impressive and intelligent lobby I have ever known. The information they supplied was reliable and complete. . . . They showed a talent for compromise. They didn't waste my time." In an interview with a Canadian marketing magazine, one tobacco industry representative said, "We won't start a public debate with Gar Mahood. We respect his point of view, but we don't agree with it."

Those words bring a smile to Mahood's face. "The fact is if they really believed that I was the bad guy—curtailing individual freedoms and all the other things that they talk about in their little propaganda sorties—they wouldn't have respect for Garfield Mahood. But deep down, they know that what we're trying to do is right. That gives the health community a tremendous opportunity to move. In order to have an issue," Mahood added, "you have to have the research on your side and you have to have justice on your side. We have both those things and the tobacco industry doesn't."

Angela Mangiacasale

How smoking promotes hunger

The link between smoking and hunger is beginning to be explored. A study in Bangladesh shows that smoking promotes hunger in low income families (*Lancet* 1981; 1:1090–1093). This and other national studies, when they are eventually undertaken, could prove to be of such importance that the smoking and health debate would take on a new dimension. The doctor behind the Bangladesh study believes that the nutrition-mediated effects of smoking could be even more serious than the direct health risks of smoking. There are hints that the tobacco industry is alarmed that more research into the link between smoking and hunger will cast the industry in a damaging light and hasten its decline.

The debate over tobacco has concentrated on the damage to the health of smokers and the effect on non-smokers who are forced to "passively smoke" in the presence of smokers. Also attracting attention are the millions of acres of land used for tobacco plants that could grow food crops, and the enormous damage that tobacco does to Third World environments because of its demand for trees (*NY State J Med* 1983; 83: 1310-1311). Despite the overwhelming medical and scientific evidence that smoking damages health, the tobacco industry still sells its products—making up for lost sales in western countries by selling more to the developing continents of Africa, Asia, and Latin America.

The health risks of smoking are not as well known to people in the developing world as they are in the West, and the tobacco industry is naturally not keen to provide information. The link between smoking and hunger, which is now being added to the tobacco controversy, will call into question the morality of an industry that promotes cigarette sales and will increase pressure for action against the industry's practices in both western and developing countries.

Growing Problem

It has long been assumed that when poor families buy cigarettes they buy less food. But what has been lacking is a rigorous analysis of the precise effects of cigarette sales on nutrition. In Bangladesh, this has now been remedied, although work still needs to be done in other developing countries.

Dr Nicholas Cohen—who works in Bangladesh (PO Box 6066, Gulshan, Dacca 12, Bangladesh)—has studied what happens to the nutrition of children in low income families when their parents smoke (*Lancet* 1981; 1:1090–1093). His conclusion is stark and pertinent: "If, as seems likely, expenditure on smoking means that they get less food, then the survival of a large number of children is being seriously endangered."

Cigarette sales have increased rapidly in Bangladesh, more than doubling over the last 10 to 15 years.[1] Some 1,500 million cigarettes and 3,000 million biri (hand-rolled thin folds of coarse tobacco) are made in the country each month. A subsidiary of the giant British American Tobacco company turns out 14 million cigarettes a day from its Dacca factory. An unpublished survey of two villages by Cohen and Measham showed that 67% of adult men smoke. Cohen points out that cancer of the lung is already the third most common cancer among men in Bangladesh. "Ominously, and surprisingly, carcinoma of the lung already rates third of carcinomas of the oral cavity and larynx as a site for cancer in males."

Bangladeshi street scene: Photographs Tom Learmonth

Bangladeshis are coming under increased pressure to smoke, because the habit is associated with upward mobility, which cigarette advertising exploits. Such advertising is extensive and makes much use of sexual themes; nowhere, either on promotional material or cigarette packets, are there warnings about the health hazards of smoking. To people on low incomes, smoking can be attractive, providing them with a brief escape from poverty and a visual display of unattainable social position.

In their survey, Cohen and Measham look at the extent to which cigarette smoking causes a reduction in the money that the poorest families have available to spend on food and other essentials. Sixteen percent of Bangladeshi households have a monthly income below $12, and it is the children of these families that are especially vulnerable if one or both parents smoke. (Men tend to be dominant in deciding how household income is spent.) These families spend about 70% of their income on food; after the purchase of other essentials such as clothing and health care, there is no money in reserve. If any of their income is diverted to other purchases, they spend less on the basic essentials needed for survival.

Cigarettes over Food

Should a parent of one of these households smoke five cigarettes a day, then their children suffer. The cigarettes would cost $1.30 a month; that is more than 10% of the household's monthly income. Assuming that 70% of the $1.30 would have been spent on food, and that 80% of food expenditure is for rice, then at current rice prices, the monthly rice purchase for the family could be 2.25 kg less. This amounts to a deficit of about 8,000 calories and 150 g of protein—nearly a quarter of the monthly maintenance energy requirements (at 100 calories/kg/day) of a 12-kg child. Reductions of food intake of this order could lessen the prospects of survival for many children of poor families, especially children under five years old.

Children in Bangladesh.

The subsistence of many young Bangladeshi children is already marginal, largely because they have insufficient food. Any reduction could tip the balance against them and cause ill health and even death. According to a survey of rural Bangladesh, about half of the children under five years old have weight-for-age levels between 60% and 74% of the Harvard standard (*Lancet* 1978; 1:1247-1250). In his paper in *The Lancet*, Cohen made a chilling prediction: "If the weight of even a quarter of 1% of these children declined by a further 10%, as a result of income being used for smoking rather than for food, then each year the prospects of survival of some 18,000 children would be halved." He acknowledges that it is impossible to be precise about the quantitative effects of nutritional shortfalls, but adds, "should these estimations be anywhere near correct, the nutrition-mediated effects of smoking in terms of chronic under-nutrition, as well as survival, are likely to be far more important than the direct consequences of smoking on health."

Bangladesh has a population of about 95 million; the developing world as a whole has almost 3 billion people. If figures on the nutrition-mediated effects of smoking prove to be much the same in other developing countries as they are in Bangladesh—which seems likely—then worldwide the number of children whose survival is threatened when their parents smoke just a few cigarettes a day is over half a million.

Therefore, tobacco must be added to the other factors that cause food shortages, child disease, and mortality in the Third World. With hunger in the world growing worse, and children particularly at risk, the increase in smoking in poor countries appears particularly unwelcome.

WHO HOLDING BACK?

Faced with the scandal of young and innocent people falling victims to smoking, the World Health Organization (WHO) might be expected to want to expose this aspect of the dismal tobacco saga. Surveys similar to Dr Cohen's in Bangladesh have not yet been done in other countries, and it seems that the WHO has done nothing to promote them. More studies would clearly be helpful, as they provide increased statistical evidence. Cohen told the *Journal* that a few years ago he approached the WHO to suggest that the organization help to finance such surveys, as part of its Program on Smoking and Health, launched in May 1980. The WHO, he maintains, seemed keen on the idea, but its enthusiasm appeared to wane. Cohen points to the possibility that the WHO came under pressure from the tobacco industry not to finance surveys that would in all probability be damaging to the future of the industry. Under constant threat, the industry works behind the scenes to exert pressure on key policy makers and officials; it may have suggested to the WHO that any financing for nutrition and smoking surveys might call its objectivity into question (an invalid argument but one that the industry might try).

Dr Roberto Masironi, coordinator of the WHO's Smoking and Health Program, claims that WHO has contributed financially to "certain smoking-related research projects in developing countries, but no definite proposals along the lines of Dr Cohen's study have been submitted." The question still remains, however, as to why the WHO is not taking the initiative and making it known that it would be willing to consider financing such studies. Furthermore, it might be asked whether the WHO is as committed as it might be to its own smoking and health program.

In his recent book *Smoke Ring: The Politics of Tobacco* (London, Bodley Head, 1984), Peter Taylor points to signs that the WHO "might be beginning to conserve its energies on smoking and health." Taylor says that in 1982 Dr Masironi was told that he was working too hard on smoking and that he should only give it half his time.

A WHO memorandum that outlined budget proposals for 1983 to 1984 stated that after lengthy discussions, the organization had decided to maintain smoking and health activities at a low level. "The WHO," says Taylor, "was beginning to adopt the more neutral position which the tobacco industry wanted." He goes on to say that if the WHO could not be drawn into the smoke ring (the protective circle of economic and political interests that has kept the power of the tobacco industry intact), at least its attacks on the industry might now be blunted.

But the position of the WHO could be more serious than Taylor proposes. It is a mystery why the WHO is only working half time on smoking and health, when by its own estimates, smoking-related diseases are reaching epidemic proportions in developing countries, and smoking is the world's largest single preventable disease.

Storefront on Bangladeshi street corner.

With or without the help of the WHO, studies similar to Cohen's could be done by universities, doctors, and medical researchers. Cohen's work opens up an important new avenue—it shows that smoking can affect health and can kill in more ways than one. If the smoking epidemic in the developing world is to be diminished, and if hungry children are not to be denied the food they need to survive, then a study into smoking and hunger is warranted for every country. As more information becomes available on how smoking threatens the survival of children, action by governments to discourage smoking becomes more likely.

John Madeley

Mr Madeley, a journalist and broadcaster in the United Kingdom, is a regular contributor to the Journal.

American Nurses' Association passes resolutions for action on smoking issues

The House of Delegates of the American Nurses' Association (ANA) voted overwhelmingly at its June 1984 biennial convention to support resolutions directed toward action to control smoking. The resolutions were initiated because of concern about smoking-related diseases, and especially their increasing incidence in women. According to a survey published in 1980 by the Department of Health and Human Services, nurses comprise the largest group of health professionals; 97% of the current 1.7 million nurses are women.

This is not the first time that the ANA has taken action against smoking. It pledged support of other groups concerned with the health hazards of smoking in 1968 and resolved to support the Non-Smoker's Bill of Rights in 1974. Since 1974 the ANA Board of Directors has banned smoking in program sessions of the ANA conventions. In May 1984 the Board of Directors announced that their investment guidelines would include the provision that "no stocks shall be purchased in the tobacco industry" (*American Nurse*, May 1984, p 18).

The new resolutions represent a further commitment. They address three major issues: personal smoking behavior of nurses; research and smoking-related illness; and policy formulation. The five resolutions are as follows:

> Resolved, that the American Nurses' Association formally recognize the intensity of the problem of smoking in the nation and reflect this awareness with action to address the issue, and be it therefore
>
> Resolved, that the American Nurses' Association support activities directed toward reducing smoking among nurses, and be it therefore
>
> Resolved, that the American Nurses' Association identify sources of funding and encourage nurses to be participants in research involving smoking behavior, and be it therefore
>
> Resolved, that the American Nurses' Association engage in the formulation of policies and use its political power to influence policy makers toward smoking control efforts, and be it therefore
>
> Resolved, that the American Nurses' Association assist nurses in increasing their awareness that smoking is a major threat to the health of all individuals.

DEVELOPING THE RESOLUTIONS

There was much support for the resolutions. They had originally been drafted by three nurses (Ann Hall Harvey, RN, ScD, School of Hygiene, Johns Hopkins University; Ingrid Swenson, RN, DPh, University of North Carolina at Chapel Hill; and this author) who are involved in smoking research and who had attended the Fifth World Conference on Smoking and Health. The nurses sent drafts of the resolutions to their respective state associations and to nurses around the country. The resolutions were then sent to the ANA Reference Committee by the New Jersey State Nurses' Association and co-sponsored by the Maryland and Montana Nurses' Associations. The ANA Division of Community Health Nursing Practice was impressed by the proposal and asked to be a co-sponsor. The Minnesota Nurses' Association formally endorsed the proposal. At the convention, testimony given at hearings on the resolutions was generally supportive. The Reference Committee subsequently recommended that the House of Delegates adopt the resolutions because they specifically address smoking issues for nurses.

The Saturday Evening Post 1932

The resolutions submitted to the ANA were accompanied by a detailed outline of suggested activities. Although funds are budgeted for the implementation of the resolutions, the affirmative vote of the delegates on any resolution does not automatically guarantee that the action plan will be carried out or that the expenditures will be made exactly as suggested in the treasurer's proposed budget. A figure of $27,000 was recommended in the proposed budget for implementation of the resolutions.

RECOMMENDED ACTIONS

Three goals concerned with the personal smoking behavior of nurses are proposed in the recommended plan of action. The proportion of nurses who smoke should be reduced to below 25% by 1990, consistent with national goals as defined by the United States Public Health Service. In order to accomplish this, the ANA is encouraged to sponsor programs to apprise nurses of current findings regarding smoking and health. Support networks for nurses attempting to stop smoking are also proposed.

The following recommendations are included under the topic of research; the ANA is asked to identify sources of funding for (and encourage nurse participation in) various research projects: a study on the smoking behavior of nurses and student nurses; surveys of educational programs for nurses to determine the type, focus, and content of learning experiences related to the risk of smoking; and preparation of guidelines to assist faculty in the development of educational programs relative to smoking. The plan of action also recommends the development of mechanisms for reporting and disseminating research findings to nurses through publications and conferences. The proposed budget includes funds for the establishment of a clearinghouse for information and for the development of continuing education programs.

Policy formulation is another important area targeted by the action plan. In a speech before the House of Delegates, Department of Health and Human Services Secretary Margaret M. Heckler emphasized that nurses are a sufficiently large group to attract the attention of policy makers in government. In recognition of this fact, the action plan for smoking issues urges that the ANA lobby for legislation that would provide regular increases in tobacco taxes; complete bans on tobacco product advertising; a ban on the sale of tobacco in health care facilities, educational facilities, drug stores, and food stores; mandatory requirements for self-extinguishing cigarettes; a ban on the sale of cigarettes to minors; and more explicit health warnings on all cigarette packages. Collaboration with such groups as the Coalition on Smoking OR Health is suggested; this activity has also been provided for in the proposed budget. Nurses are also encouraged to participate in the development and implementation of policies to assure smoke-free environments in public places, especially in health care facilities.

The successful implementation of this ambitious plan for action depends upon the willingness of many nurses to commit time and resources to the suggested activities. The formal resolutions of the ANA provide the impetus for collective and individual action on this critical health issue.

Elizabeth Wilson, RN, EdD

Ms Wilson is an assistant professor in the Division of Allied Health/Nursing at Bergen Community College, Paramus, NJ 07652.

Two physicians provided major assistance in promoting the proposed ANA resolutions among nurses. Leland Fairbanks, MD, *Indian Health Service, Phoenix, AZ helped to disseminate copies of the resolutions prior to the convention, and Paul Richburg,* MD, *Dallas, TX helped publicize the news of the adoption of the resolutions.*

At its October 1984 annual meeting the New York State Nurses' Association supported the ANA resolutions and banned smoking at all of its future business and program meetings.

—Editor

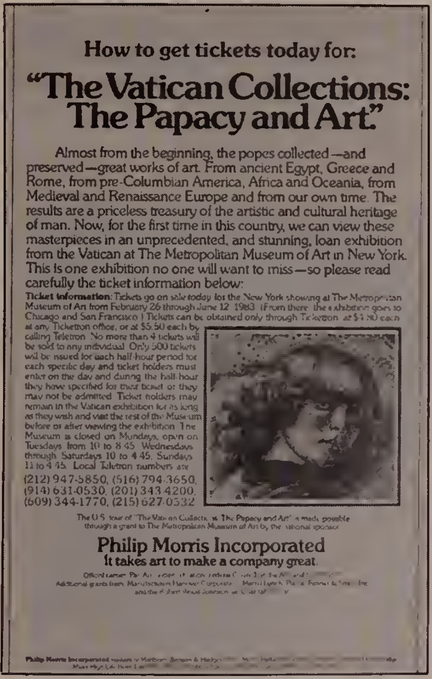
How tobacco companies have found religion

In 1983 Philip Morris Inc, America's largest cigarette manufacturer, attached itself to the prestige and influence of the Vatican. As principal sponsor of the first US tour of "The Vatican Collections: The Papacy and Art," the New York-based tobacco company was able to attain a close association in the public eye with major religious and cultural organizations. The year-long exhibition was seen by two million people at the Metropolitan Museum of Art in New York, the Art Institute of Chicago, and the MH de Young Memorial Museum in San Francisco. Along with prominent news coverage and feature articles, numerous advertisements for the exhibition appeared in the editorial and arts pages of *The New York Times, The Los Angeles Times, Chicago Sun-Times, Chicago Tribune,* and other newspapers—paid for by Philip Morris (Fig 1). Each advertisement listed the brands of cigarettes manufactured by the company. For its $3 million sponsorship—a fraction of the company's annual cigarette advertising budget in excess of $500 million—Philip Morris reaped enormous favorable exposure in the mass media, including photographs of cigarette company executives alongside church dignitaries, museum officials, patrons of the arts, politicians, and even the First Lady.

In 1984 Philip Morris sponsored the national tour of "The Precious Legacy," which displayed Jewish artwork confiscated from individuals killed in the Nazi holocaust (Fig 2). The tobacco-subsidized exhibition broke attendance records at the San Diego Museum of Art and other stops on its tour.

Hear No Evil . . .

The acceptance by Catholic, Jewish, and other religious groups of donations from cigarette manufacturers has increased in recent years. The morality of this relationship has gone virtually unquestioned in religious circles and in the mass media. One cleric who had forgotten the Philip Morris connection is Father Robert Drinan, a professor of law at Georgetown University who introduced several antismoking measures while serving in the US House of Representatives in the 1970s. "I thought religions were supposed to be against smoking," he told the *Journal.*


How to get tickets today for:

"The Vatican Collections: The Papacy and Art."

Almost from the beginning, the popes collected—and preserved—great works of art. From ancient Egypt, Greece and Rome, from pre-Columbian America, Africa and Oceania, from Medieval and Renaissance Europe and from our own time. The results are a priceless treasury of the artistic and cultural heritage of man. Now, for the first time in this country, we can view these masterpieces in an unprecedented, and stunning, loan exhibition from the Vatican at The Metropolitan Museum of Art in New York. This is one exhibition no one will want to miss—so please read carefully the ticket information below:

Ticket information: Tickets go on sale today for the New York showing at The Metropolitan Museum of Art from February 26 through June 12, 1983. (From there, the exhibition goes to Chicago and San Francisco.) Tickets can be obtained only through Ticketron, at $3.50 each at any Ticketron office, or at $5.50 each by calling Teletron. No more than 4 tickets will be sold to any individual. Only 500 tickets will be issued for each half-hour period for each specific day and ticket holders must enter on the day and during the half-hour they have specified for their ticket or they may not be admitted. Ticket holders may remain in the Vatican exhibition for as long as they wish and visit the rest of the Museum before or after viewing the exhibition. The Museum is closed on Mondays, open on Tuesdays from 10 to 8:45, Wednesdays through Saturdays 10 to 4:45, Sundays 11 to 4:45. Local Teletron numbers are (212) 947-5850, (516) 794-3650, (914) 631-0530, (201) 343-4200, (609) 344-1770, (215) 627-0532.

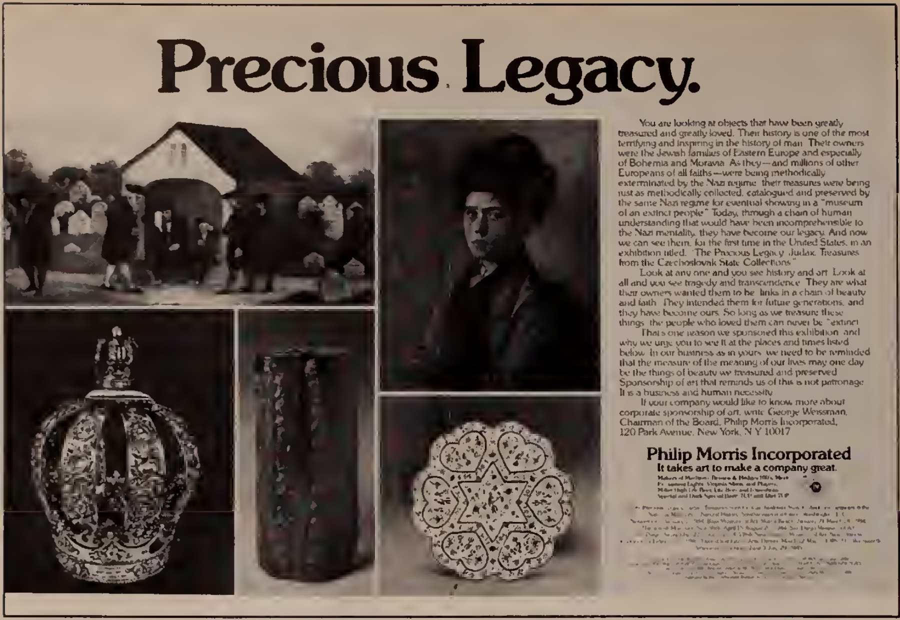
The U.S. tour of "The Vatican Collections: The Papacy and Art" is made possible through a grant to The Metropolitan Museum of Art by the national sponsor

Philip Morris Incorporated

It takes art to make a company great.

FIGURE 1. Advertisements for Philip Morris-sponsored Vatican art tour appeared in *The New York Times, The Washington Post* and other newspapers and newsmagazines. Names of the company's cigarette brands are listed at the bottom of this ad. The Surgeon General's warning does not appear.

One of the few journalists who have questioned the ethics of the acceptance by religious and cultural institutions of tobacco company gifts is Roy Larson, former religion editor of the *Chicago Sun-Times.* Following the announcement of the sponsorship of the Vatican art exhibition, Larson charged in his column (*Chicago Sun-Times,* Nov 27, 1982:18) that the Art Institute of Chicago would become "a crude outpost in Marlboro Country. For centuries," wrote Larson, "the Roman Catholic Church has been in the business of forming opinions. Generally, it has taken a 'pro-life' stance. Witness its current campaigns against abortion and nuclear war. That's what makes the Vatican look so inconsistent when it lends its vast prestige to the Philip Morris firm, whose president in 1971 stated its rationale for gifts to the arts by saying, 'It's a lot cheaper than taking out ads saying how great we think we



Precious Legacy.

You are looking at objects that have been greatly treasured and greatly loved. Their history is one of the most terrifying and inspiring in the history of man. Their owners were the Jewish families of Eastern Europe and especially of Bohemia and Moravia. As they—and millions of other Europeans of all faiths—were being methodically exterminated by the Nazi regime, their treasures were being just as methodically collected, catalogued and preserved by the same Nazi regime for eventual showing in a "museum of an extinct people." Today, through a chain of human understanding that would have been incomprehensible to the Nazi mentality, they have become our legacy. And now we can see them, for the first time in the United States, in an exhibition titled, "The Precious Legacy: Judaic Treasures from the Czechoslovak State Collections."

Look at any one and you see history and art. Look at all and you see tragedy and transcendence. They are what their owners wanted them to be: links in a chain of beauty and faith. They intended them for future generations, and they have become ours. So long as we treasure these things, the people who loved them can never be "extinct."

That's one reason we sponsored this exhibition, and why we urge you to see it at the places and times listed below. In our business as in yours, we need to be reminded that the measure of the meaning of our lives may one day be the things of beauty we treasured and preserved. Sponsorship of art that reminds us of this is not patronage. It is a business and human necessity.

If your company would like to know more about corporate sponsorship of art, write George Weissman, Chairman of the Board, Philip Morris Incorporated, 120 Park Avenue, New York, N Y 10017

Philip Morris Incorporated

It takes art to make a company great.

FIGURE 2. Advertisement in *Newsweek* for Philip Morris-sponsored Judaica art tour.

are.' "

Larson told the *Journal* there was almost no reaction to his column, "except one guy almost attacked me at a cocktail party," and one letter to the editor, from a nurse, was published in support of Larson. No religious authority challenged or commended him: "Catholic officialdom was silent," he said.

When the Vatican exhibition opened, *The New York Times* ran a short article (*New York Times* Feb 28, 1983:34) under the headline "A ceremonial picketing," about a group of 35 physicians, medical students, and parents protesting tobacco company sponsorship of the Vatican exhibition outside the Metropolitan Museum of Art (Fig 3). The article referred to Monsignor Eugene V. Clark, a former New York Archdiocese spokesman who represented the Vatican Museum in negotiations. "The sponsor is not Philip Morris as a cigarette company but Philip Morris Inc" Clark told *The Times*. Philip Morris Inc manufactures a handful of non-tobacco products, among them Miller Beer and 7-Up, but more than 90% of its income is derived from its 170 brands of cigarettes.

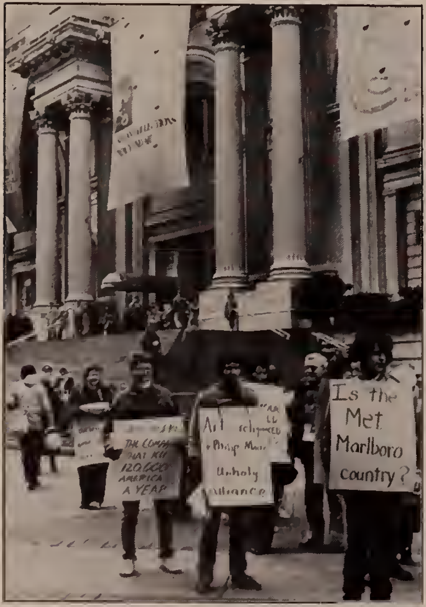
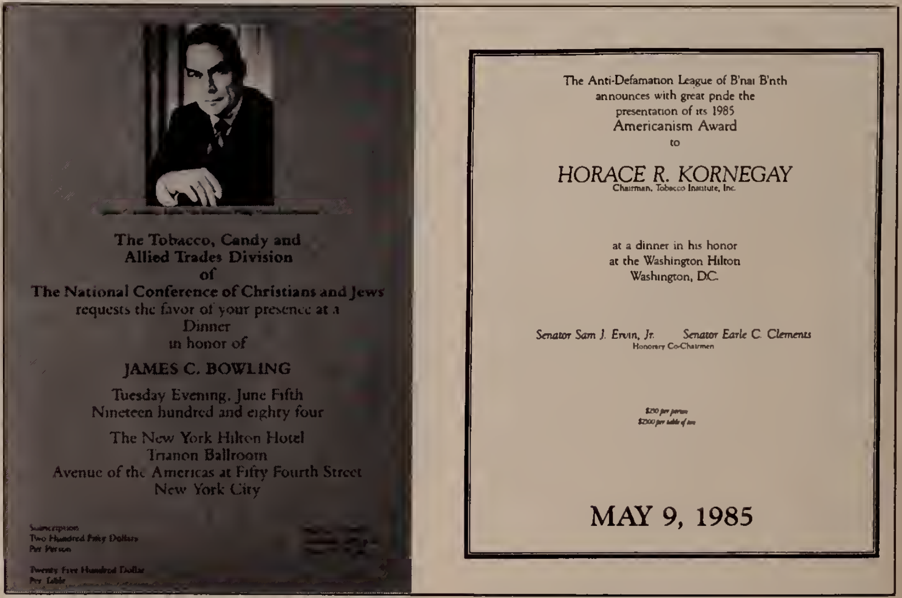

Is the Met Marlboro Country?
Unholy alliance

FIGURE 3. "Housecall" paid by physicians, medical students, and New Jersey GASP (Group Against Smoking Pollution) on Metropolitan Museum of Art, New York City to protest tobacco company sponsorship of the arts.

Testimonials

A less-publicized facet of prominent religious organizations is revealed in the bi-monthly issues of the *United States Tobacco and Candy Journal,* a New York-based trade publication for tobacco distributors. The National Conference of Christians and Jews (NCCJ), Catholic Charities, the Anti-Defamation League of B'nai B'rith (ADL), the United Jewish Appeal/Federation of Jewish Philanthropies, and the American Jewish Committee have all hosted dinners in recent years honoring tobacco company executives. Full-page advertisements announcing these activities are published in the *US Tobacco and Candy Journal* (Fig 4). The following events were among those held in 1984:

- The Tobacco, Candy and Allied Trades Division of the NCCJ honored James C. Bowling, senior vice president of Philip Morris Incorporated, at a $250-a-plate dinner June 5, at the New York Hilton. Senator Wendell Ford of Kentucky made the presentation of the organization's annual brotherhood award.
- The Man of the Year Award of the Anti-Defamation League of B'nai B'rith for 1984 went to Leon Shahun, Jr, president of the National Association of Tobacco Distributors. Shahun was honored for his "deep, abiding belief in social commitment and social service," at the St. Regis Hotel, New York City, September 18, at a $250-a-plate dinner.
- On December 11 at the Waldorf Astoria Hotel, the American Jewish Committee gave Hugh Cullman, retired



The Tobacco, Candy and
Allied Trades Division
of
The National Conference of Christians and Jews
requests the favor of your presence at a
Dinner
in honor of

JAMES C. BOWLING

Tuesday Evening, June Fifth
Nineteen hundred and eighty four

The New York Hilton Hotel
Trianon Ballroom
Avenue of the Americas at Fifty Fourth Street
New York City

Subscription
Two Hundred Fifty Dollars
Per Person

Twenty Five Hundred Dollar
Per Table

The Anti-Defamation League of B'nai B'rith
announces with great pride the
presentation of its 1985
Americanism Award
to

HORACE R. KORNEGAY
Chairman, Tobacco Institute, Inc.

at a dinner in his honor
at the Washington Hilton
Washington, D.C.

Senator Sam J. Ervin, Jr. *Senator Earle C. Clements*
Honorary Co-Chairmen

MAY 9, 1985

FIGURE 4. Advertisements in *United States Tobacco and Candy Journal* announcing awards by religious organizations to tobacco industry executives.

chairman of the board of Philip Morris its Human Relations Award. (A similar dinner was held in November 1981 to honor Edward A. Horrigan, president of RJ Reynolds.)

Why would any religious organization choose to honor an executive of a cigarette company? "You're asking a rather touchy question that's not fair to ask of the American Jewish Council," said Seymour Rich, who was coordinator for one of the testimonial dinners for a tobacco company executive. "We're honoring people who are very interested in the AJC, and we honor someone from the tobacco industry every year."

Like its counterpart organizations, the AJC honors executives from several fields that donate money to the committee, such as the supermarket and banking fields. Rich could not name a board of the AJC that examines ethics in fund-raising. "If it was a clear-cut decision about the tobacco industry, I assume the government would make it illegal," Rich told the *Journal*. The ADL spokesman, informed of the questions the *Journal* intended to pose, did not comment.

Father Joseph Funaro of the Catholic Diocese of Brooklyn has accepted funds for Catholic Charities at tobacco industry dinners in Chicago and Dallas and has entertained audiences at these functions with his singing rendition of "If I Were a Rich Man" from "Fiddler on the Roof." Funaro told the *Journal* he "didn't think about [the issue of] smoking" at the dinners. "You're seeing something dangerous in accepting money. I see the individuals I can help," said Funaro. "You're involved in the cigarette, and I'm involved with the person. I never received money with cigarette ashes on it." After discussing the health hazards of smoking and other lifestyles, Funaro said, "I never thought about not accepting the money because it came from cigarettes, but maybe I should have."

Harry Robinson, director of public relations for the National Council of Christians and Jews, said the NCCJ definitely has ethical considerations in accepting money. "We honor people who have made a contribution in their civic duty," said Robinson, "not what they do businesswise or how they earn their living. The Surgeon General has warned people that smoking is dangerous to their health, but it's not illegal." Robinson said people crusading against the cigarette industry "have forgotten that tobacco is indigenous to the economy of our country." He said that Senator Jesse Helms won re-election in North Carolina for his pro-tobacco stand. "If you really want to find out, go down and talk to these evangelical tobacco-growing folks who seem to be so religiously fervent, and see what they say about tobacco," said Robinson.

Smoking, medical ethics, and Jewish law

Although most prominent 20th century rabbinical authorities have not called for a prohibition on smoking, morally they do consider it a pernicious habit and would strongly discourage anyone from smoking. This is the opinion of Fred Rosner, MD, director of the department of medicine at Queens (NY) Hospital Center and author of three books on medical ethics. Rosner, who is co-chairman of the medical ethics committee of the Federation of Jewish Philanthropies and chairman of the newly formed committee on bioethics of the Medical Society of the State of New York, believes that Jewish law does prohibit smoking. In *Modern Medicine and Jewish Law* (New York, Bloch, 1972) and in an expanded essay, "Cigarette smoking and Jewish law" (*Journal of Halacha and Contemporary Society*, Fall, 1982), Rosner argues that in light of the overwhelming medical evidence proving the causal relationship of cigarette smoking to cancer of the lung, heart disease, and bronchitis, Jewish law absolutely forbids this practice. According to Rosner, admonitions to avoid endangering one's life or harming one's health appear throughout the Bible. For example, in Deuteronomy it is written, "take heed to thyself, and take care of thy life" (4:9) and "take good care of your lives" (4:15).

To Rosner, the subterfuge of "it is no concern of others if I endanger myself" is specifically disallowed by Maimonides and other classic rabbinical authorities. One of the few Biblical verses that has been cited by some rabbis who do not agree that smoking should be forbidden is Psalms 116: "The Lord preserveth the simple."

Neither is the fact that so many people smoke, or claim to find smoking pleasurable, a justification for condoning this life-threatening practice. Rosner has urged rabbinic authorities "to speak out on this subject without timidity" and has called on the Jewish community to "marshall its forces in an attack on the promotional activities of the tobacco industry."

SOUTHERN CHURCHES SPEAK OUT

Indeed, in North Carolina it is not easy to bring up the moral implications of profiting from tobacco, as the North Carolina Council of Churches has discovered. The state grows 42% of the tobacco produced in the United States, and 150,000 jobs (or 7% of the state's workforce) are tied to the tobacco industry, according to the North Carolina Department of Agriculture (*Wall St Journal*, May 8, 1984:37). In 1983, the 29-denomination council, following up on an inquiry by a local United Methodist Church into the problem of world hunger, began to discuss the moral dilemma of growing tobacco instead of food in a time of widespread starvation (*New York Times*, Dec 4, 1983:44). The council formed a Tobacco Study Committee which issued a report recommending that the state's tobacco farmers switch to other crops for moral reasons.

This conclusion incurred the wrath of tobacco farmers, marketers and manufacturers, who pointed out that tobacco contributes $1 billion a year to the state's economy. Concern was expressed by the Tobacco Growers' Information Committee that the council's report would be "used in propaganda wars by our enemies" (*Los Angeles Times*, Apr 27, 1984:1, 15-16). The managing director of the growers' committee noted that rural Methodist churches and all retired United Methodist ministers in North Carolina receive financial support from the Duke Endowment, which was established by tobacco baron James Buck Duke (*Wall St Journal*, May 8, 1984:37).

(Although Southern Baptists, the largest single denomination, are not represented on the council, the North Carolina Baptist Convention has not been silent on the subject of smoking. In 1982, the convention supported a US House Resolution calling for a ban on cigarette smoking on airliners [*Atlanta, Constitution*, Apr 5, 1984:1, 8]).

In 1984 at its annual meeting, the Southern Baptist Convention (the nation's largest Protestant denomination with 14 million members in 36,000 congregations) drafted a resolution that urged Baptists engaged in growing tobacco to switch to other crops. It called

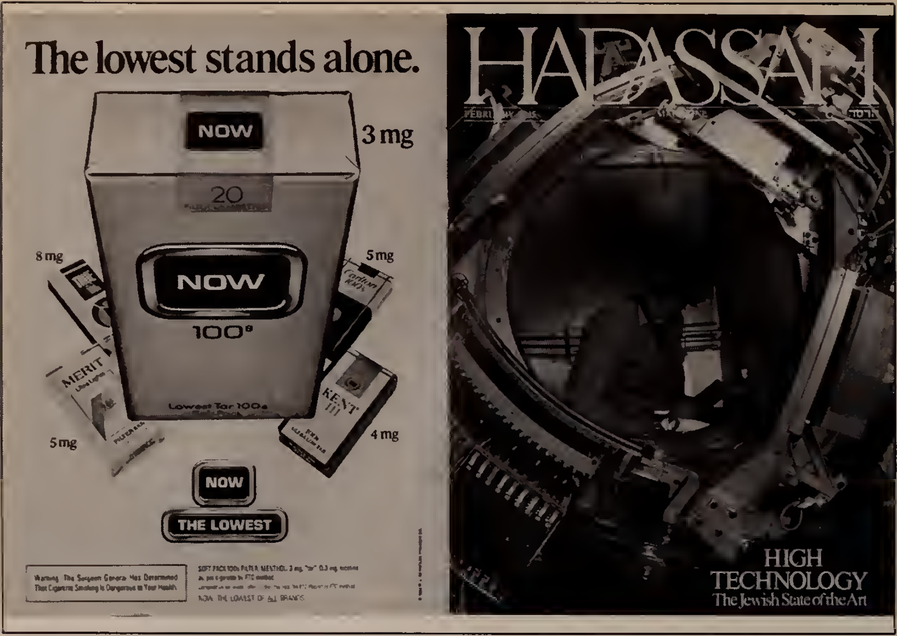


FIGURE 5. *Hadassah Magazine* publishes articles on health care and medical research alongside cigarette advertisements.

on Congress to end tobacco subsidies, asked parents (and all Baptists) not to smoke, and urged schools and churches to step up their dissemination of information on the harmfulness of smoking.

It is significant that churches in tobacco-growing states have expressed a stronger anti-tobacco sentiment than other American religious denominations apart from Seventh Day Adventist, Jehovah's Witness, Mormon, Pentecostal, Muslim, and other groups that advocate abstention from the use of tobacco.

Silent Religious Press

Are the silence or complacency of other religious organizations on the subject of smoking and the 360,000 cigarette-related deaths in the US each year tied to the acceptance of money from tobacco companies? The evidence suggests this. In addition to holding testimonial dinners for tobacco executives and welcoming tobacco sponsorship of religio-cultural events, certain religious newspapers—the editorial voices of various denominations—accept cigarette advertisements.

Consider the example of *Hadassah Magazine*, the official monthly publication of Hadassah, the 375,000-member Women's Zionist Organization of America. The major activity of the organization, founded in 1912, is fund-raising for medical research and health care in Israel. In 1981 nearly half of the $37 million raised by the organization was given to the 4,000-employee Hadassah-Hebrew University Medical Center in Jerusalem. Each issue of *Hadassah Magazine* has three to five full-page cigarette advertisements, usually for brands made by RJ Reynolds and Loews (Fig 5). These are often juxtaposed to articles describing medical advances in the diagnosis and treatment of cancer and other diseases.

Hadassah member Bernice Kleiman, who is married to a San Diego neurologist, was among the first persons to call attention to this "ridiculous and disgusting" situation. "I have belonged to Hadassah for many years," she told the *Journal*, "but I am absolutely appalled by the cigarette ads. In 1977 I wrote to the organization of my dismay and expressed my hope that once the contradiction of accepting cigarette advertising money on the one hand and financing medical research on the other was pointed out to them, they would terminate these shameful advertisements. The president answered with this incredibly poor excuse: 'You should know that Hadassah opposes boycotts in principle and therefore does not sponsor them. This policy holds even in this case where we recognize the adverse effects of tobacco advertising.' Talk about hypocrisy."

Alan Tigay, editor of *Hadassah Magazine*, told the *Journal* that the magazine accepts cigarette advertisements because it is "an economic necessity. We do get one or two complaints a month about it," he said. "Our response is that the ads follow the dictates of the Surgeon General's office, and that it's an important source of revenue without which it's possible this magazine couldn't survive." The editor said that some groups of readers object to liquor advertising too, and "if we give in to one segment of readership, we'd have to give in to all." The policy of the magazine will continue, he said, "unless the people who say there are surely other sources of revenue, can show them to me."

But Herb Brin, editor of the Jewish weekly *Heritage* newspaper, with four editions in California, has always maintained a policy against cigarette advertising, "since 1941 when I read the first

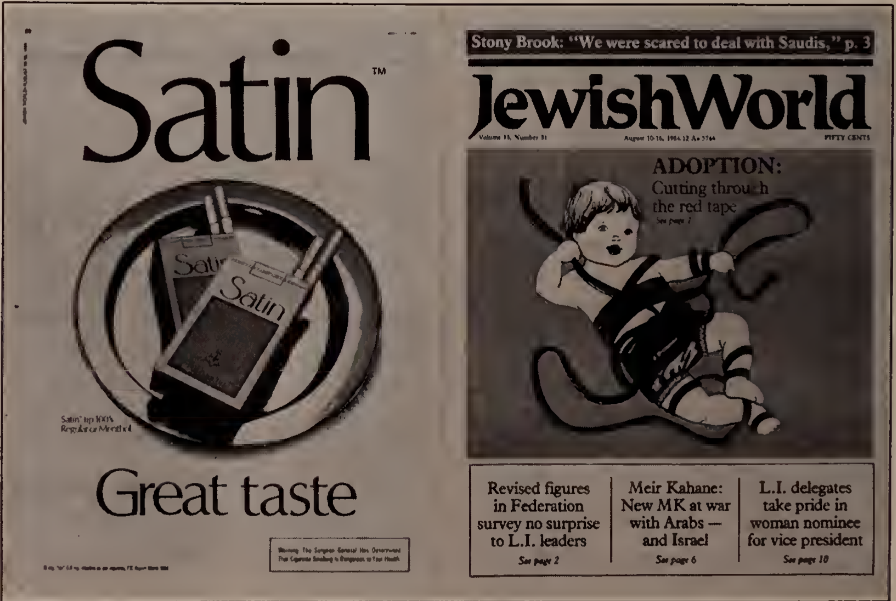

Satin™
Satin
Satin 100's
Regular or Menthol
Great taste
Stony Brook: "We were scared to deal with Saudis," p. 3
JewishWorld
ADOPTION:
Cutting through the red tape
Revised figures in Federation survey no surprise to L.I. leaders
See page 2
Meir Kahane: New MK at war with Arabs — and Israel
See page 6
L.I. delegates take pride in woman nominee for vice president
See page 10

FIGURE 6. Front and back covers of a Jewish weekly newspaper covering Long Island, NY.

Ethics and tobacco: the challenges to conscience

The following commentary is based on a presentation delivered by the Reverend Collins Kilburn, executive director of the North Carolina Council of Churches, at the 1985 North Carolina Health Council Convocation, Raleigh, May 8, 1985 (a conference entitled, "Tobacco: a dilemma for North Carolina health workers"). An earlier version of this paper appeared in the Church Council Bulletin *in March, 1985. Correspondence may be addressed to the Rev Kilburn, North Carolina Council of Churches, Byran Building, Suite 201A, Cameron Village, Raleigh, NC 27605.*

In June 1984 the Tobacco Study Committee of the North Carolina Council of Churches issued a paper entitled "Moral dimensions of tobacco." The paper, intended for study by congregations, church groups, and other interested individuals, drew considerable media attention and provoked much public discussion. Some of the discussion mistakenly supposed that the document was an attack on tobacco farmers.

To the contrary, the committee's report expressed emphatic concern for farmers, especially small farmers, who are dependent on tobacco growing. It pointed out that sudden interruption of the tobacco economy in North Carolina would bring hardship and ruin to large numbers of citizens. It insisted that farmers dependent on tobacco for their livelihood cannot be criticized as immoral "if alternative ways of economic activity are not available to them."

The committee went on to contend, however, that "the society as a whole is at fault if it acquiesces in tobacco dependency and fails to pursue with vigor and imagination the development of alternatives." Both the state and the nation, through universities and other public agencies, have devoted vast resources toward building and then perpetuating tobacco dependence. By comparison, little has been done to assist in a transition toward other forms of economic survival. The public stance, expressed through political leaders and public institutions, has been mainly one of acquiescence.

Now it looks as though the price stabilization program may be ended, and many small farmers in North Carolina may be injured. The problems with the program have little to do with moral or health questions. Foreign competition is a major factor. The large tobacco companies are increasingly buying their tobacco from Third World farmers and are assisting such farmers to expand their operations. The elimination of the stabilization program will not hurt the companies; nor will it do anything to diminish consumption or alleviate health problems. It will probably make tobacco more plentiful, cheaper, and more profitable for manufacturers.

Hope Shand, in a report recently published by the Rural Advancement Fund, discusses the likely demise of the tobacco program and the impact on small farmers. She argues that "neither blind allegiance to tobacco nor ill-conceived attacks on tobacco will do anything to support the intended beneficiaries of the tobacco program—the small family farmers. In North Carolina we have nearly lost sight of the original public purposes for this fifty-year-old farm program. Our goal should not be to save tobacco, but to save those whose livelihood depends upon tobacco. We must now take steps to salvage not the tobacco program but a state and federal farm policy which will provide for the kind of family farmers who traditionally benefited from the tobacco program." She also makes suggestions about the character of such programs.

Here is the proper focus of our moral concern—the people, not the product or the program. Defensive, knee-jerk reactions will not help, not in the long run; nor will simplistic crusades against the evils of smoking.

At the same time we need to be honest about the health hazards of smoking. The best evidence available indicates that tobacco kills about 350,000 people annually in the United States and over a million worldwide.

Is this not a moral concern for the churches? In recent years masses of church people in North Carolina rallied behind the legislation to crack down on drunk driving. No one doubted that that was an urgent moral concern, because 25,000 people were being killed each year by drinking drivers. Fourteen times that number die from smoking-related diseases. Six times as many people die from smoking-related causes as from all kinds of automobile accidents in the United States.

Another moral consideration highlighted in the report of the Tobacco Study Committee has to do with the marketing of cigarettes in impoverished countries. In the past decade cigarette consumption has leveled off in the developed nations. It has grown rapidly in the poorest and hungriest nations of the world—33% in Africa and 25% in Latin America, for example. Advertising is the force behind much of the growth.

Is it necessary for the health of our economy to export addiction and disease? Is this the "American way" of free enterprise? What is the responsibility of a society or an industry with regard to the marketing of a product which is, according to virtually unanimous medical judgement, lethal?

The subject of tobacco is many faceted and complex. That complexity does not hide the vital moral issues that are a part of the mix.

---

press release about experiments saying it might be harmful." Brin said smoking means destruction of the body, therefore it is a sin. Not accepting the ads hurts financially, he told the *Journal*. "And because 20% of out readers are college students, the cigarette companies would love to have us. But they never will."

The relatively large number of Jewish physicians and the value placed on health by the Jewish community would logically lead one to conclude that publishing of advertisements promoting cigarettes in *Hadassah Magazine* and numerous other Jewish newspapers (Fig 6) would be nothing short of scandalous. Instead, the virtual absence of protests suggests that this setting represents an ideal opportunity for tobacco companies to foster complacency, or even a positive image, about smoking. ("If Hadassah accepts it, then smoking can't really be that bad.")

In spite of the sponsorship by Philip Morris of the Vatican art exhibition and in spite of the acceptance by Catholic Charities of tobacco money, the Catholic press does not accept cigarette advertising. The weekly newspaper *New York Catholic* has long refused such advertisements because, according to sales representative Charles

Cigarette companies called sponsors of pornography

In May, 1980 *The Catholic Commentator* of Baton Rouge, LA, published a front-page article on corporations that advertise in sexually explicit magazines. Headlined "Top porn-pushers named," the report listed the leading advertisers in such magazines as *Penthouse, Playgirl,* and *Playboy*. RJ Reynolds was by far the largest purchaser of advertising space, having placed 70 pages of cigarette advertisements during a three-month period surveyed by the Mississippi-based National Federation for Decency (NFD). Reynolds was followed by liquor distiller Joseph E. Seagram & Sons and tobacco companies Brown & Williamson, Philip Morris, Loews, and Liggett. Other advertisers included CBS, Inc and Anheuser-Busch. The survey showed that tobacco and alcohol advertising accounted for 50% of revenues of such magazines.

Noted the Reverend Donald E. Wildmon, executive director of the NFD, "It is appalling to the average consumer that every can of Del Monte pineapples he buys, every 7-Up he drinks, every box of Kentucky Fried Chicken he eats, and every record he buys from CBS helps push a sickness which perverts and destroys human dignity. It is shocking that these companies fully support pornography, are in sympathy with the goals of pornography, and desire the results pornography provides."

Murray, they are "not conducive to good health." *Chicago Catholic* business manager Mary Healey told the *Journal* that cigarette advertising "doesn't seem to be congruent with the general ambiance of our newspaper."

One Catholic theologian who isn't content to let tobacco companies—and their shareholders— escape responsibility for their actions is Father Michael Crosby. Writing in the magazine *Christianity and Crisis* (May 30, 1983:212–215) Crosby accuses tobacco companies of extreme unethical conduct in their failure to include warnings on the health risks of smoking in their increasingly aggressive advertising campaigns in developing nations. Crosby believes stockholders should play a greater role in influencing corporate decisions. Crosby's religious body, the Midwest Province of the Capuchin Franciscan order, invests in stocks as a way to earn income for its work among the poor. But the order also uses the leverage of stock ownership to question the morality of company policies. With its 10 shares each in Philip Morris and RJ Reynolds, the order has sought through shareholder resolutions a review of their cigarette marketing practices in the Third World. In 1982 resolutions to this effect were vigorously opposed by management and were defeated by 98% (Philip Morris) and 96% (RJ Reynolds) of the vote. Crosby noted that the top five American insurance companies (Prudential, Metropolitan, Equitable, Aetna, and John Hancock) each holds thousands of shares of both tobacco companies and did not support the resolutions.

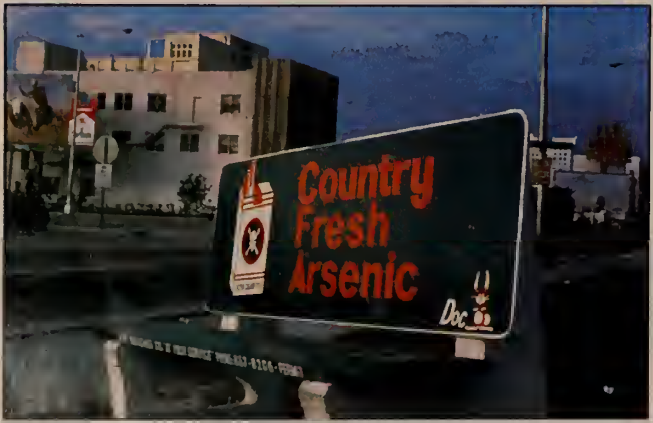
"At this point," concludes Crosby, "any shareholder protesting export policies of tobacco companies will be even more the voice of one crying in the wilderness than is the case with shareholder protests against policies of arms manufacturers or the makers of infant formula." All the more reason, he suggests, to maintain a constant presence within tobacco companies and to step up the challenges. In the short term, he says, "We expect to lose—but not to quit."

Some religious groups have begun to re-examine their investment portfolios for possible financial links to cigarette companies and other manufacturers of harmful products. (In January 1985, when the British Medical Association, in conjunction with Social Audit, an investigative journalists' group, released a comprehensive list of religious and medical organizations that owned shares in tobacco companies, there was a great hue and cry—followed by a massive sell-off.) A long-time proponent of "social investing"—the purchase of stock in companies whose products and services, employment practices, and public policies meet a certain moral standard—is the Interfaith Center on Corporate Responsibility (475 Riverside Drive, New York, NY 10115), a group affiliated with the National Council of Churches. In addition to proposing stockholder resolutions on such issues as apartheid, military production, and pharmaceutical marketing practices abroad, the center represents $10 billion in investments by 17 Protestant agencies and 200 Roman Catholic orders and dioceses.

The center's director, the Reverend Tim Smith, is dismayed by the increasing ties of cigarette companies to religious organizations. "They are pushing a series of lies to gain approval in society, in spite of what they provide and sell," Smith told the *Journal.* "It is the height of irony that the Vatican's religious art was sponsored by Philip Morris, who through advertising and promotion is one of the greatest contributors to the death and deterioration of millions of people."

Alan Blum, MD,
Kathleen Fitzgerald

Ms Fitzgerald is a freelance journalist in Chicago and a former reporter for The San Diego Tribune.



IN-THE-STREETS PREVENTION

An example of what may have been the first series of paid counteradvertisements in the United States aimed at undermining tobacco propaganda. From 1977 to 1979 bus bench ads such as this appeared throughout Miami, Florida. They were purchased by DOC (Doctors Ought to Care, Floyd Medical Center, Rome, GA 30161) after billboard companies refused to sell space to the physicians group. The low cost notwithstanding (less than $25 per month per bench), these ads, aimed at young people, sparked national publicity and led to the formation of other DOC groups. The absence of mass media counteradvertising in a community or society enables the tobacco industry to control the imagery, vocabulary, social acceptability, and flow of information in regard to cigarette smoking.

When researchers accept funding from the tobacco industry, do ethics go up in smoke?

Biologist Gordon Sato, PhD, director of the W. Alton Jones Cell Science Center in Lake Placid, NY, tells the story of Ignaz Semmelweis and his money machine. It seems that the Hungarian obstetrician was called on the carpet by the dean of his medical school for accepting money from a manufacturer of condoms. "The dean told Semmelweis 'We can't accept money from them.' Semmelweis said, 'Don't worry. In my basement, I have a machine where you put dirty money in on top and it comes out clean on the bottom.' "

Sato is making a point: In his opinion, how research money is used, not its source, is the important thing. Sato is sensitive about funding because he serves as a member of the Scientific Advisory Board of the Council for Tobacco Research-USA, the biomedical research arm of the tobacco industry.

While universities are under siege to sell off their stock in companies that do business with racist South Africa, and the warning flags have been hoisted about university financial relations with corporate America, the cigarette industry's efforts to launder its tobacco-stained profits in research go virtually unnoticed. The scientific community has yet to reach a consensus on whether the source of funds in itself can sully a project.

Abbott Lawrence Lowell, president of Harvard University from 1909 until 1933, used to say that he would accept money from anyone as long as they did not tell him how to spend it. But if he were at the helm today, some faculty members might question whether taking money from a tainted source does not somehow taint the recipient.

At Yale University, investigators can no longer do classified government research, because such work is considered antithetical to the free flow of scientific information. At Tufts University, a flap occurred in 1977 when Philippines dictator Ferdinand Marcos offered to endow a chair in his own name in the Fletcher School of Law and Diplomacy. Some Tufts faculty members argued that taking Marcos' money would legitimize a bloody regime. Tufts President Jean Mayer, PhD, asked, "Does accepting money from the Carnegie Foundation mean endorsing the shooting of miners? Does accepting money from the Rockefeller Foundation mean endorsing monopolistic practices? Obviously, we will not accept money from any and every source. But how do you draw the line?"

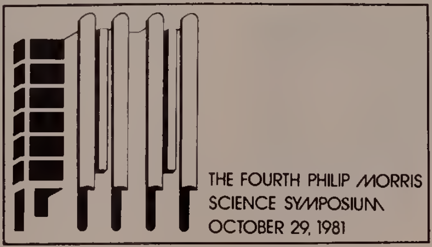
When it comes to tobacco, virtually no lines have been drawn.

Noble Research?

Cigarettes remain a legal product in this country, and the tobacco companies are happy to spend some of their profits on research programs and, apparently, most researchers would be happy to take it.

When Philip Morris, the makers of Marlboro and other brands of cigarettes, held a symposium for scientists from government, academia, and industry in 1981 in Richmond, VA, two Nobel laureates—physicists Rosalyn A. Yalow, PhD, of the Veterans Hospital, Bronx, developer of the first application of the radioimmunoassay, and Alan M. Cormack, MSc, of Tufts, whose work led to the development of computerized tomographic scanning—were among the researchers present. The tobacco companies have a knack for sponsoring the stars of science as well as of the performing arts and sports.

The industry-funded Council for Tobacco Research (CTR) boasts that it was among the patrons who supported the Nobel prize-winning immunology research by Baruj Benacerraf, MD, chairman of the department of pathology at Harvard Medical School. Some researchers may have reservations about taking tobacco-industry money, but the New York-based council's scientific director, Sheldon C. Sommers, MD, said he personally knows only one.



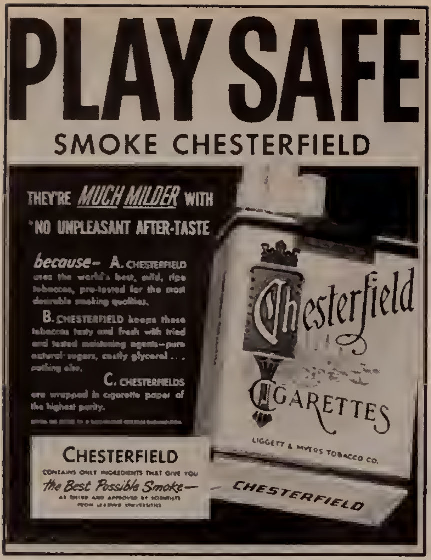
Tobacco company-sponsored conference. All in the name of science?

Perhaps only one institution in the world, the University of Sydney in Australia, has publicly disclosed its debate over the ethics of accepting tobacco money. According to the *Medical Journal of Australia* (1982; 2:391–392), the university decided in 1982 to turn down financial support from any tobacco company if the firm is publicly identified as the source. In calling for this action, cardiologist Gaston Bauer, warden of the clinical school at Royal North Shore Hospital, pointed out that by refusing funds the university would "gain the respect of the community."

According to Sommers, a consulting pathologist at Lenox Hill Hospital, New York, "Ten, 15 years ago, there were some organizations, such as Rockefeller University, that didn't want CTR money, but that's all gone by. [Now,] there's not a single organization, a university, a research institute, a college, that turns down the money."

Rockefeller University apparently has changed its tune, because it now has an RJ Reynolds Research Fellowship. Current projects funded by the CTR are being conducted at New England Deaconess Hospital, the University of California at Berkeley, Sidney Farber Cancer Institute, Johns Hopkins Oncology Center and the university's School of Hygiene and Public Health, Yale University School of Medicine, Harvard Medical School, and Albert Einstein College of Medicine. The smoky trail of funding can be followed down many other prestigious corridors.

And researchers in growing numbers are seeking funding from tobacco companies. There are at least two reasons for this. The first is that the tobacco industry has been increasing its investment in research at the same time government funds have diminished. Second, the Council for Tobacco Research has been attracting accomplished scientists to its board and gaining publicity for its research funding efforts. Sommers said the CTR has seen an increase in applicants as federal sources for research funding have dried up. "We are in a world where the National Institutes of Health are slowly making it more difficult to get money. And the side ef-



PLAY SAFE
SMOKE CHESTERFIELD

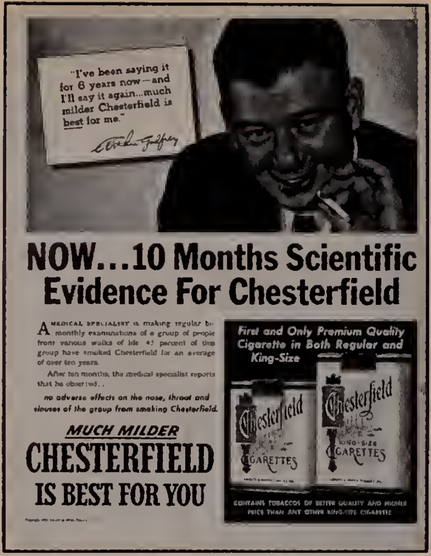
THEY'RE *MUCH MILDER* WITH
*NO UNPLEASANT AFTER-TASTE

because— A. CHESTERFIELD uses the world's best, mild, ripe tobaccos, pre-tested for the most desirable smoking qualities.

B. CHESTERFIELD keeps these tobaccos tasty and fresh with tried and tested moistening agents—pure natural sugars, costly glycerol . . . nothing else.

C. CHESTERFIELDS are wrapped in cigarette paper of the highest purity.

CHESTERFIELD
CONTAINS ONLY INGREDIENTS THAT GIVE YOU
the Best Possible Smoke—
AS TESTED AND APPROVED BY SCIENTISTS FROM LEADING UNIVERSITIES



"I've been saying it for 6 years now—and I'll say it again...much milder Chesterfield is best for me."
Arthur Godfrey

NOW...10 Months Scientific Evidence For Chesterfield

A MEDICAL SPECIALIST is making regular bi-monthly examinations of a group of people from various walks of life. 45 percent of this group have smoked Chesterfield for an average of over ten years.

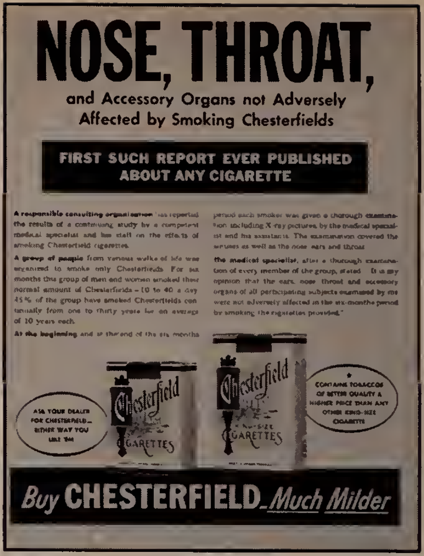
After ten months, the medical specialist reports that he observed . .

no adverse effects on the nose, throat and sinuses of the group from smoking Chesterfield.

MUCH MILDER
CHESTERFIELD
IS BEST FOR YOU

First and Only Premium Quality Cigarette in Both Regular and King-Size

CONTAINS TOBACCOS OF BETTER QUALITY AND HIGHER PRICE THAN ANY OTHER KING-SIZE CIGARETTE



NOSE, THROAT,
and Accessory Organs not Adversely Affected by Smoking Chesterfields

FIRST SUCH REPORT EVER PUBLISHED ABOUT ANY CIGARETTE

A responsible consulting organization has reported the results of a continuing study by a competent medical specialist and his staff on the effects of smoking Chesterfield cigarettes.

A group of people from various walks of life was organized to smoke only Chesterfields. For six months this group of men and women smoked their normal amount of Chesterfields—10 to 40 a day. 45% of the group have smoked Chesterfields continually from one to thirty years for an average of 10 years each.

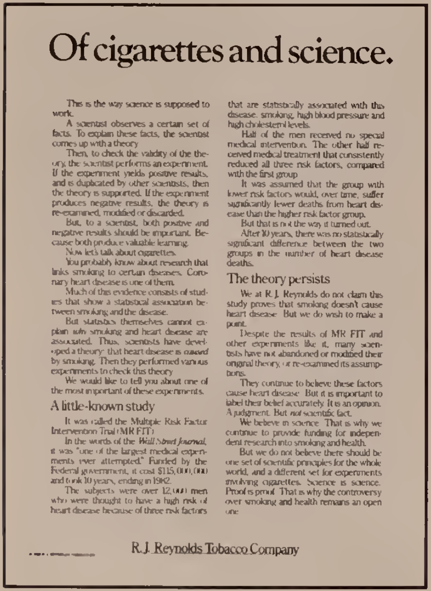
At the beginning and at the end of the six months period each smoker was given a thorough examination including X-ray pictures, by the medical specialist and his assistants. The examination covered the sinuses as well as the nose, ears and throat.

The medical specialist, after a thorough examination of every member of the group, stated: "It is my opinion that the ears, nose, throat and accessory organs of all participating subjects examined by me were not adversely affected in the six-months period by smoking the cigarettes provided."

ASK YOUR DEALER FOR CHESTERFIELD—EITHER WAY YOU LIKE 'EM

CONTAINS TOBACCOS OF BETTER QUALITY & HIGHER PRICE THAN ANY OTHER KING-SIZE CIGARETTE

Buy CHESTERFIELD—*Much Milder*



Of cigarettes and science.

This is the way science is supposed to work.

A scientist observes a certain set of facts. To explain these facts, the scientist comes up with a theory.

Then, to check the validity of the theory, the scientist performs an experiment. If the experiment yields positive results, and is duplicated by other scientists, then the theory is supported. If the experiment produces negative results, the theory is re-examined, modified or discarded.

But, to a scientist, both positive and negative results should be important. Because both produce valuable learning.

Now let's talk about cigarettes.

You probably know about research that links smoking to certain diseases. Coronary heart disease is one of them.

Much of this evidence consists of studies that show a statistical association between smoking and the disease.

But statistics themselves cannot explain *why* smoking and heart disease are associated. Thus, scientists have developed a theory: that heart disease is *caused* by smoking. Then they performed various experiments to check this theory.

We would like to tell you about one of the most important of these experiments.

A little-known study

It was called the Multiple Risk Factor Intervention Trial (MR FIT).

In the words of the *Wall Street Journal*, it was "one of the largest medical experiments ever attempted." Funded by the Federal government, it cost $115,000,000 and took 10 years, ending in 1982.

The subjects were over 12,000 men who were thought to have a high risk of heart disease because of three risk factors that are statistically associated with this disease: smoking, high blood pressure and high cholesterol levels.

Half of the men received no special medical intervention. The other half received medical treatment that consistently reduced all three risk factors, compared with the first group.

It was assumed that the group with lower risk factors would, over time, suffer significantly fewer deaths from heart disease than the higher risk factor group.

But that is not the way it turned out.

After 10 years, there was no statistically significant difference between the two groups in the number of heart disease deaths.

The theory persists

We at R.J. Reynolds do not claim this study proves that smoking doesn't cause heart disease. But we do wish to make a point.

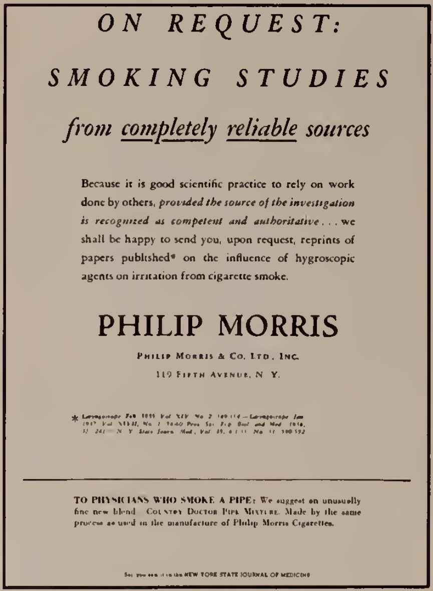
Despite the results of MR FIT and other experiments like it, many scientists have not abandoned or modified their original theory, or re-examined its assumptions.

They continue to believe these factors cause heart disease. But it is important to label their belief accurately. It is an opinion. A judgment. But *not* scientific fact.

We believe in science. That is why we continue to provide funding for independent research into smoking and health.

But we do not believe there should be one set of scientific principles for the whole world, and a different set for experiments involving cigarettes. Science is science. Proof is proof. That is why the controversy over smoking and health remains an open one.

R.J. Reynolds Tobacco Company



ON REQUEST:
SMOKING STUDIES
from completely reliable sources

Because it is good scientific practice to rely on work done by others, *provided the source of the investigation is recognized as competent and authoritative* . . . we shall be happy to send you, upon request, reprints of papers published* on the influence of hygroscopic agents on irritation from cigarette smoke.

PHILIP MORRIS
PHILIP MORRIS & CO. LTD., INC.
119 FIFTH AVENUE, N. Y.

* Laryngoscope Feb 1935 Vol XLV No 2 149-154—Laryngoscope Jan 1937 Vol XLVII, No 1 58-60 Proc Soc Exp Biol and Med 1934, 32 241—N Y State Journ Med, Vol 35, 6-1-35 No 11 590-592

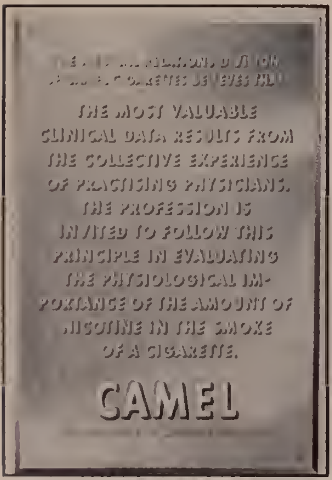
TO PHYSICIANS WHO SMOKE A PIPE: We suggest an unusually fine new blend—COUNTRY DOCTOR PIPE MIXTURE. Made by the same process as used in the manufacture of Philip Morris Cigarettes.

Say you saw it in the NEW YORK STATE JOURNAL OF MEDICINE

Important News For Smokers:
New Merit Research Released!

New survey results prove MERIT delivers in key areas of taste, ease of switch, and long-term satisfaction.

MERIT
Kings & 100's



THE MOST VALUABLE CLINICAL DATA RESULTS FROM THE COLLECTIVE EXPERIENCE OF PRACTISING PHYSICIANS. THE PROFESSION IS INVITED TO FOLLOW THIS PRINCIPLE IN EVALUATING THE PHYSIOLOGICAL IMPORTANCE OF THE AMOUNT OF NICOTINE IN THE SMOKE OF A CIGARETTE.

CAMEL

Throat Specialists report on 30-day test of Camel smokers:

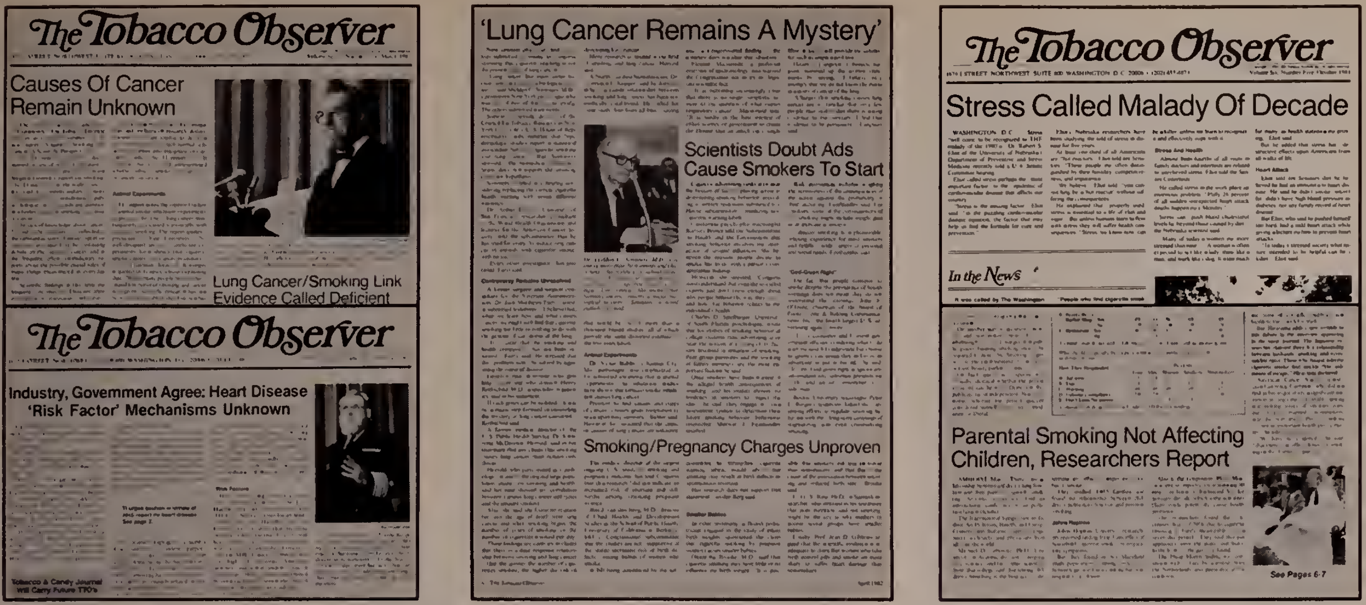
"Not one single case of throat irritation due to smoking Camels!"

More Doctors Smoke Camels

PROGRESS THROUGH RESEARCH

RESEARCH:
Key to Kent's popularity

In its promotional efforts over the past 50 years the tobacco industry has used science and research to sell cigarettes.



The Tobacco Observer

Causes Of Cancer Remain Unknown

Lung Cancer/Smoking Link Evidence Called Deficient

The Tobacco Observer

Industry, Government Agree: Heart Disease 'Risk Factor' Mechanisms Unknown

'Lung Cancer Remains A Mystery'

Scientists Doubt Ads Cause Smokers To Start

Smoking/Pregnancy Charges Unproven

The Tobacco Observer

Stress Called Malady Of Decade

In the News

Parental Smoking Not Affecting Children, Researchers Report

See Pages 6-7

The Tobacco Observer, tobacco industry publication, reports on smoking and health. The Council for Tobacco Research's Scientific Director Sheldon Sommers, MD. gave this testimony at Congressional hearings on warning labels: "Cigarette smoking has not been scientifically established to be a cause of chronic diseases, such as cancer, cardiovascular disease, or emphysema. Nor has it been shown to affect pregnancy outcome adversely."

fect is that we attract more applicants," he said.

The tobacco industry has nearly doubled its funding of CTR research to $9 million since 1980. CTR funds about 40% of its 200 applicants per year, compared with a 20% funding level in many parts of NIH.

NOT JUST SMOKING

Since it was founded in 1954, CTR claims to have spent $83 million on 865 projects in 279 medical schools, hospitals, and research institutions. Though the bulk of the money has been devoted to issues of smoking and health, the council—with the blessing of its benefactors—has recently become a general biomedical research group, devoting about half of its funds to research unrelated to smoking. The council pays a stipend of $7,000 per year to its members.

Epidemiologist Gary Friedman, MD, assistant director of the Department of Medical Methods Research, Kaiser Foundation Research Institue, Oakland, CA, a former grantee, said, "We were very concerned that they would try to influence the results. I can't speak for everything the tobacco industry supports, but that particular group, the Council for Tobacco Research-USA, seems to be an independent group that is trying to sponsor good studies. They did not say anything to us about what to publish or what not to publish. They did not try to influence us in any way." Friedman noted that several studies published by his group found harmful effects from tobacco.

The council may have avoided criticism, in part, because the researchers it funds follow a practice known as "piggybacking" or mingling money from a variety of sources. The council's annual report shows that it is co-funding research with the American Cancer Society, the National Science Foundation, the National Institutes of Health, and other leading organizations.

Joanne Luoto, MD, director of the US Office of Smoking and Health, stated that the council "is buying legitimacy when its funds are mixed with those from NCI and other federal agencies."

Sommers said that piggybacking used to feed the fires of hostility between the council and the American Cancer Society. However, the animosity has abated in recent years. In fact, Joann Schellenbach, director of press relations for the American Cancer Society in New York, said of the council, "They're legitimate. We're very critical of the tobacco industry in terms of their advertising practices and many other things that they do. But here's an area where they seem to be doing something by the book and promulgating good research. So I guess we can't criticize them across the board."

Historically, tobacco companies, like many other firms, bought research and used it to peddle their products. Scientific studies of tar and nicotine content of cigarettes often have been the subject of advertisements. Philip Morris advertisements of the 1930s, 1940s, and 1950s, as in one that appeared in *The Laryngoscope*, told physicians that they could use their "scientific knowledge" to help their patients "discriminate between mere claim and basic facts" made in cigarette promotion. The blurb concluded, "Test Philip Morris on patients suffering from congestion of the nose and throat due to smoking. Verify for yourself Philip Morris superiority." (see also *NY State J Med* 1983; 83:1347–1352)

But today's battle to win hearts and minds through the research sponsored by the Council for Tobacco Research and the individual tobacco companies is more subtle and sophisticated. By design or default, the tobacco industry seems to be reaping a bonus of good public relations for being a patron of research, just as it does from sponsoring art exhibitions, ballet, orchestra and sporting events.

AND ON AND ON

Why would the tobacco industry take the risk of sponsoring research that could place its product in an unfavorable light?

Joseph Cullen, PhD, deputy director of the National Cancer Institute, believes that the industry wins even when the research turns up negative findings. He maintains that the research always breeds other research, so that the companies can keep saying that major questions about tobacco remain unanswered. "As long as they keep funding

The research game

Publicly, little has been said in the scientific community about the possible compromise represented by the acceptance of funding from the tobacco industry. Privately, however, scientists point to the ethical problem that would arise for a colleague financed by an industry, who discovers that his research is being used in advertisements or public relations.

Yet this is precisely the modus operandi of the Tobacco Institute (TI), the information arm of cigarette manufacturers. Both the TI and the Council for Tobacco Research (CTR) are funded by the tobacco industry. Extensive coverage of the views of researchers who have received grants from the CTR has been carried in the *Tobacco Observer*, the bi-monthly newspaper of the TI that is sent to journalists, Congressmen, and thousands of other government officials. Few if any of these researchers are identified as having received remuneration from the CTR for travel or research. In contrast to the description of scientists who testify in support of stricter measures to discourage smoking as "anti-smokers," tobacco industry-financed researchers and others who oppose such measures are described as eminent, noted, or prominent authorities, experts, and scholars.

In addition to the *Tobacco Observer*, numerous tracts and pamphlets are distributed by the TI to tell "the other side" of the smoking "controversy." A review of the *Tobacco Observer* since its inception in 1976 (with the front-page headline, "Pesty anti-smokers blasted") reveals several techniques employed time and again, including the following:

- Highlighting proponents of the theory that genetics or personality are linked to diseases that most other research has attributed to smoking. This theory goes something like this: "People are born with a predisposition to smoke and to get lung cancer—and often these are the same persons."
- Pointing to the limitations of research, especially that which has implicated smoking as a major cause of lung cancer and heart disease, while praising research into the role of stress, anger, chemicals, occupation, eating habits, air pollution, and radiation as the culprits in these diseases.
- Claiming that the Tobacco Institute is not responding to the various reports and statements of the Surgeon General, but rather that it is making a "contribution to the public dialogue" on the question of whether cigarette smoking is a cause of disease.
- Likening measures to restrict smoking in public places to the segregation of blacks; and suggesting that victims of lung cancer are being made to bear guilt for having smoked, and that this alleged situation is akin to the scapegoating and massacre of Jews during outbreaks of plague in the Middle Ages.
- Citing the opinion of researchers in areas other than their field of expertise, such as Ernst Wynder, MD, one of the early discoverers of the link between smoking and lung cancer, to support the tobacco industry view that advertising does not influence people to smoke and should not be banned.
- Conversely, citing the opinion of experts in a given field to deny the very influence of that field (eg, the research director of the Advertising Association, the British and European lobbying arm of the advertising business, who suggested in Congressional testimony that "cigarette advertisements do not sell the idea of smoking. They are not intended to sell the idea of smoking. They are intended to sell brands and that is what they do").
- Pointing to newspaper articles reporting on research that either does not implicate smoking as a cause of disease or that implicates other, usually rare, suspects. Some of these reports have reached the news media through press releases of the TI.
- Fostering the notion that there exists a serious scientific dispute about the risks of smoking, and implying that some scientists (eg, the late Dr Hans Sellye) have considered smoking to have benefits to health.
- Suggesting that the emphasis on smoking is diverting attention from other kinds of research such as that related to Alzheimer's disease or interferon. (Tobacco companies are increasingly funding health research in areas unrelated to smoking.)

Although the CTR claims to be an independent research organization funding independent researchers, several members of its Scientific Advisory Board have been awarded grants from the board. This is not to suggest that the researchers are compromised, but rather that the type of funding in question puts researchers in the position of having to be careful not to be compromised. After comparing the public testimony of researchers receiving funding from the CTR with the use made of that testimony by the TI's *Tobacco Observer*, one might conclude that the tobacco industry would find it in its best interest to fund research only in areas in which it feels safe or has the expectation of being able to use or publicize the data. However generous tobacco companies may be toward research, and however dedicated their grant recipients may be, the main concern of tobacco companies is to increase the sale of cigarettes—and the main concern of the Tobacco Institute is to help deflect threats to the sale of cigarettes, including scientific evidence indicting cigarette smoking as the leading preventable cause of death.

—Alan Blum, MD

science, it makes them look like they truly are concerned and interested in what the truth and the facts are," added Cullen. "They have some terrific people on their board. It is in their [the tobacco industry's] interest to look honest, to look as if they are scientifically curious about the real truth."

Luoto agreed, saying, "Funding research helps the industry keep alive the notion that the dangers of smoking are still in question. As long as they keep funding research and people keep doing research, even if it's into the subcellular molecular basis of carcinogenesis, John Q. Public is not going to know that distinction so he's going to think, 'Hmmm, they haven't proved that it causes cancer yet.' They're using research funds directly or indirectly to further their aims. It's an issue that has not been raised before. It is conceivable that they are doing legitimate research while they are getting illegitimate public relations benefit from it."

The funding offers the industry another bonus: It has culled a group of scientists who can testify on the industry's behalf. During cigarette-labeling hearings in March 1983, at least nine researchers, including CTR's Sommers, who had received council money presented statements to the House Subcommittee on Health and the Environment.

CTR's Sommers discounts these arguments, holding that the only purpose of the research is to uncover scientific truths. "We are disease-oriented, not public relations-oriented people as a group," said the pathologist, who believes that tobacco primarily acts in concert with other factors to cause disease. "I'm not a propagandist. I don't give a damn what happens to the tobacco industry."

Cullen believes that despite pressures on researchers to find grants where they are available, investigators are starting to realize that by accepting tobacco money they are aiding an industry responsible for 1,000 deaths per day in the United States. "I've been an investigator for many years. I wouldn't take a nickel from them," the behavioral psychologist said.

Fredrick Stare, MD, PhD, founder of Harvard's Department of Nutrition, accepted grants from the council 30 years ago, but says that, with a single exception, he wouldn't touch their money today. "The case against tobacco wasn't as well documented when I accepted their money as it is now," he said. "I wouldn't accept their money today because it would help improve the image of an industry whose main product is killing so many people. However, there is an exception. I'd accept their money to research tobacco as a food source. Maybe if they could sell tobacco as food for humans or livestock, they wouldn't have to sell it as cigarettes."

Howard Wolinsky

Howard Wolinsky is the medical reporter for the Chicago Sun-Times.

Tobacco dilemma intensifying in North Carolina

This day in early May many of his fellow tobacco farmers are well into another uncertain season, transplanting tender seedlings from bed to fields; but John Vollmer takes time off to plow a different field.

At North Carolina State University in Raleigh, the state's leading agricultural school and a stalwart tobacco institution, Vollmer is an emissary. Despite the setting, he meets not with tobacco compatriots, but with their long time nemeses, health professionals. About 100 have congregated for a unique North Carolina Health Council Convocation on the tobacco dilemma—a first in the heart of tobacco's dominion.

Trained in the tobacco leadership program of the Philip Morris Company, Vollmer nevertheless appears uncomfortable. Loosening his tie, he warms the too-quiet audience by acknowledging, "I've been wanting to sneak off to the men's room for a smoke all morning."

He is here to tell them about the other side: what tobacco farmers do and why. He does not dispute the health consequences of smoking; in fact, he acknowledges his father has emphysema and that this children are well-schooled in the hazards of smoking. He talks of choice and freedom. He appeals for their understanding, saying, "It's important that we begin to talk to one another."

No more incongruous scene could have been imagined five years ago in the capital of the nation's leading tobacco and cigarette producing state. Despite more than 20 years of assault by medical research and surgeon generals' warnings, here tobacco remains the golden currency.

FADING GLOW

The North Carolina tobacco crop is worth about $1 billion a year. Long the leading farm income producer, tobacco's throne now is being challenged by a fast growing poultry industry. Still, tobacco represents about 25% of the state's agricultural income and is the mainstay for some 60,000 tobacco farmers. Additionally, five of the nation's six cigarette manufacturers have plants in the state. These employ about 25,000 people who take home annual gross wages of about $600 million. In aggregate, the North Carolina tobacco industry from farm to auction warehouse to cigarette factory approaches $2 billion and provides 147,000 jobs.

Even though the leaf's glow has faded over the last two decades, it is ingrained in the Carolina heritage. It is money to be sure. But also it is steeped in tradition, pegged to the ritual of the seasons, as Vollmer's slide show demonstrates. It is smothered in lifestyle. It is colored the red, white, and blue of American self-reliance and independence. The call for a smoke-free society within 15 years is like a declaration of hostilities. Yet, the real battle has been brewing for years and is far more immediate. Farmers, cigarette manufacturers, and politicians are trying to fashion a new alliance out of the now divisive and failing tobacco price support and quota program.

Price supports are too high, making American tobacco noncompetitive on international markets. Tobacco warehouses are bulging with government-financed surpluses. Even though it will mean reduced income, many farmers agree with manufacturers that the support price must come down. Quota owners, however, do not favor price support reductions because that will affect the value of their allotments. And farmers are wary of a heavy 25 cent per pound assessment on their leaf sales this year, to guarantee the stabilization program loans. They are pressing for legislation to allow cigarette manufacturers to buy out the surplus leaf at large discounts. That will result in huge program losses at a time when the tobacco program faces its severest criticism in Congress. Not even the experts are willing to predict the outcome.

"NO NET COST" MAY BE COSTLY

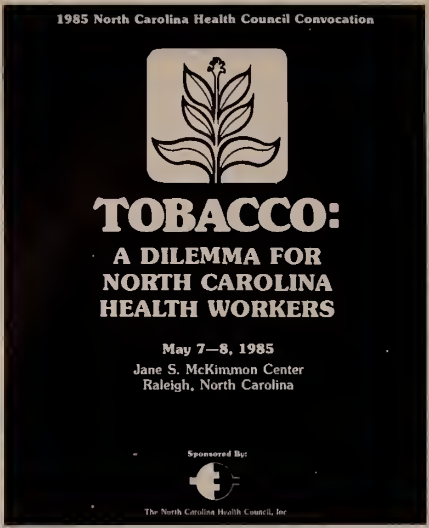
At first glance, the tobacco system looks simple; in fact, it is incredibly complex. Created by New Deal legislation in the 1930s, the federal price support system guarantees farmers a price, currently $1.70 a pound for flue-cured tobacco. Leaf, not purchased by tobacco company buyers at auction, is automatically bought up at the federally guaranteed price by the Flue-Cured Cooperative Stabilization Corp. But because the guaranteed price is higher than world prices, and manufacturers have been importing greater amounts of cheaper foreign leaf, the stabilization program is anything but stable. Its warehouses are filled with 812 million pounds of unsold flue-cured leaf, dating to the 1975 crop. Flue-cured, or bright leaf, is the major ingredient in cigarettes. It is the type overwhelmingly grown in North Carolina, with some burley tobacco grown in the mountain counties. Burley, also used in cigarette blends, as well as in smoking and chewing tobacco, is the most widely grown. Raised in 12 states, including Kansas, Ohio, and Indiana, burley is mainly produced in Kentucky. About 512 million pounds of burley are in surplus warehouses. Clearing out these large stores through a discount manufacturers' buyout will cost, primarily taxpayers, an estimated $500 million to $1 billion in failed subsidy loans. Farmers will share the burden for loans made since 1982 when the No Net Cost feature began. It assesses a per pound charge to farmers to insure the government loan. Initially it was a few cents, but market conditions forced it to more than triple this year to 25 cents per pound. Farmers feel caught in a squeeze between rising tobacco imports, crushing assessments, and high leases for growing quota.

Most growers today, operating on 5 to 20 acres, must lease the right to grow—from a quota owner. As leaf surpluses grew in recent years, the government was compelled to reduce quotas (about 50% since 1978). But this increased quota demand among competing growers and drove up quota lease prices. It is estimated that more than 111,000 North Carolinians own quota for flue-cured tobacco, but less than 25% actually grow the crop. And a study by the Government Accounting Office showed that about 25% of the quotas are held by retired farmers or their widows for whom the lease income is important. Lending irony to the dilemma, D. G. Harwood, assistant director of the state's Agricultural Extension Service, observes, "If you owned an allotment [quota], you could clear $1,169 an acre, never touch the crop, and be a health worker on the side."

Top Cash Crop

Having sufficient quota is crucial to tobacco farmers, but many say they are paying too heavily for the privilege. In some fertile eastern counties, quota is leased for as much as 80 cents per pound, almost half of the existing support price. Critics say the system has become archaic, sustaining absentee, but politically powerful, allotment holders who are said to dominate the 232,000 member North Carolina Farm Bureau. James Canoy is among small tobacco farmers who say it has become a medieval, feudal system. "The tobacco farmer's not much more than a serf, the way the system is now," he says. For him the difficulty of acquiring sufficient quota at reasonable lease prices has become too much. Instead, he struggles with milo and soybeans.

Others, like John Vollmer, have diversified their operations. But on the small family farms that still checker the Carolina countryside, tobacco generates the cash. Farm extension service studies show that tobacco is the undisputed king of the field, yielding as much as $2,450 per acre above direct cash outlays (such as for seed and fertilizer, but excluding capital expenses). By comparison, corn yields about $142 per acre; wheat, $100; soybeans, $66; and peanuts, $399.



1985 North Carolina Health Council Convocation

TOBACCO:
A DILEMMA FOR
NORTH CAROLINA
HEALTH WORKERS

May 7—8, 1985
Jane S. McKimmon Center
Raleigh, North Carolina

Sponsored By:
The North Carolina Health Council, Inc

The boldness of North Carolina's health professionals, newspaper editorialists, and religious leaders in calling for stepped up measures to discourage tobacco consumption has worried growers.

Without tobacco, the family farm and supporting communities would be gobbled up like they were decades ago with mechanization in the midwest, says T. Carlton Blalock, president of the Tobacco Growers Association, adding "Heck, those big tractors and harvesters they use in Iowa, you couldn't even turn 'em around in one of our fields." Yet, NC State University economics professor Daniel Sumner says that, in terms of revenue, Iowa grain farms and Carolina tobacco farms are comparable. And in a recent study of potential effects of eliminating the tobacco program, Sumner forecast that prices would fall as production increased 50% to 100%. Overall farm revenue would jump 12% to 60% in a free market, and cheaper prices would stimulate increased cigarette consumption—by about 2%, he predicted. "That point is so obvious, when you realize this is in effect a cartel that restricts production and keeps price high. Take it away, what do you think's going to happen. Supply increases, prices fall. There would be more smoking."

Manufacturers' Profits Rising

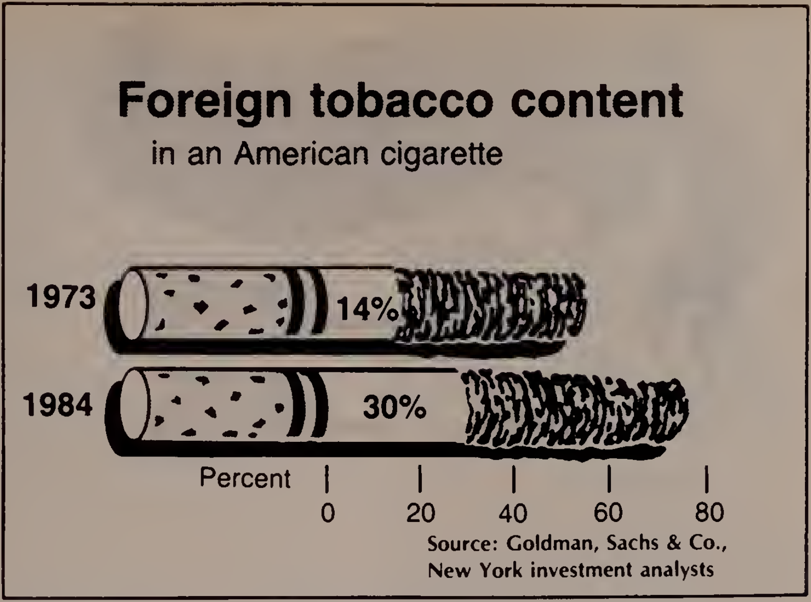
Blalock acknowledges the small family farm is probably doomed and that only the riches of tobacco have maintained it so long in North Carolina and neighboring tobacco states. However, he warns that eliminating the tobacco program in one fell swoop would have a devastating impact: an immediate $400 million loss in income to quota holders; a 25% drop in farm land values, which include the lucrative allotments; drastic property tax increases and/or budget cuts in many counties to address the lost tax base value; and shifting of tobacco production to more efficient manufacturer-contract farms and to other states with longer growing seasons. Manufacturers say they want a reliable supply of American flue-cured tobacco and support the federal controls, providing prices are moderated to reflect world market conditions. But should the program collapse, growers and analysts figure the big companies could be the big winners again, at least on paper. In spite of the cloud that hangs over the tobacco family, the manufacturers could not be healthier. According to industry analysts, manufacturers earned a neat 14% increase in profits in 1983, with Philip Morris piling up a 21% gain. In contrast to the myths about diversification, cigarette revenues continue to dominate company profits. In turn, these have been used to make cigarette plants more efficient and profitable, and to finance diversification, chiefly into the food and beverage industries.

In the latest effort RJ Reynolds Industries and Nabisco Brands have announced plans for a $4.9 billion merger. If the number two tobacco company and the number four food company had been joined in 1984, tobacco would have accounted for 40% of Reynolds' sales, rather than 58%. But tobacco would still contribute about 55% of operating income for the merged companies.

On the other hand, politicians have asked where would the manufacturers be without the rest of the tobacco family—the growers and the quota owners, not to mention the extended family of bankers, farm suppliers, auction and warehouse workers, and a host of tobacco-reliant small businesses. Without these constituencies, it is argued, the manufacturers have little clout in their rear guard against the advancing column of health concerns. When North Carolina Senator Jesse Helms and Representative Charlie Rose talk about stemming tobacco imports and proposals to buy out the surplus leaf, the companies have listened. Whether the arm twisting can produce a down home tobacco family reunion is uncertain. North Carolina state and federal legislators have upped the ante by introducing bills that would tax cigarette manufacturing within North Carolina and that would raise the federal cigarette excise taxes to help finance the flailing tobacco program.

In early June representatives of

growers and manufacturers reached a tentative agreement in Washington. Manufacturers would buy the 1.3 million pounds of surplus burley and flue-cured tobacco; the flue-cured price support would drop from the current $1.70 to $1.40 per pound. For the first time the tobacco buyers would share the program assessment costs with farmers, to insure the stabilization program loans at no cost to taxpayers. The agreement would require federal legislation.



Source: Goldman, Sachs & Co., New York investment analysts

Figure reprinted courtesy of *The Greensboro News & Record.*

MICHAEL HALL

Imports Increase

Current reports from the US Department of Agriculture show a decline in imported foreign leaf for last year. But that was atop a pattern that saw manufacturers increase the percentage of foreign tobacco in an average cigarette from 14% in 1973 to 30% today. In 1983, growers were stunned to learn that for the first time the country actually had imported more tobacco than it had exported. Manufacturers say this occurred because of import stockpiling in late 1983 to avoid increasing customs duties. Regardless, other trends do not suggest an expanding market. For example, manufacturers have systematically reduced tobacco volume per cigarette through rigid manufacturing standards and techniques like homogenizing stem and leaf, and puffing the blend. Since the 1950s, the amount of tobacco per 1,000 cigarettes has decreased from 2.7 pounds to 1.7 pounds, Sumner says.

Such factors are disconcerting to farmer Vollmer, who thinks the companies have "been talking out of both sides of their mouth." Vollmer and his wife Betty worry about their family's future in tobacco; yet, it is hard to conceive of one without it, he tells the health workers in Raleigh. In fact, tobacco income helped build his hometown's new health center. The dilemma is just that direct for some of the citizens, whose fathers, brothers, or grandmothers hold allotments. For others, it is being unable to post no smoking signs in some rural public health clinics. Vollmer says, "It's tobacco that buys the groceries and pays the bills at my house, and without tobacco the town of Bunn, all the time, would look like it does on Sunday morning—dead."

John H. Cyrus, chief of tobacco affairs for the NC Department of Agriculture says, "If we should ever lose tobacco, North Carolina would be the biggest welfare state in the nation."

The stakes are high, but the other side of the coin is gaining attention in tobacco's realm. It is estimated that health care costs for smoking-related illnesses and lost earnings in North Carolina are in the $1.2 billion range. State health statistics indicate that at least 10,000 deaths annually from cancer, heart disease, and lung disease are attributable to smoking.

Unquestionably, the economic and political power of tobacco still dominate the state legislature and congressional delegation. Within the constituent ranks, though, there are heretical murmurings.

In 1984, the NC Council of Churches issued a moral study of tobacco, saying its health effects and economic impact on poor people warrant "alternatives in the economy of the state." In the same year, a small group of Greensboro physicians and nonsmokers—not insensitive to the 2,400 Lorillard employees in the city—placed a newspaper advertisement urging city restaurants to provide nonsmoking sections. Similarly, the Guilford County Medical Society devoted its 1984 professional seminar to respiratory ailments, with several speakers zeroing in on smoking.

Medical Society Opposes Tobacco

In May, days before the "tobacco dilemma" symposium in Raleigh, the North Carolina medical society voted to oppose the use of tobacco and the advertising of smokeless tobacco. Conceding to legislative reality, however, the physicians' house of delegates voted down resolutions recommending an increased state tax on cigarettes, which is the lowest in the nation. One proposal would have used the new revenue to help finance state Medicaid treatment of smoking related illness. Another suggested the funds be used to assist farmers who abandon tobacco.

Ironically, the University of North Carolina at Chapel Hill now commands a lion's share of federal research grants for smoking cessation programs, some $5.6 million. At the nearby National Institute for Environmental Health Sciences, in Research Triangle Park, critical reports have recently been issued on passive smoking and on the effects of smoking on female fertility. Also close to home, Burlington Industries, with headquarters in Greensboro, quietly introduced a smoking cessation effort among its 40,000 employees. Some of them moonlight in Piedmont tobacco fields. The textile giant embarked on the program without fanfare after calculating that an average worker who smokes costs the company $589 annually. Assuming its workforce has the average percentage of smokers, the annual cost to Burlington is about $8 million.

Less dramatic but telling challenges are also coming in the manufacturer's backyards, where for some the leaf's home turf insulation has frayed. A Greensboro woman, who lost her husband to lung cancer last year, just 30 days after the couple's 50th wedding anniversary, posted a no smoking sign on the front door of her house. It is a short drive from the Lorillard plant where high speed, computerized machines are making thousands of cigarettes per minute. "I know what they'll be saying about me, but I don't care," says a soft-spoken, gray haired Eunice Robbins. "I put it up and there it's going to stay. The men have to earn a living, but I always felt there's another way. What I really feel is they are profiting from killing people. They're getting rich off of other people's suffering. I see all these young girls and boys smoking and I just have to shake my head. If only they knew."

TABLES TURNING

Similarly, a quiet skirmish was waged by Charlotte Williams, from her home in Kernersville, a suburb of Winston-Salem. There the names of Reynolds and Winston adorn theaters, parks, and schools. It is the headquarters of the RJ Reynolds Tobacco Co, which employs nearly 16% of the county work force. The company's influence is far from waning, as it completes a $2 billion, 2 million square foot cigarette factory that will produce 7,000 to 8,000 cigarettes per minute. Some RJ Reynolds employees are neighbors of Williams. Nevertheless, last year she wrote Reynolds about its advertising campaign calling for an open debate about smoking. She felt compelled to inform the company "of one more death to add to your list of murders." Her father-in-law had died of lung cancer caused by smoking. "This company does not regard itself as being in any way responsible," responded David Fischel, director of public relations for Reynolds. "We firmly believe that cigarettes have been unfairly blamed as the cause of human disease."

The Winston-Salem/Forsyth County School Board isn't debating anymore. Last year, amid efforts to extend smoking privileges to younger high school students, the board banned smoking for all high school students. Other North Carolina school boards are considering similar policies. In Greensboro, junior high school students have volunteered to be "peer" teachers in classroom instruction on the health effects of smoking. This in a state where, not long ago, children were excused from school to help harvest the leaf.

Lawrence Spohn

Mr Spohn is the medical, environmental, and science writer for the Greensboro News & Record, *Greensboro, NC.*

In September 1984 the News & Record *published an eight-part series "The Tobacco Story," written by Mr Spohn and fellow staff writers Stan Swofford, Greta Tilley, and Beth McLeod. Mr Spohn's article in the* Journal *is based in part on that series, which has received top honors in three national business journalism competitions and was a runner-up for a Pulitzer Prize in 1985.*

Automobile racing: slaughter on tobacco road

"Gentlemen, start your coffins."
Jim Murray
Los Angeles Times

Much compelling material has been published in recent years concerning boxing-related injuries and deaths. The British Medical Association, the American Medical Association, the Canadian Medical Association, and the World Medical Association have all called for the abolition of boxing, as has the Medical Society of the State of New York. The last is significant because the state is the veritable home of boxing, is home to the greatest number of fighters, and has been the site of many championship events in this country.

Indeed, even ardent supporters of the art of self defense have difficulty defending a sport that rewards a participant for striking his opponent in the head and face. The debate over proof of delayed neurologic sequelae due to blows to the head seems unlikely to end in the exoneration of boxing.

One argument often made in defense of boxing is that it is a popular spectator activity among lower income groups and a means of constructive activity and upward social mobility for ghetto youngsters. Many people also believe that boxers are among the best-conditioned athletes in the world.

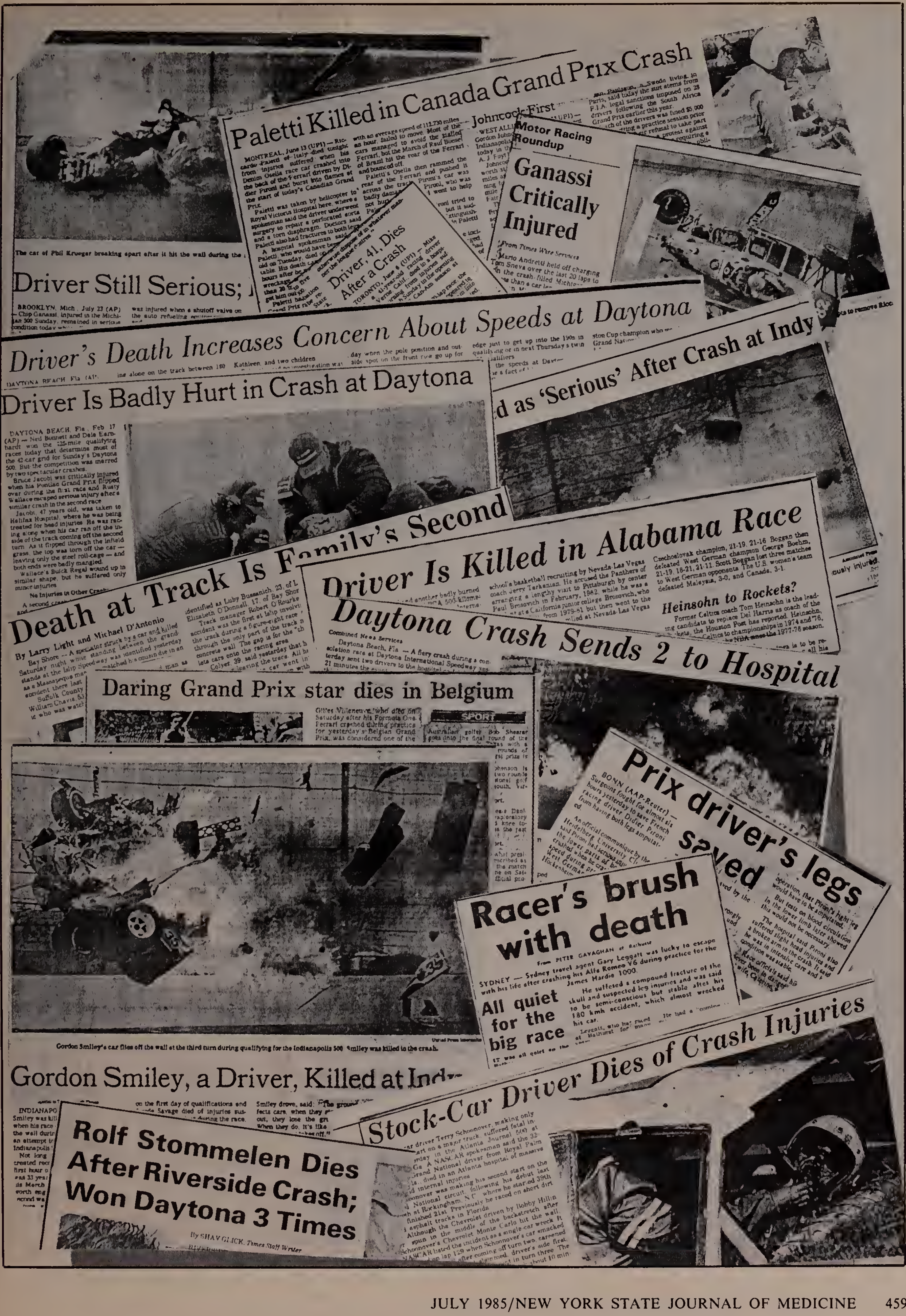
One indication of boxing's self-destruction is the absence of head guards at the professional level, a measure doubtless resisted in an effort to retain an ever-dwindling number of spectators.

No such hope exists for another violent activity that makes boxing look as safe as a church social. Euphemistically called "motor sports," automobile and motorcycle racing have grown in popularity, notwithstanding (or perhaps because of) what may be the highest fatality rate of any human endeavor.

INSIDIOUS HARM

In contrast to widespread attention given to criticism of boxing and calls for its abolition, little has been written about the ongoing lethality and destructiveness of motor vehicle racing. To the contrary (and in contrast to boxing), the amount of coverage given to this activity in the mass media has grown by leaps and bounds. No weekend goes by without a televised showing of an automobile, motorcycle, or powerboat race on either CBS, ABC, NBC, or ESPN. Motorcycle and dirt bike racing are also attracting an increasing number of teenagers and even children. Segments of motor racing are actively directed at children. The June 1985 issue of *National Geographic World* (a magazine for children published by the National Geographic Society) featured a four-page article about pocket bike racing—30 pound 20 inch high motorcycles that reach speeds of 35 miles an hour. Racing of these vehicles has drawn thousands of participants in Japan and the United States, many of whom are aged 8 to 11.

The insidious harm of such activities as pocket bike racing may be to socialize children to extreme risk-taking behavior under the guise of healthy competition and achievement. Although promoters claim that there have been few injuries among these children, this is not the case with motorcycle and



The car of Phil Krueger breaking apart after it hit the wall during the

Driver Still Serious;

BROOKLYN, Mich., July 23 (AP) — Chip Ganassi, injured in the Michigan 500 Sunday, remained in serious condition today

was injured when a shutoff valve on the auto refueling

Paletti Killed in Canada Grand Prix Crash

MONTREAL, June 13 (UPI) — Riccardo Paletti of Italy died tonight from injuries suffered when his Denim Osella race car crashed into the back of the Ferrari driven by Didier Pironi and burst into flames at the start of today's Canadian Grand Prix.

Paletti was taken by helicopter to Royal Victoria Hospital here, where a spokesman said the driver underwent surgery to repair a perforated aorta and a torn diaphragm. Doctors said Paletti also had fractures to both legs.

with an average speed of 112.730 miles an hour, failed to move. Most of the cars managed to avoid the stalled Ferrari, but the March of Raul Boesel of Brazil hit the rear of the Ferrari and bounced off.

Paletti's Osella then rammed the rear of the Ferrari and pushed it across the track. Pironi's car was badly damaged

Driver, 41, Dies After a Crash

Johncock First

Paris, said today the suit stems from F.I.A. legal sanctions imposed on 28 drivers following the South Africa Grand Prix earlier this year.

Motor Racing Roundup

Ganassi Critically Injured

From Times Wire Services

Mario Andretti held off charging Tom Sneva over the last 20 laps to in the crash-filled Michigan

Driver's Death Increases Concern About Speeds at Daytona

DAYTONA BEACH, Fla. (AP)

Driver Is Badly Hurt in Crash at Daytona

DAYTONA BEACH, Fla., Feb. 17 (AP) — Neil Bonnett and Dale Earnhardt won the 125-mile qualifying races today that determine most of the 42-car grid for Sunday's Daytona 500. But the competition was marred by two spectacular crashes.

Bruce Jacobi was critically injured when his Pontiac Grand Prix flipped over during the first race and Rusty Wallace escaped serious injury after a similar crash in the second race.

Jacobi, 47 years old, was taken to Halifax Hospital, where he was being treated for head injuries. He was racing alone when his car ran off the second side of the track coming off the second turn. As it flipped through the infield grass, the top was torn off the car — leaving only the steel roll-cage — and both ends were badly mangled.

Wallace's Buick Regal wound up in similar shape, but he suffered only minor injuries.

No Injuries in Other Crash

'Serious' After Crash at Indy

Death at Track Is Family's Second

By Larry Light and Michael D'Antonio

Driver Is Killed in Alabama Race

Heinsohn to Rockets?

Former Celtics coach Tom Heinsohn is the leading candidate to replace Del Harris as coach of the Rockets, the Houston Post has reported.

Daytona Crash Sends 2 to Hospital

Combined News Services

Daytona Beach, Fla. — A fiery crash during a consolation race at Daytona International Speedway yesterday sent two drivers to the hospital

Daring Grand Prix star dies in Belgium

Gilles Villeneuve, who died on Saturday after his Formula One Ferrari crashed during practice for yesterday's Belgian Grand Prix, was considered one of the

SPORT

Prix driver's legs saved

BONN (AAP-Reuter) — Surgeons fought for almost six hours yesterday to save French racing driver Didier Pironi from having both legs amputated.

But tests on blood circulation in the lower limb later showed this would not be necessary.

The hospital said Pironi also suffered slight head injuries and a broken arm in the crash.

Racer's brush with death

From PETER GAVAGHAN at Bathurst

SYDNEY — Sydney travel agent Gary Leggatt was lucky to escape with his life after crashing his Alfa Romeo V6 during practice for the James Hardie 1000.

He suffered a compound fracture of the skull and suspected leg injuries and was said to be semi-conscious but stable after his 180 kmh accident, which almost wrecked his car.

All quiet for the big race

Gordon Smiley's car flies off the wall at the third turn during qualifying for the Indianapolis 500. Smiley was killed in the crash.

United Press International

Gordon Smiley, a Driver, Killed at Indy

Stock-Car Driver Dies of Crash Injuries

Rolf Stommelen Dies After Riverside Crash; Won Daytona 3 Times

By SHAV GLICK, Times Staff Writer

automobile racing as a whole. When the statistics on auto racing have been compiled and analyzed, they offer a sobering picture seldom reported in the mass media.

According to the Metropolitan Life Insurance Company, among motor vehicle racing drivers (approximately 364,000 racers in 1976—probably a high estimate since some compete in more than one category) there were 67 fatalities (*Statistical Bulletin* 1976; 57:2-4). This information was compiled from newspaper reports, sports agencies, and racing clubs because there is no central register of racing fatalities. Compounding the problem was that apart from Vermont, Connecticut, and New Jersey, all states coded racing deaths under "miscellaneous fatalities."

Since 1918 more than 400 deaths among boxers have been reported. The highest number in one year was 23 in 1953. In the four-year period between 1975 and 1978, 10 deaths occurred in boxers, according to the Metropolitan Life Insurance Company (*Statistical Bulletin* 1979; 60:2-5). The company reported that there were 5,500 boxers in the US in 1978, when four deaths occurred.

In contrast, in just the nine-year period between 1967 and 1976 there were 436 known fatalities among motor vehicle racing drivers (*Statistical Bulletin* 1976; 57:2-4). The large number of racers that is reported is misleading, since many drivers compete in different categories (and are thus counted several times) and many others may enter only one or two races. The lack of official records makes it difficult to learn whether the same individual entering different races is recorded as a different entry.

DEADLY INDY

The situation becomes even more compelling when one considers the fatalities at the Indianapolis 500, the pinnacle of auto racing. As race tracks go, the two-mile oval in Indianapolis is considered one of the "safest" in the world. Yet since the annual Memorial Day event began, 16 drivers have been killed in the race, and 23 other drivers have been killed in qualifying events or in practice. The fatality rate of drivers competing in the Indianapolis 500 between 1955 and 1970 was 35.4/1,000/year (*Statistical Bulletin* 1972; 53:8-9). According to Metropolitan Life this figure understates the hazard to which these racing drivers are exposed. For one thing, most of the fatalities among drivers who race at Indianapolis occur on other race tracks and often in a different type of vehicle from the championship car class driven in the Indianapolis 500. For another, the exposure to the risk of death is overstated, "inasmuch as all drivers whose racing careers have not been terminated either by death or announced retirement have been included in the exposure to the end of the period of observation even though they may have ceased to be active in racing because of age, injuries, or a switch to other sports" (*Statistical Bulletin* 1972; 53:8-9).

In any case, the fatality rate of 35.4 per 1,000 per year is more than 10 times the estimated mortality rate for men of the same age in the general population of the United States (3.0 per 1,000). The *Statistical Bulletin* likens the order of magnitude of death rate among drivers racing at Indianapolis to that of the Marine Corps in the Vietnam War at the height of the battle.

In addition to the 39 racing drivers who have been killed in Indianapolis, 14 mechanics, one guard, and nine spectators have been killed during the race or in practice laps.

BLOOD AND/OR GUTS

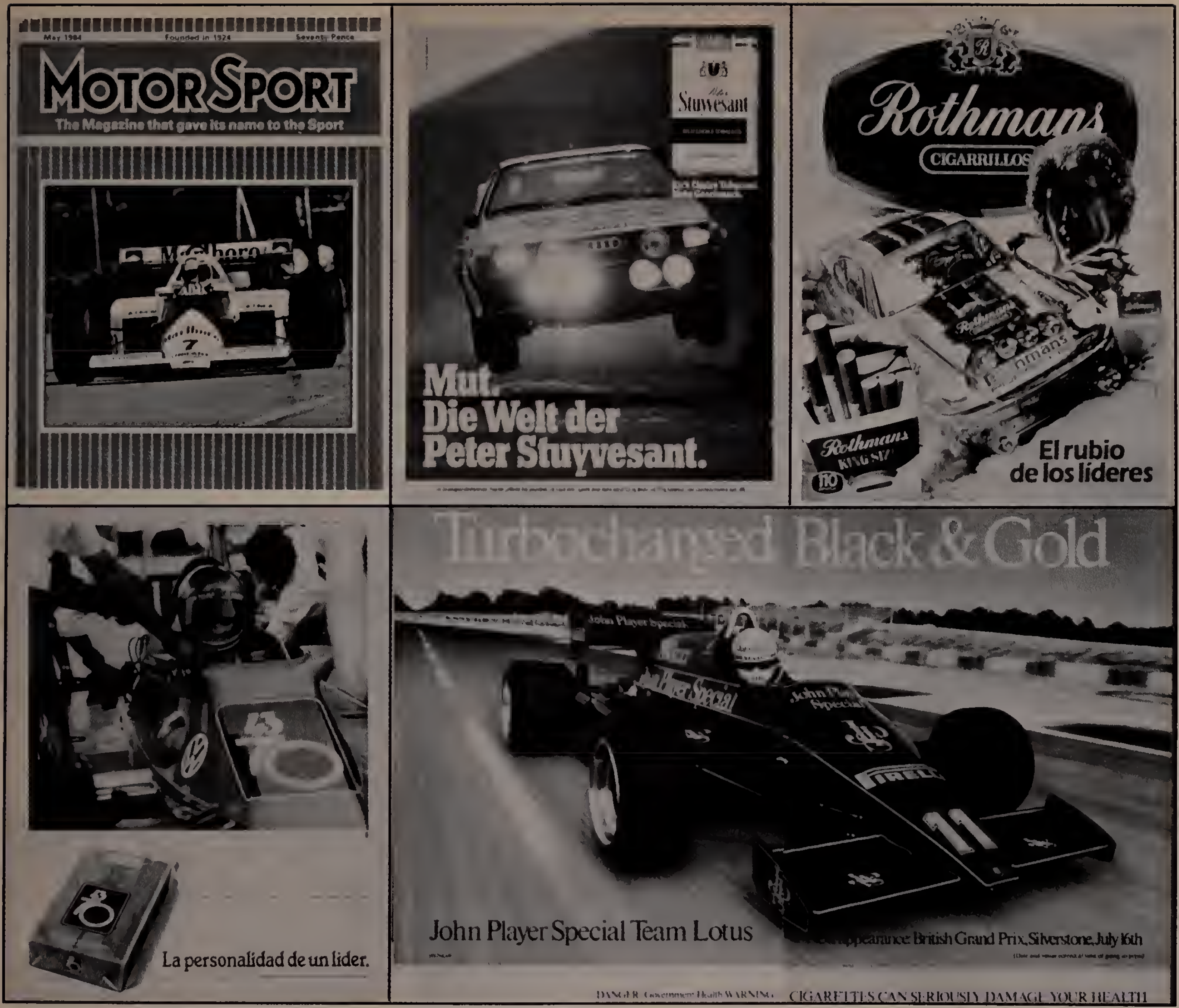
On the prurient level, there is no mystery why so few questions are raised in the mass media about the continuation of motor vehicle racing. Quite simply, some spectators are waiting for the crashes, and the more spectacular the better. The results of these accidents are deaths of the most horrifying kind: decapitation, incineration, dismemberment, asphyxiation, exsanguination. Injuries from the high-velocity impact include severe brain damage, severed spinal cords, shattered bones, rupture of the heart or aorta, avulsion of the lungs, and severe hemorrhage at the site of closed fractures of major limbs (*Practitioner* 1975; 215:178-187). Only a handful of articles have appeared in the medical literature that review the types of deaths and injuries in automobile racing, and no author appears to have called for the curtailment or abolition of this activity. At the annual meeting of the Medical Society of the State of New York in 1984, the violence of automobile racing was touched on in the debate at the reference committee on public health over whether to call for the abolition of boxing. The analogy was dismissed with the argument that automobile racing is a contest of "man versus machine" and does not have *as its object* the knocking down of one human being by another.

Rarely, a sports writer will include a brief comment questioning the purpose or direction of automobile racing. Writing in 1982 about the increasing danger to drivers of Formula One cars (single-seat, open-wheel racing vehicles built to specification—the "formula"—of size, weight, and engine capacity, such cars race in the Grand Prix of Monte Carlo), Australian sports commentator Chris de Fraga noted, "While money continues to pour into Formula One racing, the whiff of the Roman arenas grows stronger. In this case, the gladiators are strapped into 580-kilogram projectiles and hurled around the circuit at dizzying speeds." (*The Age* (Melbourne), June 19, 1982).

The mystery of why this destructive activity has had virtual immunity from serious criticism by sports commentators and the mass media in general becomes clear when one considers that the sponsors of motor racing—of the drivers, the vehicles, the individual events, the race tracks, the administration of racing itself, and, indirectly, the sportswriters themselves—are the largest and most aggressive advertisers in the mass media: tobacco companies, breweries, and automobile manufacturers. To question whether motor racing should exist is to threaten the sales of cigarettes, beer, and cars. Arguments by some writers and spectators that all of life has dangers and risks are not dissimilar to arguments that "everything causes cancer" and "you have to die of something." Thus with impunity newspapers accept ads describing racing as "an orgy of speed" or the race course as a "high speed playground." At least one American newspaper, the *Los Angeles Times* (owner of New York's *Newsday*) has become a major co-sponsor of road races and blends coverage of the event with advertising for it. In 1984 a death occurred during the six hour Grand Prix of Endurance, co-sponsored by the *LA Times* and Datsun, a Japanese automobile company. The *Times* coverage of the race included this description: "[Rolf Stommelen] died of cardiac arrest from the impact of his Porsche 935 knocking over two two-ton concrete barriers and then cartwheeling a hundred yards down the track."

SPEEDING COMMERCIALS

Television has catapulted auto racing


May 1984
Founded in 1924
Seventy Pence
MOTOR SPORT
The Magazine that gave its name to the Sport
Peter Stuyvesant
Mut.
Die Welt der
Peter Stuyvesant.
Rothmans
CIGARRILLOS
Rothmans
KING SIZE
El rubio
de los líderes
La personalidad de un lider.
Turbocharged Black & Gold
John Player Special
PIRELLI
11
John Player Special Team Lotus
appearance: British Grand Prix, Silverstone, July 16th
DANGER: Government Health WARNING: CIGARETTES CAN SERIOUSLY DAMAGE YOUR HEALTH

Automobile racing and cigarette brands: belabored fantasy of life in the fast lane. (Advertisements from popular magazines in Germany, Spain, Argentina, and the United Kingdom.)

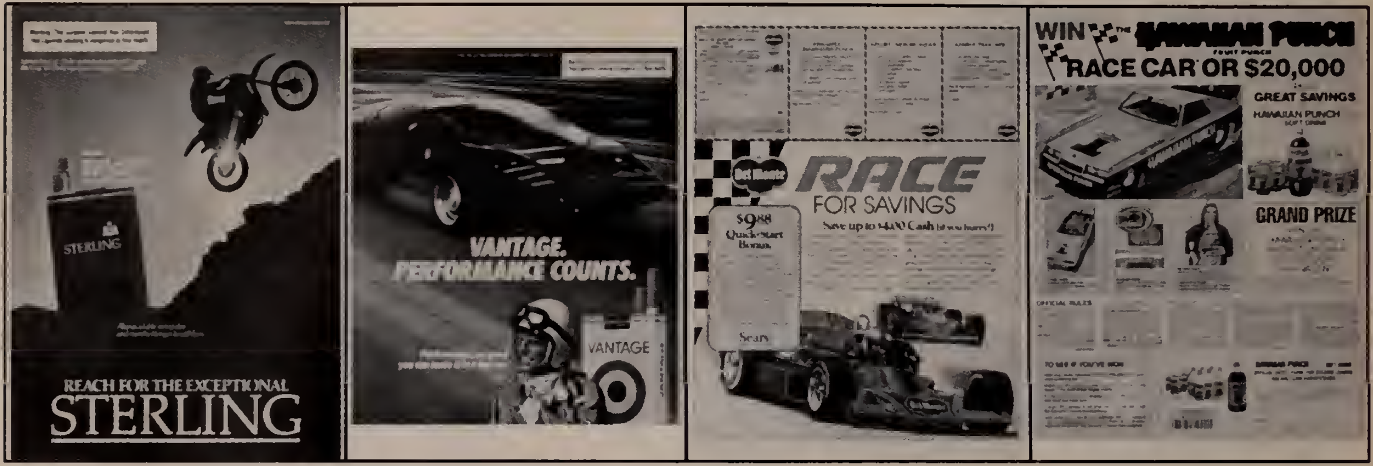
into big-time sport status. In its three-hour, tape-delayed, prime-time coverage of the Indianapolis 500, ABC employs over 200 personnel, including seven announcers, and uses 26 cameras, including several mounted on the rollbars of the vehicles. The TV formula hardly varies: unusual camera angles to highlight the Doppler effect; instant replays of crashes and out-of-control cars; admiring profiles of the drivers; frequent mentions of the sponsors (in addition to the sponsors' commercials); interviews with physicians at the hospital who express hope that the injuries were not fatal to the driver; constant shots of anxious wives, girlfriends, and mothers; and even more shots of the even more anxious pit crew wearing the sponsors' logos.

The networks are hard-pressed to find variety because there is none. What one is watching are, in the words of one advertising agency, "200 mile per hour billboards." An advertisement in a financial newspaper seeking sponsors for the Australian Grand Prix noted that the race would provide eight hours of live national television, an audience of millions, and other multi-media coverage before and after the event. Sponsors can advertise on the car, the driver, a sign around the track, or supporting events. "The short-term benefits are considerable," noted the ad, "Long term potential is enormous." Television advertising of cigarettes is banned in Australia, but among the leading auto racing partnerships in recent years has been that of General Motors and Marlboro (Philip Morris).

In 1981, *Advertising Age,* the magazine of the marketing field, described the attraction of television automobile racing and other sports for certain sponsors: " 'When an advertiser gets involved in sponsorship, the company usually wants a fixed audience in a situation it can control,' a tobacco industry analyst said. That usually comes in the form of a marathon or a stock car race or a rodeo. Tobacco companies each spend millions of dollars sponsoring sporting events" (*Advertising Age,* Jan 19, 1981).

If there is any doubt that automobile racing is little more than a pure commercial, one has only to consider the comments of the 1985 Indianapolis winner, Danny Sullivan, who speaks of a goal to become a spokesman for Miller Beer, made by his sponsor, Philip Morris (*Advertising Age,* May 23,

Through its Camel and Winston cigarette brands, RJ Reynolds has become the principal sponsor of automobile and motorcycle racing in the United States. Advertisements for several other Reynolds products help reinforce the social acceptability of motor racing—and, by association, cigarette smoking.



1985). The sponsorship costs are staggering (and are therefore limited to a handful of sponsors, usually of mass consumer products): $10 million to field a race car team; $5 million to buy the advertising space on the car; $1 million a year to keep the team going; and $300,000 for the ad space on the helmet. The driver can sell his chest, back, and shoulders, and the higher the logo on the driver's clothing, the more money the sponsor pays. On May 27, 1985 the front page of *The New York Times* featured a photograph of the Indianapolis 500 winner Sullivan with his Marlboro and Miller chest patches. Photographs of such racers make their way into bicycle-racing magazines aimed at young boys, many of whom now don fast-selling replicas of the billboard-like jackets worn by motorcycle racers and race car drivers.

BEYOND TRANSPORTATION

Ironically, in 1955 automobile companies agreed not to promote the speed and horsepower of automobiles or to support automobile racing, out of concern for public safety. Almost immediately this rule was violated, so that by the mid-1960s some companies were putting up $30-$40 million a year (*The Car Culture,* Cambridge, MIT Press, 1975, p 215). *Indianapolis News* sports editor Wayne Fuson confirms that the companies made such a pact."They did, but they cheated on it behind their back," he told the *Journal.* "Now they're in with both feet." Indeed, the theme of increasing numbers of automobile commercials is likening the performance of the family car to that of the race car. The use of the automobile as a means of transportation plays little part in commercials. A different kind of risk-taking associated with automobile racing has occurred in the past decade with the influx of cigarette and alcohol sponsors. The move occurred shortly after the ban on television advertising of cigarettes. This has upset Joseph G. Barkey, MD, an anaesthesiologist in Findlay, Ohio and one of the few physicians to have driven race cars professionally. "I talked to [a top executive of] RJ Reynolds at the Mid-Ohio Sports Car Course," Barkey told the *Journal,* "and I asked him why they were sponsoring the races. He said we cannot buy space on TV but we can have our name on an entire car and the wall, and we're covered by *Wide World of Sports,* so we get our ads on TV." Barkey added that "there's no way in God's green earth that Budweiser could afford to buy the exposure it gets," citing the use of such drivers as Paul Newman who have worn the beer company logo. He also cited Burt Reynolds' Skoal Bandit (United States Tobacco) race car. "Some of these guys don't smoke or drink," noted Barkey, "but the kids don't know that." And racing has become popular among children. Several toymakers, including Mattel, manufacture racing cars with Marlboro decals (*Med J Aust* 1983; 1:238). One video arcade game, "Pole Position" has featured race cars with Marlboro logos (*Med J Aust* 1983; 1:242).

In 1975, Brown & Williamson Tobacco Company began sponsoring a race car—the Viceroy Special—with the same colors as a package of Viceroy cigarettes (*Advertising Age,* Jan 6, 1975). Drivers such as Mario Andretti and Al Unser were dressed in the Viceroy colors. The cigarette company backed up its racing efforts with advertising in male-oriented magazines and auto racing books. One of the advertisements featured a race car driver standing by the Viceroy special and the caption: "After going 212 miles per hour, he's not about to smoke a boring cigarette. Viceroy. Where excitement is now a taste."

Since 1975 RJ Reynolds and Philip

Morris have provided a large share of the sponsorship of automobile racing. Reynolds sponsors several entire categories of racing under the title of the Winston Cup and Camel GT. The race tracks often are painted in the colors of the respective brand of cigarettes. Almost all of Reynolds' products have been tied in to the theme of auto racing (Fig), including Hawaiian Punch, Canada Dry, Kentucky Fried Chicken, Del Monte, and the cigarette brands Sterling, Vantage, Camel, and Winston. Advertising for Hawaiian Punch race car toys has appeared in the comic strips of Sunday newspapers.

DYING LEGENDS

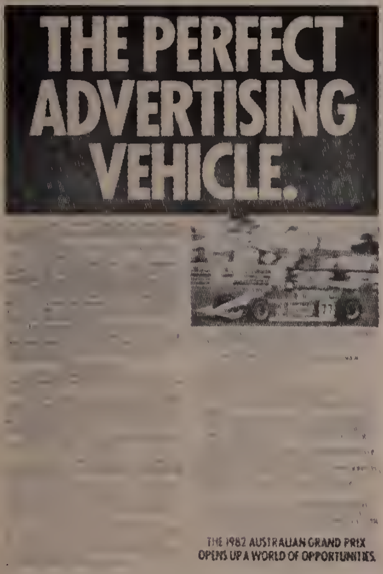
No sportswriter or TV commentator (and few civic organizations) has raised questions about the mixed message of alcohol product logos emblazoned on the clothing of speeding, risk-taking drivers (eg, Bobby Allison and Danny Sullivan for Philip Morris' Miller Beer, Al Hobert for Philip Morris' Lowenbrau, and others for Coors, Strohs, and Budweiser). And only in Australia has some progress been made in challenging the use of cigarette logos on race cars in televised events. A hopeful sign in that country is the dropping of a cigarette company by a race driver. Unfortunately, in the United States auto racing is being promoted at an increasing clip. In the past five years, civic leaders in Dallas, Detroit, and Miami, and most recently New York City have permitted Grand Prix promoters to hold races in once-peaceful parks or on public thoroughfares. Physicians and all others who care about preserving the remaining safety, serenity, and beauty of their communities would do well to oppose the use of public property for automobile racing.

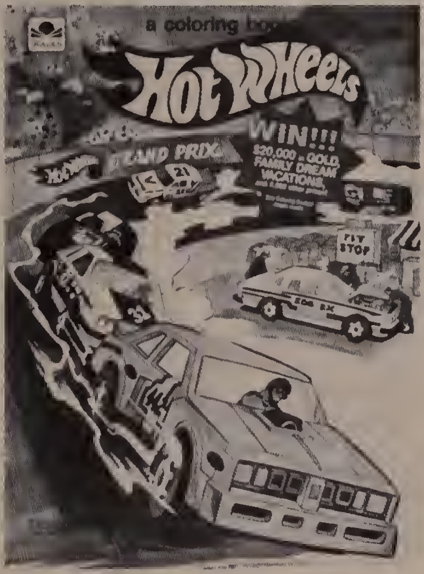
Television networks have been challenged to reduce the amount of violence. Nowhere could they do better than to phase out automobile racing and the obsession with car chases on such programs as the *Dukes of Hazzard* and *Miami Vice*. (Auto races sponsored by cigarette makers and breweries have even been shown on early morning television, pre-empting a children's program [*NY State J Med* 1984; 84:60].)

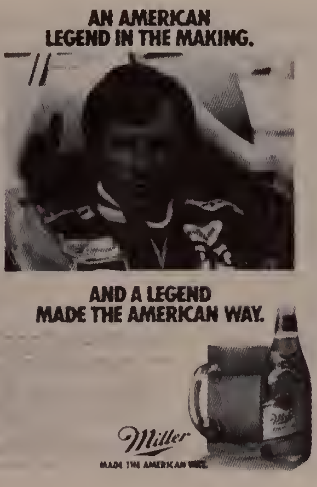
If a ban on auto racing from the airwaves cannot be realistically hoped for in the near future from the networks, then the networks should be challenged by the Federal Communications Commission and the Federal Trade Commission to respond to the violation of the Public Health Cigarette Smoking Act that precludes the advertising of cigarettes on TV. The time is long overdue for the House Subcommittee on Health to hold hearings on the flaunting of the law by television networks and their cigarette advertisers.

As for the future of automobile racing itself, it is tragic that this activity has become a model of risk-taking behavior. It is no exaggeration to suggest that adolescent afficionados of automobile racing believe that they are only as powerful as their gas pedal foot.

Alan Blum, MD


THE PERFECT ADVERTISING VEHICLE.
THE 1982 AUSTRALIAN GRAND PRIX OPENS UP A WORLD OF OPPORTUNITIES.




AN AMERICAN LEGEND IN THE MAKING.
AND A LEGEND MADE THE AMERICAN WAY.
Miller
MADE THE AMERICAN WAY.

Top left, Australian Grand Prix promoters promise television exposure for advertisers in spite of ban on tobacco ads. Top right, In Australia children can purchase toy race cars with Marlboro cigarette logos, made by Mattel (Hawthorne, CA). Mattel's "Hot Wheels" race cars are advertised on television programs aimed at American children. (Another manufacturer of such items is Gay Toys, Inc (Walled Lake, MI), which makes a Skoal Bandit Racing Team car and truck.) Bottom left, Through ads for Winston cigarettes, RJ Reynolds helps teach drag racers the rules of the road. Among the sponsors of the Skoal Bandit Racing Team are *Newsweek*, e-z wider cigarette rolling paper, Goodyear Tires, Valvoline, Sears DieHard batteries, and Hilton Hotels. Bottom right, Philip Morris-sponsored race driver Danny Sullivan dresses up in Miller Beer uniform in ad in *New York Times*. Who says alcohol and driving don't mix?

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042.*

Tobacco related diseases among native Americans

TO THE EDITOR: The *New York State Journal of Medicine* issue of December 1983, devoted to a thoughtful analysis of the world cigarette pandemic, was truly a classic review. One overlooked aspect was the growing incidence of tobacco related disease among native Americans. Despite the traditional use of tobacco during special ceremonies among many of the more than 250 American Indian tribal groups, there have been remarkably few habituated smokers.

An example of the type of ceremonial use of tobacco by the largest of the American Indian tribes (as told to me by a former director of the Navajo Medicine Man's Association) is as follows: In a "smoking ceremony" to aid an ill person, the medicine man would place tobacco and corn husks in a pipe. The patient was then instructed to inhale deeply until it caused vomiting, and also often diarrhea. The aim was to thus clean out the body from both ends. The toxicity of tobacco was thus turned into constructive use as a purgative.

In the mid 1970s, however, the increased daily cigarette smoking by American Indians (especially in Alaska, Oklahoma, and the Dakotas) was leading to an increased incidence of lung cancer, chronic obstructive pulmonary disease, and coronary artery disease. Great attention has since been directed by physicians of the Indian Health Service to vigorous antismoking education. There is still a low prevalence of habitual smoking among Southwestern Indians. If the spread of regular cigarette use, with its consequent negative health impact, can be halted, there is still an opportunity to prevent the upsurge of cigarette induced diseases secondary to the world cigarette pandemic which has emerged among so many groups and peoples elsewhere.

A positive first step was recently taken by the Keams Canyon (Arizona) USPHS Indian Hospital on the Hopi Indian Reservation. With full staff and community support, the hospital is now 100% free of tobacco smoke.

LELAND L. FAIRBANKS, MD
Director, Office of Continuing Education
Clinical Support Center
Indian Health Service,
Phoenix, AZ 85016

Hospital smoking policy

TO THE EDITOR: In the autumn of 1983 the Executive Committee of the 26-bed Public Health Service Indian Hospital in Keams Canyon, AZ decided to adopt a policy that prohibits smoking in the facility. We drafted such a policy (see box) and officially approved it in December 1983. The members of the Executive Committee include the Director of Nursing, the Director of Com-

Keams Canyon Service Unit, Administrative Services Policy

Subject. Smoking and use of other forms of tobacco.

Purpose. To provide a safe and healthful environment for patients and employees.

Background. Cigarette smoking has been definitely determined to be hazardous to one's health by the Surgeon General who, moreover, advises avoiding smoke-filled rooms.

The Department (DHHS) recognizes the rights of individuals working or visiting DHHS controlled buildings to an environment reasonably free of contaminants. The department also recognizes the rights of individuals to smoke provided such action does not endanger life or property, cause discomfort or unreasonable annoyance to non-smokers, or infringe upon the rights of others.

In May of 1974, the Arizona State Legislators enacted a law prohibiting *"Smoking in certain public areas"* (Section 36-601-01, Arizona Revised Statutes) and provided penalties for violations of the law.

Inhaling secondhand smoke ("sidestream" or passive smoking) is an irritant to children, asthmatics and other persons with respiratory illnesses, and has been shown to cause unhealthy changes in otherwise healthy people.

Fire hazards exist when cigarettes are not properly extinguished and when patients smoke in bed.

Policy. Smoking or the use of any form of tobacco is prohibited within the confines of the hospital and all annex buildings of the Keams Canyon Indian Hospital, including the Second Mesa Medical and Dental Clinics.

Smoking or the use of any form of tobacco is permitted outside the confines of the buildings except in areas clearly marked as "No Smoking," such as the oxygen storage dock, fuel storage tanks, inflammable material storage areas, or other designated areas.

Procedure. All entrances to the hospital, annex buildings, and Second Mesa clinics will be posted with signs clearly stating: "PLEASE Do Not Smoke Or Use Tobacco In This Building Thank You." No smoking signs will be visibly posted in the elevators, stairwells, hallways, offices, and throughout public locations of the hospital. Appropriate receptacles will be provided only at entrances for disposing of cigarettes, cigars, or other forms of tobacco.

All employees of the hospital are permitted and authorized to enforce this policy.

1. Employees throughout the hospital will be expected to enforce the policy with courtesy and diplomacy extended at all times. Supervisors will include this policy in the orientation of all incoming staff.
2. Inpatient nursing staff will enforce the policy with all inpatients and visitors. To the extent possible, the tobacco-use policy will be explained to all inpatients.
3. The Clinical Director will provide notice and enforcement of this policy with all visiting clinical professional staff. Department Supervisors will provide the same with all other visiting staff.
4. Violators will be asked to either dispose of the tobacco substance or to leave the building until the tobacco has been discarded.

In order to establish a health care environment which discourages smoking and which reinforces non-smoking practices among patients, employees and visitors, the following will be enforced:

1. Free distribution of tobacco will not be allowed.
2. Tobacco will not be sold on the hospital grounds.
3. Professional personnel and staff will be prohibited from using tobacco substances in the presence of patients.

munity Health Nursing, the Administrative Officer, a social worker, the Clinical Director, and the Service Unit Director.

Little opposition was raised to this policy because smoking is considered unacceptable behavior in Hopi and Navajo Indian society. The few smokers on the staff have either stopped smoking or now refrain from smoking while at work. Visitors have complied with the policy and have noticed the smoke-free atmosphere. We encourage other public and private hospitals to adopt similar policies to provide a healthy environment and to demonstrate the attractiveness of a smoke-free atmosphere.

CHARLES NORTH, MD
Clinical Director
Public Health Service Indian Hospital
801 Vassar Drive NE
Albuquerque, NM 87106

Dr North was Service Unit Director, Public Health Service Indian Hospital, Keams Canyon, AZ from July 1982 to July 1984.

Registry of health facilities that fail to provide clean indoor air

TO THE EDITOR: Hospitals are supposed to encourage patients to make healthful choices. Drug-withdrawal programs are supposed to help patients choose drug-free living. But when it comes to smoking cigarettes, which the head of the National Institute on Drug Abuse has called America's number one drug abuse problem, there is frequently an absence of concern in health care facilities. In 11 years' work as an advocate for nonsmokers' rights, I have learned of numerous instances in which such facilities have abdicated their role as reinforcers of healthful behavior. The following are examples:

- A psychiatric hospital in New York that specializes in drug-withdrawal programs confronts addicts about their poor choices and challenges the families to consider whether they may be helping to reinforce drug use. But this hospital refuses to curtail or even question smoking by patients and staff.
- In Philadelphia a therapeutic center for teenagers with drug-related problems, emotional difficulties, and academic failure proclaims that it is a drug-free community. It will not accept patients who take prescription drugs, including lithium. In the list of items incoming residents may not bring, the center lists drugs. But in the list of things to bring, the center suggests two cartons of cigarettes a month.
- For infractions of minor rules by residents, a psychiatric hospital in New Jersey with a rigorous drug-withdrawal program punishes patients who smoke by having them forfeit two cigarettes. Those who do not smoke must wash dirty dishes as punishment.
- In New Jersey a woman with bronchitis cannot find a psychiatric hospital where she can go for withdrawal from antidepressant medication; all of the hospitals she has investigated are smoke-filled.
- In Paramus, NJ, a man undergoing treatment for Parkinson's disease had to leave the hospital because the smoke was intolerable.
- In Veteran's Administration hospitals throughout the United States public money is spent to buy cigarettes for patients.
- A nurse in central New Jersey is trying to be transferred to the critical care unit of her hospital; she is missing work because of asthma induced by smoke in the ward where she works.

A national registry of such conduct by hospitals and treatment centers is being started by New Jersey GASP (Group Against Smoking Pollution). It is hoped that bringing episodes of this kind to the attention of legislators and health officials will lead to more healthful attitudes and policies in medical institutions.

REGINA CARLSON
Executive Director
New Jersey GASP (Group Against Smoking Pollution)
105 Mountain Avenue
Summit, NJ 07901

On several occasions the American Hospital Association (AHA) was invited to submit a commentary on smoking and hospitals for possible inclusion in this issue. The AHA declined to do so "at this time."

—Editor

Use of *Journal* in schools

TO THE EDITOR: "The World Cigarette Pandemic" issue of the *Journal* has greatly increased interest at our institution in this problem. The influence of advertising on induction of dependence on cigarettes, and the relationship of smoking to cancer, lung disease, and heart disease was the focus of a one-hour special visit by me and 20 first-year medical students on April 30, 1984 at New Rochelle High School. We used slides made from the December 1983 issue of the *Journal* to show the high school students how much the cigarette industry is doing to get them to smoke.

This year we are going to visit the Albert Einstein Jr High School, also in New Rochelle, to bring these insights to younger children. Twenty first-year medical students who have been studying about the world cigarette pandemic will come along, and we will break up into discussion groups as we did at New Rochelle High School for discussions in small groups.

ROBERT BASES, MD
Chief
Radiobiology Section
Albert Einstein College of Medicine
1300 Morris Park Ave
Bronx, NY 10461

Inappropriate magazines for the waiting room

TO THE EDITOR: I would like to endorse the suggestions made by Richards (*NY State J Med* 1983; 83:1958–1959) for turning the office waiting room into a place for health promotion rather than tobacco promotion. In my rural practice I incorporated many of the DOC (Doctors Ought to Care) waiting room strategies. Patients were enthusiastic about the health care materials, but I did notice that some patients were embarrassed to be seen reading sensitive materials in the waiting room. I therefore placed the same materials in the examination rooms. When I entered these rooms, I often caught patients off guard. They would hide the materials that they were reading, on such subjects as peptic ulcers, breast cancer, or Alzheimer's disease. Patients may need to be reeducated about physicians' interest in serving as their personal health consultants.

Dr Richards lists 26 magazines that do not contain tobacco advertisements and are therefore suitable for the waiting room. Of these, only three rank in the top 25 American magazines as rated by total revenue. Clearly, it does not yet pay for them to exclude tobacco. I have recently spent an afternoon in the public library determining what percent of the total advertising in the most popular magazines is for tobacco (Table). This is not meant to be a scientifically designed sample, but may help physicians to recognize the leading "health violators." Of special note is the high percentage of tobacco advertising in the "Sunday magazines" such as *Parade* and *Family Weekly*. These publications have a large audience,

Twenty Most Widely Circulated American Magazines

Magazine	Publisher	Estimated 1983 Average Circulation[1]	Estimated 1983 Advertising Revenue[1] (in thousands of dollars)	Estimated Percent Tobacco Advertising
Parade	Newhouse Newspapers	23,168,789	168,564	40%
Reader's Digest	Reader's Digest, Inc	18,118,068	101,649	0%
TV Guide	Triangle Publishing, Inc.	17,170,799	269,422	25%
Family Weekly	Gannett	12,675,921	102,509	37%
National Geographic	National Geographic Society	10,492,039	27,379	0%
Better Homes and Gardens	Meredith Corp	8,032,373	109,038	18%
Family Circle	New York Times Co	7,251,531	118,830	17%
Woman's Day	CBS, Inc	6,987,317	105,865	22%
McCall's	McCall Publishing Co	6,317,793	64,413	10%
Good Housekeeping	Hearst Corp	5,406,959	127,243	0%
Ladies' Home Journal	Family Media, Inc	5,226,575	57,423	12%
National Enquirer	National Enquirer, Inc	4,890,644	28,300	23%
Time	Time, Inc	4,667,469	298,612	12%
Playboy	Playboy International	4,229,824	60,562	14%
The Star	New Group Publications, Inc	3,757,065	19,675	44%
Penthouse	Penthouse International Ltd	3,476,994	25,793	27%
Newsweek	The Washington Post Co	3,030,780	218,723	18%
Cosmopolitan	Hearst Corp	3,011,190	81,270	6%
People	Time, Inc	2,741,029	175,515	23%
Sports Illustrated	Time, Inc	2,476,147	188,222	11%

[1] Based on data in *Folio*, Folio Publishing Corporation, 1984.

reaching a total of 60 million households each Sunday (*Advertising Age*, Jan 24, 1985:28). This is about the size of television's Super Bowl audience. Tobacco companies can place a full-page color advertisement in each of these 60 million homes for less than a penny per home. These magazines frequently publish articles that are directed to the teenage audience, from which new smokers are captured.

Physicians might refer to this information when their magazine subscriptions are up for renewal. If the publishers are informed that the subscription is being cancelled because of their advertising policy towards tobacco, then some changes may be made. Meanwhile, as copies of these magazines continue to be delivered, I would suggest buying a large lettered rubber stamp and stamping "Smoking Stinks" or some similar statement in red ink across these advertisements. An entire week's issues can be stamped by an office person in a few minutes. This counter advertising can be equally effective, as it will cause the reader to pause and consider the physician's caring, reinforcing message.

PAUL FISCHER, MD
Department of Family Medicine
Medical College of Georgia
Augusta, GA 30912

Progress against smoking in Kuwait

TO THE EDITOR: In the past three decades there has been an enormous increase in the production and consumption of cigarettes in the Eastern Mediterranean region. No fewer than 14 countries in the Middle East now have cigarette factories. Shipments of imported cigarettes have also increased. However, since 1980, following a conference of the General Secretariat of the Board of Health Ministers of the Arab Countries of the Gulf, an extensive smoking control program has been initiated in Kuwait and the States of the Arabian Gulf.

As recommended by the World Health Organization Expert Committee on Smoking Control Strategies in Developing Countries, there are two main categories of smoking control measures: those leading to changes in practice among persons engaged in the manufacture, promotion, or sale of cigarettes (for example, promotional bans); and those leading to changes in practice among smokers, such as restrictions on smoking in public places.

In Kuwait (population 1,358,000), tobacco taxes have been increased by 30%, and limits on tar and nicotine levels have been imposed on imported cigarettes. All tobacco advertising in public places has been removed by the Kuwait municipality. The Ministry of Social Affairs has issued a decree banning any sporting event or public festival sponsored by cigarette companies, and the Ministry of Commerce and Industry has banned the distribution of gifts that encourage smoking and has ordered a new warning statement on every pack of cigarettes ("Smoking is a main cause of cancer, lung disease, and cardiovascular diseases"). The Ministry of Information has held a meeting with the heads of the local press to discuss stopping cigarette advertisements.

A special watchdog committee made up of individuals from a variety of governmental and community organizations has been formed to monitor the smoking control regulations passed by the Kuwaiti Council of Ministers. Recently the nation's first smoking cessation clinic opened. The clinic's program employs both individual and group therapy, and patients may be referred to client hospitals or other health centers for further treatment.

In 1980 the Kuwait Society for Smoking and Cancer Prevention was formed to coordinate numerous governmental and non-governmental activities

in the field of smoking control. The group produces television programs and arranges for public awareness seminars, poster exhibitions in hospitals and health centers, and educational projects for school children and the public at large.

In 1983 Kuwait was the site of the first UICC (Union Internationale Contre le Cancer) Gulf Regional Workshop on Cancer Education in Schools, and the Kuwait Society for Smoking and Cancer Prevention translated and edited the UICC publication, *Teachers' Guide to Cancer Education in Schools.* In cooperation with the Ministry of Health, they designed a school-based program to discourage smoking.

ABDUL RAHMAN AL-AWADI, MD
Minister of Public Health

HUSSAIN A. AL-MUMEN, MD
General Secretary
Kuwait Smoking and Cancer Prevention Society
Ministry of Public Health
PO Box No. 5
Kuwait-Arabian Gulf

The Sudan: physicians act against cigarette advertising

TO THE EDITOR: In the Sudan (population 20,539,000), foreign tobacco companies have suffered a major defeat. Pressure from the Sudan Society of Physicians resulted in the passage, in January 1983, of the Regulation of Smoking Act, which banned all cigarette advertising. Until then enormous billboards for Philip Morris' Marlboro and British American Tobacco's (BAT) Benson & Hedges cigarettes were visible throughout the capital city of Khartoum. Philip Morris attempted to circumvent the new act by promoting Marlboro cigarette lighters in otherwise identical advertisements. These were eventually removed. The act also mandated the repainting of all tobacco company cars, previously emblazoned with cigarette advertising slogans and brand colors.

The popularity of Benson & Hedges, which had captured more than 90% of the import market, was costing the Sudanese economy £6 million in hard currency each year. Imports of American tobacco for blending with local brands cost another £1 million. The value of imported tobacco had increased sevenfold since 1976, which put further pressure on an economy aggravated by the dramatic fall of the Sudanese pound against the dollar. In addition, a considerable amount of tobacco was smuggled across the Red Sea from Saudi Arabia in exchange for valuable livestock, grain, and gum arabic.

Thus more than one motive prompted the act. Economic reasons were as compelling as the health consideration. Restricting foreign companies was a stimulus to the national cigarette company. The chief blow for the importing companies came in 1984 with a decree that the tar content of cigarettes could not exceed 15 mg. Every consignment of imported cigarettes was required to include a statement issued by a recognized laboratory certifying the tar content. Since imported cigarettes apparently exceeded 15 mg of tar, sales virtually ceased (although these cigarettes can still be bought at exhorbitant prices on the black market). With BAT, Philip Morris, and Rothmans in disarray, Gallahers (subsidiary of American Brands) entered the field with its Silk Cut and Sovereign brands, which contained 15 mg of tar. However, a rumor soon circulated that the cigarette paper used was made of plastic, which could result in impotence and infertility. It was also rumored that the British gave these cigarettes to prisoners to reduce sexual urge. The general consensus was that the rumors had been started by a rival cigarette company.

Sudanese doctors deserve praise for pressing their government to take preventive action against cigarette smoking. The country is beset by more immediate health problems such as malaria, bilharzia, and malnutrition, but the physicians recognize that action now may help to avert the epidemic of smoking-related diseases currently suffered by many industrialized nations. The Sudanese have not been smoking long enough to develop high rates of lung cancer, since the incubation period may be as long as 30 or 40 years. But myocardial infarction is increasingly seen among the business and professional classes in Khartoum, and many patients from the Sudan, Egypt, and other Arab nations now come to London for coronary bypass surgery.

KEITH BALL, MD
Department of Community Medicine
The Middlesex Hospital Medical School
London, England NW10 7NS

Dr Ball is vice president of Action on Smoking and Health (ASH), in England, and has visited several developing countries in an effort to combat smoking. His account of the attempts to control smoking in Pakistan appeared in The Lancet *of December 17, 1983 (2:1413). Another individual who has made a major contribution in an Arab country is Mrs Jihan Sadat of Egypt, who has long fought for a nationwide ban on cigarette advertising (see the* Journal of the Addiction Research Institute, *September 1, 1980). An additional recommended reference is an editorial in* The Lancet *(1983; 1:1256-1257) entitled "British and African hearts."*

—Editor

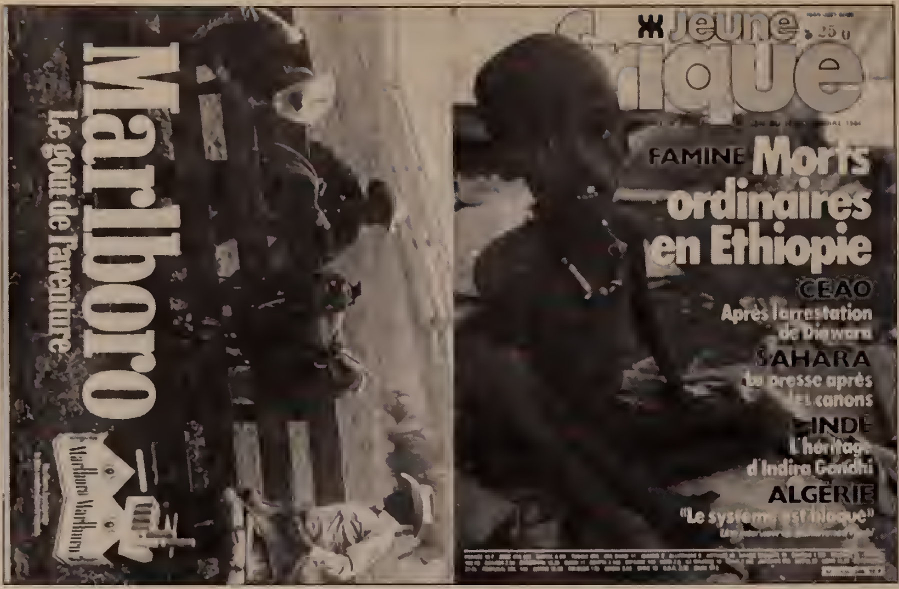
Cigarette advertising in the Third World

TO THE EDITOR: Your December 1983 issue has inspired me to become more active in the fight against the promotion of smoking in the Third World.

While working in West Africa for three years, helping to organize vaccination programs, I encountered many cigarette advertisements, but few people who appreciated the dangers of cigarette smoking. I offered one young man who worked for me a 10% raise if he stopped smoking, to no avail. During one discussion of the dangers of smoking, he remarked that the Marlboro man was really impressive. That remark caused me to reevaluate the power of advertising.

In the United States we are bombarded with so much advertising that we generally consider it ineffective. Upon returning to the United States from a stay overseas in a developing nation, one realizes the impact of advertising, especially on children.

It might seem at first that an Ameri-


Marlboro
Le goût de l'aventure
Jeune Afrique
FAMINE Morts ordinaires en Ethiopie
CEAO
Après l'arrestation de Diawara
SAHARA
La presse après les canons
INDE
L'héritage d'Indira Gandhi
ALGÉRIE
"Le système est bloqué"

Cover of French magazine *Jeune Afrique* shows a victim of the famine in Ethiopia. Back cover uses American imagery to sell cigarettes.

can cowboy would be an unlikely symbol to appeal to African children. Consider the picture of a sun-bronzed cowboy on a handsome horse rounding up fat, healthy cattle. To a rural African child, this is wealth and prosperity. In many villages, if there is a horse at all it belongs to the village chief. Most herdsmen go on foot, and most cattle are very thin.

The tobacco companies are going to great lengths to promote cigarette smoking, with billboards that are reaching more and more deeply into rural Africa. More sophisticated, urban-based decision makers and trend-setters are not being ignored. *Jeune Afrique* (Young Africa) is a French-language news magazine similar to *Newsweek* or *Time*. A typical weekly issue contains nine pages of full-color, glossy advertisements for cigarettes—more than half the total advertising space.

Cigarette vendor near entrance to the National Hospital in Ouagadougou, Burkina Faso, West Africa. Cigarettes can be purchased singly for the equivalent of 2¢ to 4¢ or by the pack for 37¢ to 57¢.

After many years, the World Health Organization was successful in obtaining an agreement with infant formula manufacturers to refrain from advertising. A more vigorous attempt to ban cigarette advertising is needed. A skull and crossbones and a warning in the country's official language should occupy one full side of every cigarette pack sold.

Unlike the struggle against malaria, smallpox, measles, or famine, the control of the cigarette pandemic does not require enormous sums of money or resources. It simply requires wisdom and cooperation among health workers and policy makers in many countries, who must generate the political and social determination to discourage smoking. Cigarette companies should be revealed as the merchants of death that they are, and the promotion of their products should be outlawed.

Health workers in each country must determine what strategy is most appropriate in a given social and political environment. An important step forward in making more health workers aware of the dangers of smoking and moving them to action would be the translation and distribution of the documentary *Death in the West* and all or part of the December 1983 issue of the *Journal* in French and Spanish. I would be interested in working with others to help coordinate such an effort, as well as in developing anti-smoking counter-advertising programs patterned after the technique pioneered by DOC (*NY State J Med* 1983; 83:1377).

DAVID C. SOKAL, MD
Epidemiologist
Medical Care Development, Inc.
s/c Ambassade des États-Unis
B.P. 35
Ouagadougou
Burkina Faso
West Africa

The tobacco industry and legislative redress

TO THE EDITOR: Debates in legislative bodies regarding restrictions on smoking in public places are not only drawn out processes, but also unequal contests between David and Goliath. On one side the cigarette companies can afford almost unlimited resources for presenting their interests and views, while the initiators of the anti-smoking measures are mostly volunteers backed up "only" by the medical findings and a sound public opinion shared by smokers and non-smokers, according to a 1983 Gallup poll.

In Israel, when the restrictions concerning smoking in public places and cigarette advertising were discussed in the Knesset (Israeli parliament), the manufacturer and importer of cigarettes hired two lawyers to represent them, both former Ministers of Justice and members of the parliament.

As yet, attention has not been focused on the fact that the tobacco companies may deduct lobbying expenses from their income tax, and, consequently, share these lobbying expenses with the general tax-paying public.

In the United States a provision of the Internal Revenue Code (section 162(e)) allows the deduction of expenses for types of activities relating to promoting or combating legislation, such as those in connection with appearances before, sending communications to, or sending statements to legislative bodies. Those who suffer from passive smoking—the non-smoking majority of the population—are also those who share a considerable portion of these deductable lobbying expenses with the cigarette companies. The amounts involved are very likely of high magnitude, although at present the companies are not required to disclose these figures to the public.

This and other laws applicable to the cigarette industry suggest that when a certain product, the production and sale of which are lawful, is harmful, potentially addictive, and capable of causing suffering to non-users exposed to its byproducts as well as users, current laws must be modified. Among such legal provisions would be the imposition of personal liability on directors of companies who authorize the production and sale of this harmful product while aware of the dangerous consequences.

In order to achieve the highest level of effectiveness and enforcibility of these laws throughout the world, international cooperation is required. Therefore it is suggested that legislators who truly care about the health of their constituents should start proposing changes without delay. Simultaneously, an international treaty adopting these laws should be drawn up by those countries who are concerned about discouraging smoking. The idea that appropriate legislative measures are ineffective in the elimination of smoking is a myth created by the cigarette companies. An extensive program of legislation can achieve far-reaching results.

AMOS HAUSNER
Legal Counsel
Israel Society for the Prevention of Smoking
3 Metudella St
Jerusalem, Israel

Chronic tobacco use confounds the diagnosis of thyroid illness

TO THE EDITOR: A growing number of studies has shown alterations in the metabolism and disposition of endogenous substances in chronic cigarette smokers.[1-3] Reductions in several hormone concentrations in heavy tobacco users have led us to investigate serum thyroid hormone levels in cigarette smokers.[4-7] Our findings show reduced triiodothyronine (T_3) and thyroxine (T_4) levels in chronic heavy smokers (33 ± 1.5 cigarettes/day).[8] Whether these changes are induced by the direct action of a particular tobacco component or are indirect effects caused by the induction of hepatic microsomal enzyme systems remains to be clarified.

Studies conducted in our laboratories with rats implicate nicotine or its metabolites in the reduction of thyroid function. We have found that administration of N′-oxide diastereoisomers, formed in nicotine metabolism, have an effect on thyroid function similar to that noted in human subjects who smoke. After chronic administration of either pure *trans*-nicotine-N′-oxide or a 64%/36% *trans/cis* diastereoisomeric mixture of nicotine-N′-oxides, a profile of thyroid hormone levels was observed that was markedly similar to the "low T_3 syndrome" observed in humans. This syndrome characterizes a variety of systemic illnesses in which T_3 neogenesis is impaired, resulting in low serum T_3 levels and correspondingly elevated reverse T_3 concentrations.

We also observed the in vivo back conversion of these N′-oxides to nicotine in the treated rats. This raises the possibility that the observed changes in thyroid status may be related to nicotine toxicity rather than nicotine-N′-oxide metabolites. Furthermore, the *trans* isomer seems to be more readily back converted than *cis* nicotine-N′-oxide. In vitro studies indicate a 2:1 *trans:cis* ratio of these diastereoisomers.[9]

These findings have relevance for the diagnosis of thyroid hormone related disorders and present the possibility that observed reductions in serum T_3 levels in chronic heavy cigarette smokers may actually be the result of non-thyroid related illness caused by the effects of nicotine.

Of relevance, then, are other nicotine-containing products, such as chewing tobacco. Aids to stop smoking, like nicotine chewing gum, which is designed to aid in cigarette withdrawal, produce mean steady-state plasma levels of 11.8 ng/mL to 23.2 ng/mL of nicotine or the equivalent of a cigarette containing 1.1 mg of nicotine.[10]

Since these data implicate nicotine or its metabolites in thyroid depression, the use of any nicotine containing product should be questioned when a diagnosis of "low T_3 syndrome" is not accounted for by an obvious disorder.

D. W. SEPKOVIC, PhD
N. J. HALEY, PhD
E. L. WYNDER, MD
Naylor Dana Institute
for Disease Prevention
American Health Foundation
Department of Nutrition
and Endocrinology
Dana Road
Valhalla, NY 10595

1. Pomerleau OF, Fertig J, Seyler LE, Jaffe J: Neuroendocrine reactivity to nicotine in smokers. *Psychopharmacology* 1983; 81:61-67.
2. Husain MK, Frantz AC, Crarochi F, Robinson AF: Nicotine stimulated release of neurophysin and vasopressin in humans. *J Clin Endocrinol Metab* 1983; 41:1113-1117.
3. Divers Jr WA, Wilkes MM, Babaknia A, Yen SSC: Maternal smoking and elevation of catecholamines and metabolites in the amniotic fluid. *Am J Obstet Gynecol* 1981; 141:625-628.
4. Sloss EM, Frerichs RR: Smoking and menstrual disorders. *Int J Epidemiol* 1983; 12:107-109.
5. Wilkins JN, Carlson HE, Van Vunakis H, Hill MA, Gritz E, Jarvik ME: Nicotine from cigarette smoking increases circulating levels of cortisol, growth hormone, and prolactin in male chronic smokers. *Psychopharmacology* 1982; 78:305-308.
6. Shaarawy M, Mahmoud KZ: Endocrine profile and semen characteristics in male smokers. *Fertil Steril* 1982; 30:255-257.
7. MacMahan B, Trichopoulos D, Cole P, Brown J: Cigarette smoking and urinary estrogens. *N Engl J Med* 1982; 307:1062-1065.
8. Sepkovic DW, Haley NJ, Wynder EL: Thyroid activity in cigarette smoking. *Arch Intern Med* 1984; 144:501-503.
9. Gorrod JW, Jenner P: The metabolism of tobacco alkaloids, in *Essays in Toxicology* 1975; 6: 35-78.
10. McNabb ME, Ebert RV, McCusker K: Plasma nicotine levels produced by chewing nicotine gum. *JAMA* 1982; 248:865-868.

Ad agency conflict: selling cigarettes and dentistry

TO THE EDITOR: In September 1984 the American Dental Association (ADA) House of Delegates voted against awarding $12.5 million to the Leo Burnett advertising agency to create a television advertising campaign intended to increase the number of visits to dentists and to improve oral health. Although the principal reason for the defeat of the proposal was cost (annual dues would have increased by $125), opposition was also raised because of an irreconcilable conflict-of-interest: the major client of Leo Burnett is Philip Morris, the nation's largest advertiser of cigarettes. Leo Burnett is responsible for the creation and perpetuation of the macho cowboy image for Marlboro cigarettes and the ubiquitous billboard slogan, "Come to Marlboro Country."

I am one of the ADA members who attempted to point out to the association many weeks prior to the vote (in a letter to *ADA News* that was not printed) that awarding a contract to such an advertising agency might have subjected the ADA to public ridicule once word had reached other health professionals and the press. Someone was bound to have suggested as the slogan for the campaign, "Come to cavity country." (In addition, the actor chosen by the agency as spokesman in the commercials is also the television advertising spokesman for a chain of tobacco shops.)

Responding to my objections over the choice of Leo Burnett, the ADA insisted that all major advertising agencies accept cigarette accounts. This is untrue. The trade publication *ADWEEK* (March 15, 1982), identified several large agencies that refuse to accept such accounts. These include Della Femina, Travisano & Partners (New

York); Scali, McCabe, Sloves (New York); McCaffrey & McCall (New York); and Jennings & Epstein (Atlanta).

The dental profession has strongly opposed smoking. Meanwhile the tobacco companies and their advertising agencies have worked ever harder to sow confusion concerning the adverse health consequences of smoking. Is there any doubt that an advertising agency that derives a major portion of its revenues from pushing cigarettes at young people would have avoided suggesting that the ADA step up its efforts to increase public awareness of the association between smoking and oral cancer, periodontal disease, stained teeth, and halitosis? Indeed, it is worth recalling a well-documented episode that occurred in 1979 when Leo Burnett leaked word to Philip Morris of an impending advertising campaign of another of its clients, Allstate Insurance. At that time Allstate was about to introduce its Healthy American Life Insurance Plan, television commercials for which highlighted a 2.5% discount for non-smokers. According to *Advertising Age* (May 12, 1980), a Philip Morris vice-president, James Bowling, recruited the Tobacco Institute and the Tobacco Growers Information Committee of North Carolina to pressure Allstate not to run the campaign. Such pressure may have led Allstate to curtail its advertising campaign and the emphasis on the non-smokers' discount.

For any organization of health professionals or a manufacturer of health-related products or services to select an advertising agency that also promotes cigarettes raises serious ethical concerns.

DOUGLAS LEE CHESTER, DDS
1226 Burnet Avenue
Union, New Jersey 07083

Dr Chester is president of New Jersey GASP (Group Against Smoking Pollution).

Smoking prevalence rates for Western Australian adolescents

TO THE EDITOR: The Australian Council on Smoking and Health recently investigated smoking prevalence rates for adolescents in Western Australian schools. Responses on cigarette smoking and drinking of alcohol were elicited through a 23-item questionnaire, which was administered by trained interviewers during August 1984. The sample involved year 7–12 (12 to 17 year old) students in 65 government and non-government primary and secondary schools in both the country and the metropolitan area. Within each school, students were selected at random from class lists. Of the 3,028 children surveyed, results for 3,020 were analyzed.

Adolescent Smoking Prevalence Rates—1983, 1984 (Percentages)

	Year				
	7	8	9	10	(Adults)*
Girls (1984)	2	10	16	27	30
Girls (1983)**	3	11	18	26	
Boys (1984)	3	12	16	26	37
Boys (1983)**	6	15	16	23	

* Hill and Gray, Community Health Studies, 1984, vol 8, no 3.
** Health Department of Western Australia, 1983.

Smoking rates for adolescents are high, with prevalence rates for regular 15-year-old smokers approaching those for adult smokers. In years 7 and 8, however, there has been a substantial fall in prevalence rates since 1983. Such a decline should be interpreted with the knowledge that the Health Department of Western Australia's "Smoking Prevention Programme for Schools" was introduced into year 7 of all primary schools in the state in September 1983. Increased teacher awareness of smoking control through teacher in-service, the programme itself, and the Health Department of Western Australia's "anti-smoking" mass media campaign may well be responsible for this decline in prevalence rates.

RUTH E. SHEAN
Director
Australian Council on Smoking and Health
705 Murray Street
West Perth, 6005
Western Australia
Australia

The Australian Council on Smoking and Health wishes to acknowledge the financial support of the Cancer Foundation of Western Australia, and the Alcohol and Drug Authority for this research, conducted on behalf of the Anti Cancer Council of Victoria, as part of a national survey on smoking prevalence.

As found in previous Australian studies, brand preference correlated with the intensity of advertising campaigns for certain brands. —*Editor*

Death in the West curriculum project

TO THE EDITOR: In 1976, Thames Television of Great Britain produced a 27-minute documentary, *Death in the West,* that contrasted the Marlboro man image with that of six actual cowboys who were dying of smoking-induced diseases. The film included interviews with two executives from Philip Morris, manufacturers of Marlboro, who presented the tobacco industry's position that there is no convincing evidence that cigarette smoking is harmful. This is perhaps the only time that executives from a cigarette company (as opposed to public relations officials from the Tobacco Institute) agreed to interviews in a documentary.

Rather than simply presenting a procession of diseased lungs, *Death in the West* approached the subject of smoking by turning topsy-turvy the most successful advertising image ever created: the Marlboro cowboy.

The most important aspect of *Death in the West* developed after the film aired in Britain in September 1975. When word got out that the documentary was under consideration for broadcast on the most watched news program in the United States, CBS' *60 Minutes,* Philip Morris sued Thames in a British court in order to prevent the film from ever being shown again.

A legal settlement was reached in 1979, when Thames and Philip Morris entered into a consent decree in England under which Thames agreed to turn over all copies of the film to Philip Morris, together with the material used to make the film and never to show or grant permission for the film to be shown. Moreover, the entire settlement was to be secret. Essentially, Philip Morris not only suppressed the film, but it also suppressed the fact that it suppressed the film.

This legal action removed *Death in the West* from public circulation until May 1982, when KRON-TV, the NBC affiliate in San Francisco, obtained a videocassette of *Death in the West* and broadcast it in prime time. Since then, television stations across the United States have shown *Death in the West* to large audiences (although no New York station has aired the film).

In 1983, the California Nonsmokers' Rights Foundation (CNRF), working with the University of California (UC), Berkeley, developed a five-day curriculum keyed to the film for school children. The project, which grew from a five-year research contract between the National Cancer Institute and the Lawrence Hall of Science at UC, Berkeley, was aimed at communicating the addictive nature of cigarettes and the false nature of cigarette advertising. CNRF distributed curriculum guides to about 3,000 teachers in conjunction with a third airing of *Death in the West* on KRON-TV, and reached about 90,000 children.

Short-term follow-up of a sample of 583 sixth grade students demonstrated a significant increase in knowledge about the hazards of cigarette smoking and a significant decline in the credibility of the cigarette manufacturer ($P < .01$). Knowledge and attitudes were measured in a 50-item questionnaire that was administered to 335 study subjects and 248 controls one week prior to the curriculum and two weeks afterward.

Most importantly, it made the Marlboro cowboy less attractive to the children. This last result is especially noteworthy, since half the children who admitted to smoking said they chose Marlboro.

Death in the West curriculum projects have been implemented throughout the country, in schools in Arkansas, New Mexico, North Carolina, and Pennsylvania, with a total of about 300,000 children involved, aided by grants from corporations and foundations.

Death in the West is also used in several of the pre-clinical and clinical courses in the UC-San Francisco, School of Medicine and other medical schools and continuing education programs, and in various smoking cessation clinics.

Individuals and organizations interested in sponsoring local projects can obtain more information by contacting the California Nonsmokers' Rights Foundation, 2054 University Avenue, Suite 500, Berkeley, CA 94704 (415-841-3032).

Copies of *Death in the West* on both videocassette and 16mm film may be purchased from Pyramid Film and Video, Box 1048, Santa Monica, CA 90406 (213-828-7577 or 800-421-2304).

STANTON A. GLANTZ, PhD
Division of Cardiology
Room 1186 M
University of California
San Francisco, CA 94143

Dr Glantz, who helped bring about the first broadcast of Death in the West *in the United States, is president of the California Nonsmokers' Rights Foundation.*

Attorney General Abrams speaks out against smokeless tobacco

TO THE EDITOR: The Centers for Disease Control estimates that 22 million Americans use smokeless tobacco, and that the average user is between the ages of 18 and 30 years. The federal health agency further estimates that the use of smokeless tobacco has increased at an annual rate of approximately 11% since 1974. In 1983, retail sales in the moist and fine cut tobacco snuff category were estimated at $500 million.

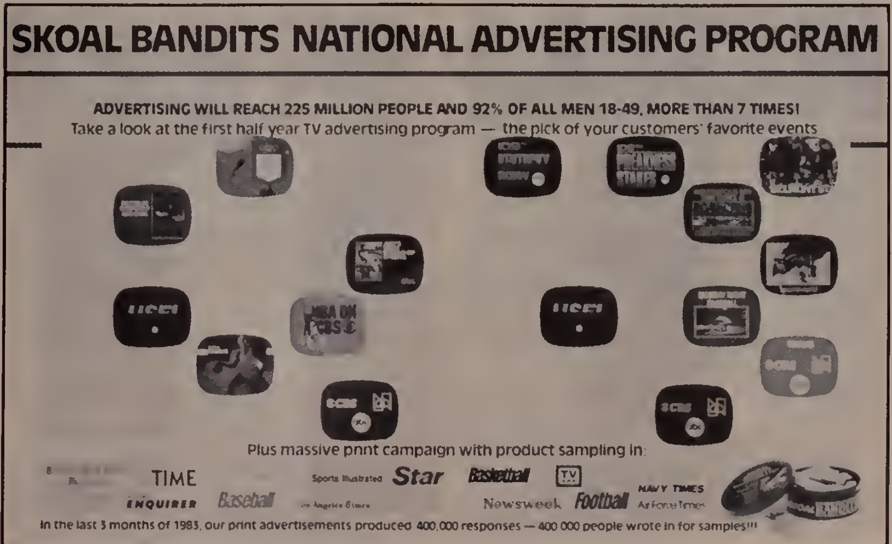
The increasing number of users of smokeless tobacco products is the result of highly financed industry advertising and promotional campaigns which are primarily designed to attract young consumers. Prominent athletes and rock performers star in the commercials, which portray use of smokeless tobacco as socially desirable and as a safe alternative to smoking. Yet, noticeably missing from these advertisements and promotions is any health warning. Such an alert is clearly justified by the large body of respected scientific evidence accumulated over the last 70 years pointing to a definite link between use of these products and oral cancer and other serious adverse health consequences.

The smokeless tobacco industry has shown no disposition to adopt voluntarily a responsible position with reference to health warnings. To the contrary, its advertising aggressively promotes the so-called advantages of these products. Indeed, the industry leader, the United States Tobacco Company, has recently engaged in a multi-million dollar national advertising campaign directly implying that the use of its product is a safe alternative to smoking.

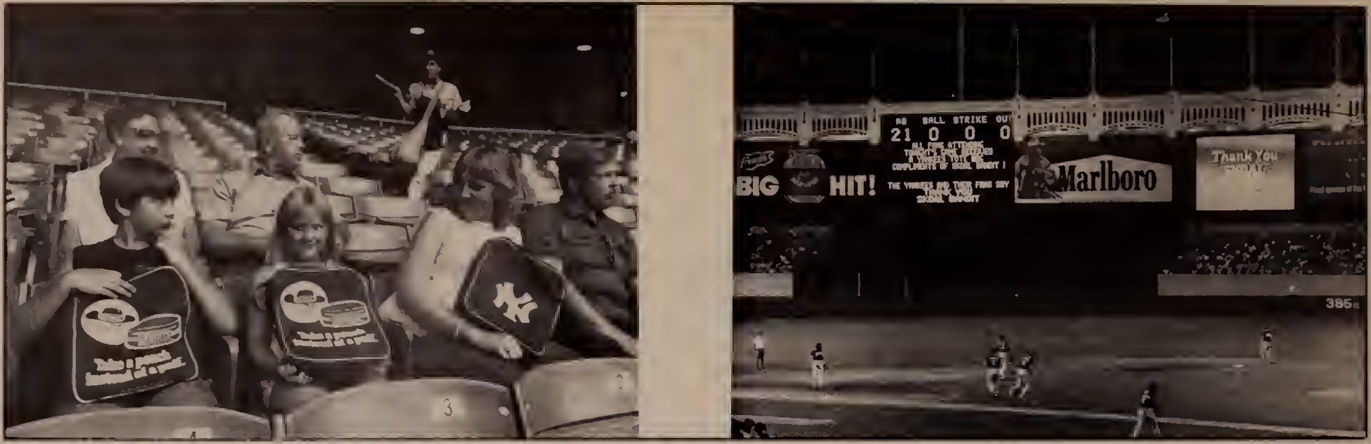
My office took firm action against this company to assure that this false representation will be banned from the

We put the pinch between the cheek and gum of America.
U.S. Tobacco. The smokeless people

Cheeky ad in tobacco magazine.


SKOAL BANDITS NATIONAL ADVERTISING PROGRAM
ADVERTISING WILL REACH 225 MILLION PEOPLE AND 92% OF ALL MEN 18-49, MORE THAN 7 TIMES!
Take a look at the first half year TV advertising program — the pick of your customers' favorite events
USFL
NBA ON CBS
USFL
Plus massive print campaign with product sampling in:
TIME
Sports Illustrated
Star
Basketball
TV
NAVY TIMES
ENQUIRER
Baseball
Newsweek
Football
Air Force Times
In the last 3 months of 1983, our print advertisements produced 400,000 responses — 400 000 people wrote in for samples!!!

United States Tobacco Company putting the pinch on American media.



Children display their Skoal Bandit tote bags given to them in 1984 at Yankee Stadium. The warning labels mandated in a last-minute agreement with Attorney General Abrams were placed on the plastic packaging and not on the bags themselves. The statement that "the slogan 'Take a pouch instead of a puff' is not intended to imply that the use of this product is a safe alternative to smoking" may not have displeased Marlboro cigarette maker Philip Morris. (Photographs: Andrew Skolnick) —Editor

company's advertising and product labels in New York State. It is hoped that the company will withdraw the offending ads and labels in other parts of the country as well. We took further action against this company in 1984 when it sponsored a promotion in which thousands of tote bags bearing the company's offensive slogan were given away to fans at a major league baseball game at Yankee Stadium. Our office required that the company place a conspicuous sticker bearing a disclaimer on each tote bag to the effect that its slogan did not imply that use of its product was a safe alternative to smoking.

In the absence of federal action, as part of my 1985 consumer protection legislative package, I have submitted a bill to the legislature requiring the inclusion of health warnings on advertisements and on product labels for all smokeless tobacco products sold in New York. The warning would alert consumers that use of smokeless tobacco products is hazardous to health. Violators would face a penalty of $5,000 for each violation.

Our proposed bill would also address another vital concern: the industry spends millions in distributing free samples of its products. Such distribution would also be prohibited and would carry a $5,000 penalty for its violation.

Smokeless tobacco is not a safe alternative to smoking, and consumers should not be misled to believe otherwise.

HON. ROBERT ABRAMS
Attorney General
State of New York
Department of Law
Two World Trade Center
New York, NY 10047

In 1985 the New York State Assembly passed a bill to mandate warning labels on packages of smokeless tobacco, but the Senate took no action on the measure, which was endorsed by MSSNY. Among those mobilized by the United States Tobacco Company to oppose the bill and similar legislation around the country is a noted lecturer on alcohol problems, Father Joseph C. Martin. Father Martin, a Catholic priest and president of a resident treatment center for alcohol abuse in Maryland, told the Journal *he had been asked by US Tobacco Company president Louis Bantle (who serves on the board of the treatment center) to send letters in opposition to the labeling of smokeless tobacco as addictive. Father Martin admitted to being uninformed about the hazards of using smokeless tobacco.*

—Editor

Towards smoke-free comic strips

TO THE EDITOR: Newspaper comic strips have been a cherished American tradition since their introduction in the early 1900s. Cartoon characters often have become symbols of American values and role models for children.

Unfortunately, in reflecting our way of life, some comic strips have included characters with unhealthful lifestyles, including smoking. In the 1930s and 1940s especially, many of the young readers' favorite characters in "Smoky Stover," "Gasoline Alley," "Smitty," "Moon Mullins," among others, were drawn with a prominently protruding tobacco product. (Cigarette advertising was also a regular feature of the Sunday funnies during that era.)

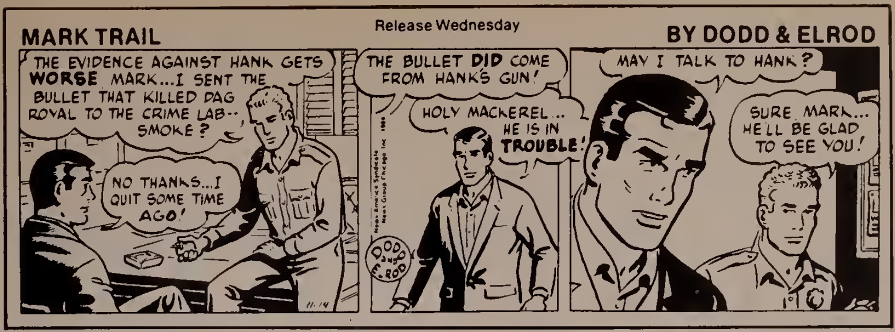
In light of all that has been learned about the effects of smoking since the 1950s, it is regrettable that comic strips such as characters in "Andy Capp," "Beetle Bailey," "Tumbleweeds," "Dennis the Menace," to name a few, persist in portraying smoking as socially acceptable behavior.

My six-year-old son Daniel recently made this point in questioning why his favorite comic strip character, naturalist Mark Trail, was shown smoking a pipe. Encouraged to communicate with the cartoonist, Daniel wrote: "I do not think that Mark Trail should smoke, because smoking is bad to your health and it pollutes the air which could hurt the birds."

This simple complaint made sense to Mark Trail's cartoonist, Jack Elrod, who responded, "Because of your letter, I'm not going to let Mark smoke any more." The pipe soon disappeared from Mark's lips, and within a few months, Mark's friend Cherry commented to him, "You look healthy and happy!" Mark replied, "I feel great. . . . I stopped smoking, and I feel like a new person!"

"Mark Trail" was responsive to Daniel's criticism because it preaches clean living and stresses ecological lessons. Elrod recognizes an obligation to the children in his readership. Will other cartoonists, comic strip syndicates, publishers, and editors be equally sensitive? Or should the newspaper comic pages be rated "R"?

HENRY S. KAHN, MD
Associate Professor
Department of Community Health
Emory University School of Medicine
69 Butler St.
Atlanta, GA 30303


MARK TRAIL
Release Wednesday
BY DODD & ELROD
THE EVIDENCE AGAINST HANK GETS WORSE MARK...I SENT THE BULLET THAT KILLED DAG ROYAL TO THE CRIME LAB-- SMOKE ?
NO THANKS...I QUIT SOME TIME AGO!
THE BULLET DID COME FROM HANK'S GUN!
HOLY MACKEREL .. HE IS IN TROUBLE!
MAY I TALK TO HANK?
SURE, MARK... HE'LL BE GLAD TO SEE YOU!

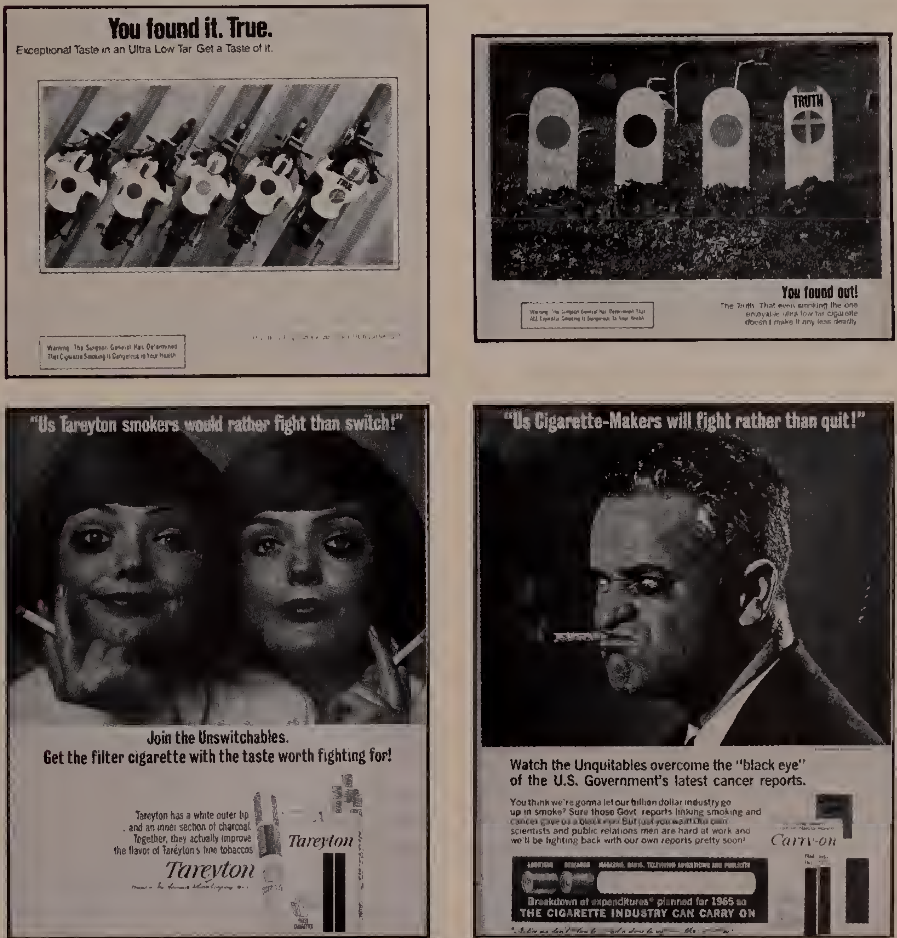
Cartoonist Jack Elrod responds to concern of young reader for the health of Mark Trail and those around him.

One of Europe's favorite cartoon characters, cowboy Lucky Luke, also gave up smoking in 1983, after nearly 40 years. According to World Health *(March 1984), Belgian cartoonist Maurisce de Bevere replaced Luke's cigarette with a hayseed because he realized the hero's smoking set a bad example for youth.*

Credit must be given to cartoonists who have ridiculed the image of smoking and have doubtless helped convince young readers of the absurdity of lighting up. Foremost among the American comic artists and writers who have poked fun at smoking is the staff of MAD Magazine. *Others include Berke Breathed ("Bloom County"), Bill Keane ("The Family Circus") Ralph Dunagin ("Dunagin's People"), Ted Key ("Hazel"), Morrie Brickman ("Small Society"), Mel Lazarus ("Miss Peach"), Johnny Parker ("The Wizard of Id"), Phil Frank ("Travels with Farley"), Morrie Robinson ("The Wee Pals"), Gary Larson ("The Far Side") and Ben Wicks ("Wicks").*

*Editorial page cartoonists have also played a leading role in challenging the power and image of the tobacco industry. Foremost among the American cartoonists who have explored this theme are Wayne Stayskal (*Tampa Tribune*), Herb Block (*Washington Post*), Doug Marlette (*Charlotte Observer*), Don Wright (*Miami News*), Dick Adair (*Honolulu Advertiser*), Jules Feiffer (*Village Voice*), Tony Auth (*Philadelphia Inquirer*), George Fisher (*Arkansas Gazette*), Frank Evers (*New York Daily News*), Tom Darcy (*Newsday*), M.G. Lord (*Newsday*), Bill Mauldin (*Chicago Sun-Times*), Ed Stein (*Rocky Mountain News*), Milt Priggee (Copley Newspapers), Brian Barling (*Christian Science Monitor*), Dick Locher (*Chicago Tribune*), Paul Conrad (*Los Angeles Times*), Tom Engelhardt, (*St. Louis Post-Dispatch*), Bob Englehart (*Hartford Courant*), Etta Hulme (*Fort Worth Star Telegram*), Pat Oliphant (Universal Press), Jimmie Johnson (*Jackson *(Miss.)* Daily News*), Dana Summers (*Orlando Sentinel*), David Horsey (*Seattle Post-Intelligencer*), Ben Sargent (*Austin American Statesman*), Mike Kelley (*San Diego Union*), Doc Goodwin (*Columbus Dispatch*), Ray Orsin (*Cleveland Plain Dealer*), and Tom Gibb (*Arizona Mirros*).*

—Editor


You found it. True.
Exceptional Taste in an Ultra Low Tar Get a Taste of it.
Warning: The Surgeon General Has Determined That Cigarette Smoking Is Dangerous to Your Health
TRUTH
You found out!
The Truth. That even smoking the one enjoyable ultra low tar cigarette doesn't make it any less deadly
"Us Tareyton smokers would rather fight than switch!"
Join the Unswitchables.
Get the filter cigarette with the taste worth fighting for!
Tareyton has a white outer tip and an inner section of charcoal. Together, they actually improve the flavor of Tareyton's fine tobaccos
Tareyton
"Us Cigarette-Makers will fight rather than quit!"
Watch the Unquitables overcome the "black eye" of the U.S. Government's latest cancer reports.
You think we're gonna let our billion dollar industry go up in smoke? Sure those Govt. reports linking smoking and cancer gave us a black eye! But just you wait! Our own scientists and public relations men are hard at work and we'll be fighting back with our own reports pretty soon!
Carry-on
Breakdown of expenditures* planned for 1965 so THE CIGARETTE INDUSTRY CAN CARRY ON

COPYRIGHT 1983, E.C. PUBLICATIONS, INC

COPYRIGHT 1964, E.C. PUBLICATIONS, INC

Satire by *MAD Magazine* helps adolescents see through cigarette ads.

Preventing complications of tobacco use

THE SMOKE RING

By Peter Taylor. 330 pp. New York, Pantheon Books, 1984. $18.95.

A SMOKING GUN: HOW THE TOBACCO INDUSTRY GETS AWAY WITH MURDER

By Elizabeth M. Whelan. 244 pp. Philadelphia, George F. Stickley Co, 1984. $15.95.

CHRONIC EXPOSURE OF MICE TO CIGARETTE SMOKE

By Carol J. Henry and Richard E. Kouri. 187 pp. New York, Field, Rich & Associates, Inc, for the Council for Tobacco Research, 1984.

THE PSYCHOPHARMACOLOGY OF SMOKING

By G. L. Mangan and J. F. Golding. 257 pp. New York, Cambridge University Press, 1984. $49.50.

The previous generation of health workers has documented the enormous burden of disease and death that tobacco produces; it is the current generation's task to apply this knowledge to reduce the morbidity and mortality caused by tobacco in this country and abroad. These four books provide useful background material for the job ahead.

The British journalist Peter Taylor has produced a briskly written book which summarizes the major scientific, economic, and political realities of the tobacco industry. The book begins with an account of the scientific evidence that cigarettes cause cancer, heart disease, and emphysema and bronchitis. He relates this with the story of how the data emerged in the major public health reports on smoking and with several anecdotes about the tobacco industry's own research on the health effects of smoking. Several examples of the industry's denial in the face of clear evidence are presented.

The next chapter summarizes the major features of the great (largely British and American) tobacco companies. The origins and interconnections of the six largest multinational firms are presented.

When Taylor discusses the mass media's habit of downplaying the relationship of smoking and disease, he provides a valuable, behind the scenes account of his own 1976 documentary, *Death in the West.* This film contrasts the stories of six men from the American West who developed major cigarette-associated diseases with promotions for Marlboro cigarettes and interviews with executives from Marlboro's manufacturer, Philip Morris. The film undercuts the seductive appeal of cigarette advertising. Philip Morris succeeded in having the film removed from circulation in Great Britain, but copies have recently become available again. *Death in the West* has become part of several innovative school-based tobacco education programs.

The remainder of Taylor's book contains sometimes over-long accounts of the political maneuvering around cigarettes and health in Great Britain and the United States in recent years. These case studies are nonetheless valuable: the ability of the tobacco industry to gain an advantage in arena after arena is impressive, and Taylor has documented many of the industry's techniques and approaches.

Elizabeth Whelan, a public health worker, is also concerned with the social, political, and economic aspects of the tobacco problem. She shares with Taylor the conviction that active opposition from the tobacco industry and its supporters is the major reason that the abundant scientific evidence of harm from smoking has not yet led to effective control measures.

Whelan's book is organized into three parts. The first three chapters outline the evidence that tobacco is a major cause of disease and highlight 14 "ploys" that the tobacco industry has developed in response to the medical evidence. This analysis should be helpful to people whose major information source on the subject has been the mass media. All too often, a news story containing "bad" news about tobacco and disease is "balanced" with distracting and evasive comments from the Tobacco Institute.

The major portion of the book is devoted to an historical narrative about the problem. This provides a useful overview. For instance, cigarettes only became widely available with the perfection of mass production machinery in 1884. Mass production itself generated the need for mass promotion; cigarette advertising has been an important part of the industry for as long as cigarettes have been produced by machine.

Finally, the third portion of Whelan's book outlines a variety of actions and activities that can help reduce tobacco consumption. The prescription is broad and unfocused, but this is the state of the art. What has been tried heretofore has produced few results. Determining the appropriate strategy and assembling resources to implement it are the major challenges now facing public health. Whelan has summarized the problem and has provided a set of suggestions. It is a beginning.

The books by Carol Henry and Richard Kouri and by G. L. Mangan and J. F. Golding stand in marked contrast to those of Taylor and Whelan.

Henry and Kouri report the results of investigations they conducted while under contract to the Council for Tobacco Research—USA, Inc between 1975 and 1983. The key experiments involved exposing mice to cigarette smoke for up to 110 weeks and observing whether neoplasia occurred in the tracheas or lungs of exposed animals to a greater extent than in unexposed animals. A number of ancillary studies are also described. The main experiments were burdened with parallel investigations that sought to determine whether cigarette smoke exposure was associated with the more rapid appearance of pulmonary neoplasia in mice pretreated with intratracheal instillations of known carcinogens.

Tobacco industry press reports and at least one feature article carried by United Press International have uncritically described the enormous dimensions and conclusions of this undertaking. Over nine years, 10,000 mice were exposed to smoke from 800,000 cigarettes at a cost of $12 million. No cancers attributable to cigarettes were observed. However, the results of these investigations have been published and publicized without the scrutiny of peer review. It is tempting to borrow a phrase from the Tobacco Institute spokesman who was commenting on the classic smoking beagle experiment of Auerbach and Hammond: This reviewer does not, "and the public should not, accept at face value the findings of this study."

The Henry and Kouri experiments featured two major chronic exposure conditions. In the first trial, 2,925 animals were divided into nine subgroups. Only 645 animals were exposed to cigarette smoke and no other carcinogen. Harsh experimental conditions resulted in a high first-year mortality rate. Thus, of the 645 animals exposed to smoke who received no additional carcinogen, only 152 were examined in the second year of study and 13 in the third year.

The second major experiment included 4,226 mice. The 2,053 mice in the smoke-only condition received a dose of cigarette smoke that was just 20% of that in the first experiment. An equipment modification was made to improve the longevity of the animals. Even with the changes in delivered dose and equipment, only 20.5% of the mice exposed to smoke were examined in the second year, and 19.8% in the third year. The last three surviving mice were examined in the first weeks of the fourth year. Additional long-term experiments were designed and funded, but in July 1980 a decision was made to discontinue this part of the work. The final experiments were all of a short-term nature.

A few observations will give the reader some additional clues about this work. The brief explanatory text that accompanies the abundant tables and figures does not explain the protocols followed for preparing and reading the histopathology slides. Nowhere is there an indication that the slides were read by an unbiased investigator, and there is no suggestion that refinements such as examining metaplastic lesions with additional sections to seek small foci of carcinoma were part of the protocol. Histopathology of laryngeal tissue is not reported, and nasal histology was reviewed in less than a third of the 987 animals exposed to smoke that were examined in the second experiment.

Although the ancillary studies include experiments with radiolabelled tracers to calculate the delivered dose of cigarette smoke particulates, the authors should have addressed the issues raised by Wynder and Hoffman concerning the limitations of cigarette smoke exposure experiments in small animals. In addition, it would have been appropriate to discuss the results of these studies in light of the three reports published in a peer-reviewed journal which found respiratory tract carcinomas in other species of rodents chronically exposed to cigarette smoke. It is unlikely that Henry and Kouri are unfamiliar with these reports: two of the coauthors of one paper collaborated with Henry and Kouri on dosimetry studies during the same time period.

In sum, this book scarcely lives up to its advance notices. It clearly represents an enormous effort and a great expense. Publication in the present form leads one to wonder why the authors have not published their work in an appropriate scientific journal.

While Henry and Kouri present the results of a series of animal experiments, Mangan and Golding present an overview of the tobacco problem to develop their psychologically oriented perspective. The book purports to be a review of why people start to smoke and of the factors that sustain continued tobacco use. It also contains a review of the smoking cessation literature and seems directed to people interested in smoking cessation problems. The writing is often muddy, but when the authors confine themselves to the narrow tasks implied by their title, the book provides useful summaries. However, in other areas, Mangan and Golding generate somewhat more heat than light.

In reviewing the literature on smoking and disease, these psychologists give an enormous amount of attention to the hypothesis that one's genetic makeup determines both whether one becomes a smoker and whether one will develop heart disease or lung cancer. The data they cite and laboriously review actually support the conventional hypothesis that smoking causes lung cancer. While they admit this in passing, they imply that there are important alternative explanations when, in a summation at the end of the book, they characterize those who subscribe to this notion as "extreme." They argue that there remain unanswered scientific questions so that "it seems unwise to pre-judge the issue."

One can always imagine additional studies of any scientific subject; whether any are needed before rational action is taken is a matter of judgment. The observations that permitted effective public health control of cholera by avoiding contaminated water supplies preceded the recognition that a vibrio was the etiologic agent. Yet, even in the case of the cholera vibrio, unanswered questions remain which are the subject of considerable, ongoing scientific research.

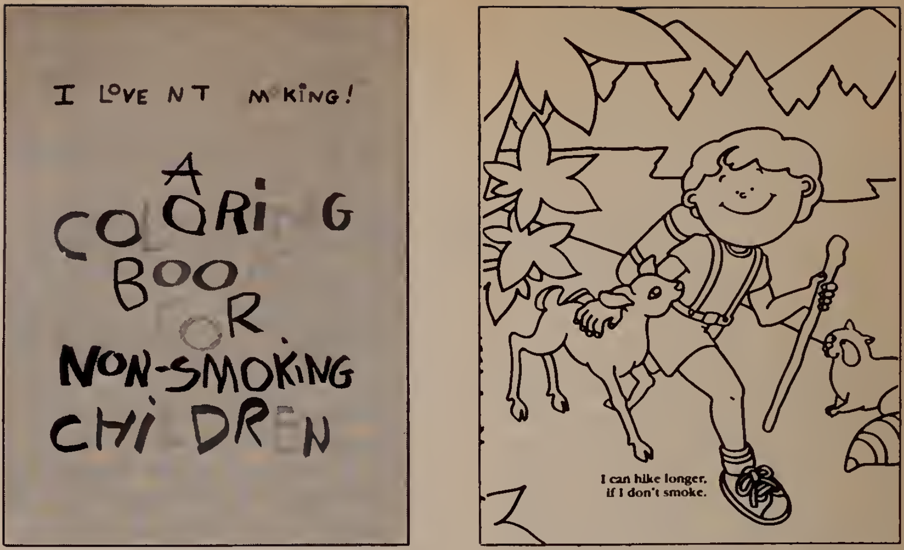
There are other peculiarities in this book. For instance, early in the book, Mangan and Golding seem charmed by the unusual hypothesis that, were it not for cigarettes, other forms of chemical dependency (ranging from alcoholism to narcotic addiction) would be more widespread. In their overview at the end of the book, the closing paragraph refers to this hypothesis: "Does [the polydrug model] imply that if the habitual user is denied his source of supply, for whatever reason (material in terms of price, psychological in terms of health threat), then he will migrate to other, more readily available drugs, a finding which has been reported with heroin users?" They suggest that if tobacco is a part of the polydrug problem, then the tobacco cessation problem is more difficult than commonly supposed. Meeting the challenge would require a broader attempt to change attitudes and activities "to meet what are clearly perceived by the individual as important and compelling needs."

The authors' implication seems clear. Until there is Utopia, society may be better off living in Wonderland: the tobacco merchants are saving millions of people from the consequences of other, more fearsome chemical dependencies.

This odd book may be a bit more understandable upon reflection that the authors were supported in its writing by the Western world's largest tobacco company, British American Tobacco (BAT is represented in the United States by BATUS; the company's tobacco subsidiary here is Brown and Williamson.) Considered in this light, the book is, perhaps, an example of the analyses that tobacco's supporters have developed to convince themselves that the health fuss is yet unsettled and may, in the end, be a false alarm after all.

Taken together, these four books provide an excellent introduction to the tobacco-disease problem. The books are especially important for health care professionals because they provide provocative insights into some of the social and cultural forces that work against efforts to achieve control of the worldwide tobacco-use pandemic. The second two books appear to be examples of some ways in which points of view discredited by an abundance of well-regarded scientific studies and analyses can still be advanced through the skillful use of seemingly scientific procedures, rhetoric, and propaganda.

JOHN SLADE, MD
Department of Medicine
University of Medicine and Dentistry of
New Jersey-Rutgers Medical School
New Brunswick, NJ 08901



This is the book tobacco companies ought to be distributing to back up their claims that they don't want children to smoke.

"I LOVE NOT SMOKING!" A COLORING BOOK FOR NON-SMOKING CHILDREN

By Roma Dehr and Ron Bazar. Illustrated by Sharon Hind-Smith. Published by Namchi United Enterprises, PO Box 33852, Station D, Vancouver, British Columbia, Canada V6J 4L6.

This engaging 30-page booklet is a positive way to reinforce the desirability of not smoking. Physicians, teachers, and parents would do well to give a copy to every five to eight year old they know. Children will identify with the boys and girls who are shown having fun in various activities such as swimming and hiking. A stack of *"I love not smoking!"* ought to be as essential for the pediatrician's or family doctor's office as a supply of tongue depressors. For that matter, every physician who treats children or their parents or grandparents should keep copies on hand for distribution. For an ophthalmologist, plastic surgeon, or orthopedist to hand out a copy of *"I love not smoking!"* may be as powerful an educational moment as when the booklet is presented by physicians who deal more frequently and more directly with cigarette-related diseases. Copies can be purchased in bulk and printed according to the specific needs of the purchaser. *"I love not smoking!"* has been translated into several languages, including French, Swahili, and Cree. Prices are as follows: single copy, $1 (US); 2–10, 75¢; 10–100, 60¢; 100–250, 55¢; 250–500, 50¢.

—AB

RECOMMENDED READING

The Cigarette Underworld. (Updated reprinting of "The World Cigarette Pandemic," December 1983 issue of New York State Journal of Medicine) 128 pp, illustrated. Secaucus, NJ, Lyle Stuart Inc, 1985. $9.95 (paperback)

Selling Death. By Thomas Whiteside. 150 pp. New York, NY, Liveright, 1971. Out of print.

Cigarette Country: Tobacco in American History and Politics. By Susan Wagner. 248 pp. New York, NY, Praeger Publishers, 1971. Out of print.

The Tobacco Industry in Transition. Edited by William R. Finger. 339 pp. Lexington, MA, Lexington Books, 1981. $26.50 (hardcover)

Coffin Nails and Corporate Strategies. By Robert H. Miles in collaboration with Kim S. Cameron. 298 pp. Englewood Cliffs, NJ, Prentice-Hall, Inc., 1982. $13.95 (paperback)

Smoking Psychology and Pharmacology. By Heather Ashton and Rob Stepney. 222 pp, illustrated. New York, NY, Tavistock Publications Ltd, 1982. $8.95 (paperback)

Research and Manufacturing in the U.S. Cigarette Industry. By Max Samfield. 169 pp, illustrated. New York, NY, Lockwood Book Publishing Co. Inc., 1980.

Everybody's Business, An Almanac: The Irreverent Guide to Corporate America. Edited by Milton Moskowitz, Michael Katz, and Robert Levering. 916 pp, illustrated. San Francisco, CA, Harper & Row, Publishers, Inc., 1980. $9.95 (paperback)

The Ladykillers: Why Smoking is a Feminist Issue. By Bobbie Jacobson. 135 pp. London, Pluto Press, 1981. $6.95 (paperback)

Cigarette Marketing in the Third World. By Bo Wickstrom. 88 pp, illustrated. Sweden, University of Gothenburg, 1979. (paperback)

Tobacco and the Third World: Tomorrow's epidemic? By Mike Muller. 112 pp., illustrated. London, War on Want, 1978. (paperback)

Toward a Smoke-Free Workplace. By Regina Carlson. 20 pp. New Jersey, New Jersey Group Against Smoking Pollution, 1985. $5 (paperback)

Staff Report on the Cigarette Advertising Investigation. By Matthew L. Myers, Craig Iscoe, Carol Jennings, William Lenox, Eleanor Minsky, and Andrew Sacks. Washington, D.C., Federal Trade Commission, 1981.

Periodicals that report on smoking

The coverage of smoking by medical journals and the mass media is largely confined to reports and discussions of the adverse health consequences of tobacco use. For a closer examination of the strategies of the tobacco industry to increase demand for its product, it is essential to delve beyond the context of health. The following periodicals provide information on the political, social, legal, agricultural, and economic aspects of tobacco and its promotion and manufacture throughout the world.

Action On Smoking and Health (ASH) Smoking and Health Review
2013 H Street, NW, Washington, DC 20006
Cost: At least a $5 yearly donation (six issues/year)

ASH reports on the attempts of nonsmokers to secure their right to clean air.

(Excellent newsletters are also published by GASP of Massachusetts and New Jersey as well as Californians for Nonsmokers Rights; an updated list of nonsmokers' rights organizations can be obtained from New Jersey GASP, 105 Mountain Avenue, Summit, NJ 07901.)

Advertising Age
Crain Communications, Inc, 220 E 42nd St, New York NY 10017
$55/year (101 issues)

Covers marketing, public relations, and mass media. Cigarette campaigns receive detailed coverage.

Adweek
A/S/M Communications Inc, 820 Second Ave, New York, NY 10017
$50/year (57 issues)

Rival weekly of *Advertising Age.*

ACSH News and Views
1995 Broadway, New York, NY 10023
$10/year (six issues)

Published by the American Council on Science and Health, a nonprofit organization supported largely by industries. Publishes in-depth evaluations of food, chemicals, the environment, and health, with frequent coverage of tobacco. ACSH Executive Director, Elizabeth Whelan, PhD, is the author of *The Smoking Gun* (see p 474).

Multinational Monitor
1346 Connecticut Avenue, NW, Suite 411, Washington, DC 20036
$18/year (12 issues)

Published by Essential Information, a nonprofit, educational research organization that covers issues not normally reviewed in the mass media. Reports on corporate marketing practices around the world, occasionally addressing the tobacco industry.

Smoking and Health Bulletin
US Department of Health and Human Services, Public Health Service, Office on Smoking and Health, Rockville, MD 20857
Free (six issues a year)

The abstracts published in the *Bulletin* represent the most complete world bibliography of research articles and other publications on all aspects of tobacco, including economics, chemistry, agriculture, legislation, epidemiology, advertising, and health. The *Bulletin* is edited by Donald Shopland, leading authority in categorizing the literature on tobacco. The Office on Smoking and Health, directed by Joanne Luoto, MD, prepares the annual Surgeon General's report on smoking, and serves researchers by providing literature searches.

Smoking and Health Reporter
Center for Health and Safety Studies, HPER Building, Rm 116, Indiana University, Bloomington, IN 47405
Free (four issues a year)

Newsletter of the National Interagency Council on Smoking and Health. Highlights both local and national efforts to curtail smoking; culls brief news items from the mass media on all aspects of smoking.

Smoking or Health Update
Canadian Council on Smoking and Health, 725 Churchill Ave, Ottawa, Ontario K1Z 5G7 Canada
Free (four issues a year)

Comprehensive newsletter focusing on strategies to curtail smoking in Canada.

Tobacco International
Lockwood Trade Journal Inc, 551 Fifth Ave, New York, NY 10176
$20/year (26 issues)

Features articles on growing, producing, and distributing tobacco.

Tobacco Observer
The Tobacco Institute, 1875 I Street NW, Suite 800, Washington, DC 20036
Free (six issues a year)

Newspaper of the public relations arm of the tobacco industry, which offers the industry view on research and legislation. Features articles on the history of tobacco in the United States.

Tobacco Reporter
Specialized Agricultural Publications Inc, 3000 Highwoods Blvd, Suite 300, Raleigh, NC 27625
$20/year (12 issues)

Covers economic and manufacturing aspects of the tobacco industry worldwide.

United States Tobacco and Candy Journal
BMT Publications Inc, 254 W 31st St, New York, NY 10001
$24/year (17 issues)

Trade publication for tobacco and confectionery distributors that contains articles and editorials on fostering the growth of cigarette sales.

World Smoking and Health
American Cancer Society, 777 Third Ave, New York, NY 10017
Free (three times a year)

A digest of articles from different countries that was established to facilitate international communication between the quadrennial conferences.

Certain general circulation magazines also report on aspects of cigarette smoking, including Business Week, Mother Jones, The Washington Monthly, *and* Columbia Journalism Review. The Reader's Digest *reprints condensations of material that has appeared in other publications; for instance, an article in the September 1984 issue entitled "Cigarette makers are getting away with murder" used the December 1983 issue of the* New York State Journal of Medicine *as its primary source. Several daily newspapers have published major series on smoking and continue to report in detail on the subject:* The Greensboro News & Record *("The tobacco story," Sept 23–30, 1984), Greensboro, NC 27420-0848;* The Courier-Journal *("Up in smoke: the troubled tobacco industry," Nov 11–15, 1984; "Selling cigarettes," June 12–14, 1983), Louisville, KY 40202;* The Boston Globe *("Battle on smoking intensifies in US," Sept 30–Oct 2, 1984), Boston, MA 02107; and* The Charlotte Observer *(20-page section entitled "Our tobacco dilemma," March 25, 1979), Charlotte, NC 28232. A weekly newspaper in Westchester County, NY,* Suburban Street *(White Plains, NY 10601), publishes front-page articles on tobacco industry trade practices in almost every issue.*

Need A Temporary Physician?
During: vacations, CME's, illness, recruiting.
Want Free Time While You Practice Medicine?
Join CompHealth's Locum Tenens Physician Group.
For further information, call 412-741-3310.

CompHealth A Physician Group
WILSON ROSS, Regional Administrator
114 Centennial Ave., Sewickley, PA 15143

NATIONAL EMERGENCY SERVICES, INC. (N.E.S.)
Directorships, full- and part-time emergency department opportunities are available throughout New York State and the country for primary care physicians with ER experience. Enjoy a variety of practice settings, competitive remuneration, paid malpractice insurance, and flexible scheduling. For information and application materials contact the recruitment department at (516) 365-4010, (Outside N.Y. State 1-800-645-4848) or send C.V. to N.E.S., One Hollow Lane, Suite 304, Lake Success, N.Y. 11042.

New Opportunities From The Physician Resource Group
Roblin Associates has openings in all specialties nationwide. We'll place you in a location of your choice . . . at no cost to you. Complete financial packages available. For more information, send CV in confidence.

Roblin Associates, Inc.
602 Market Street Mall
P.O. Box 397N
Wilmington, DE 19899 • 302/429-8600
RA

CHIEF OF MEDICAL SERVICES
Rochester Psychiatric Center, a progressive New York State JCAH accredited facility, is recruiting for a full time Chief of Medical Services, who will have responsibility for the administration and supervision of the medical treatment program delivered to approximately 850 inpatients and also specialty and subspecialty clinics that we work with, both on the grounds and at community hospitals. Rochester Psychiatric Center has a close working relationship with the University of Rochester Medical Center, and other community hospitals.

Qualifications: NYS license to practice medicine; certification by the appropriate American Medical Specialty Board or a foreign board clearly equivalent; and one year of post-certification experience in your specialty.

Excellent salary and fringe benefits. Send CV to Frank Soults, M.D., Medical Director, Rochester Psychiatric Center, 1600 South Avenue, Rochester, New York 14620. EOE.

This Publication is available in Microform.

University Microfilms International

Please send additional information
for ______________________
(name of publication)
Name ______________________
Institution ______________________
Street ______________________
City ______________________
State __________ Zip __________
300 North Zeeb Road, Dept P.R., Ann Arbor, Mi. 48106

Index to Advertisers

Ayerst Laboratories	2A,3A,4A
Classified Advertising	6A,8A,12A,14A, 16A
Eli Lilly and Co.	2nd Cover
Marion Laboratories	17A,18A
Medical Quadrangle	12A
Roche Laboratories	3rd & 4th Covers
Stuart Pharmaceuticals	7A,8A
Upjohn Company	15A
U.S. Army	11A
Winthrop Breon Laboratories	5A,6A
Wyeth Laboratories	13A, 14A

BALANCED CALCIUM CHANNEL BLOCKADE!

Low incidence of side effects
CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

*Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

References:
1. Strauss WE, McIntyre KM, Parisi AF, et al: Safety and efficacy of diltiazem hydrochloride for the treatment of stable angina pectoris: Report of a cooperative clinical trial. Am J Cardiol 49:560-566, 1982.
2. Pool PE, Seagren SC, Bonanno JA, et al: The treatment of exercise-inducible chronic stable angina with diltiazem: Effect on treadmill exercise. Chest 78 (July suppl):234-238, 1980.

Reduces angina attack frequency*
42% to 46% decrease reported in multicenter study.[1]

Increases exercise tolerance*
In Bruce exercise test,[2] control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes ($P<.005$).

CARDIZEM®
(diltiazem HCl)

THE BALANCED CALCIUM CHANNEL BLOCKER

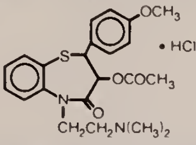
Please see full prescribing information on following page. 2/84

PROFESSIONAL USE INFORMATION

cardizem®
(diltiazem HCl)
30 mg and 60 mg tablets

DESCRIPTION

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one,3-(acetyloxy)-5-[2-(dimethylamino)ethyl]-2,3-dihydro-2-(4-methoxyphenyl)-, monohydrochloride,(+)-cis-. The chemical structure is:



Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform. It has a molecular weight of 450.98. Each tablet of CARDIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration.

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth muscle.

Mechanisms of Action. Although precise mechanisms of its antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways:

1. Angina Due to Coronary Artery Spasm: CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial and subendocardial. Spontaneous and ergonovine-induced coronary artery spasm are inhibited by CARDIZEM.
2. Exertional Angina: CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal and maximal exercise work loads.

In animal models, diltiazem interferes with the slow inward (depolarizing) current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Diltiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance.

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricular conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load. Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect; cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of diltiazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by diltiazem.

Intravenous diltiazem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Diltiazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, diltiazem significantly prolongs sinus cycle length (up to 50% in some cases).

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Diltiazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40%. CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 60 mg are given; a 120-mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem.

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm.** CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina).** CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS).

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing
Cardiac Failure	Administer inotropic agents (isoproterenol, dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

The oral/LD_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort acute anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy**—CARDIZEM may be safely coadministered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

Cardizem 30-mg tablets are supplied in bottles of 100 (NDC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1771-49). Each green tablet is engraved with MARION on one side and 1771 engraved on the other. CARDIZEM 60-mg scored tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1772-49). Each yellow tablet is engraved with MARION on one side and 1772 on the other.

Issued 4/1/84

Another patient benefit product from
PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MISSOURI 64137

EXCERPTS FROM A SYMPOSIUM
"THE TREATMENT OF SLEEP DISORDERS"[8]

"...highly effective for both sleep induction and sleep maintenance"

Sleep Laboratory Investigator
Pennsylvania

"...onset of action is rapid...provides sleep with no rebound effect to agitate the patient the following day"

Psychiatrist
California

"...appears to have the best safety record of any of the benzodiazepines"

Psychiatrist
California

After 15 years, the experts still concur about the continuing value of Dalmane (flurazepam HCl/ Roche). It provides sleep that satisfies patients... and the wide margin of safety that satisfies you.

The recommended dose in elderly or debilitated patients is 15 mg. Contraindicated in pregnancy.

DALMANE®
flurazepam HCl/Roche Ⓒ
sleep that satisfies

15-mg/30-mg
capsules

References: **1.** Kales J, *et al: Clin Pharmacol Ther 12*:691-697, Jul-Aug 1971. **2.** Kales A, *et al: Clin Pharmacol Ther 18*:356-363, Sep 1975. **3.** Kales A, *et al: Clin Pharmacol Ther 19*:576-583, May 1976. **4.** Kales A, *et al: Clin Pharmacol Ther 32*:781-788, Dec 1982. **5.** Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc 27*:541-546, Dec 1979. **6.** Dement WC, *et al: Behav Med*, pp. 25-31, Oct 1978. **7.** Kales A, Kales JD: *J Clin Psychopharmacol 3*:140-150, Apr 1983. **8.** Tennant FS, *et al:* Symposium on the Treatment of Sleep Disorders, Teleconference, Oct 16, 1984 **9.** Greenblatt DJ, Allen MD, Shader RI: *Clin Pharmacol Ther 21*:355-361, Mar 1977.

DALMANE®
flurazepam HCl/Roche Ⓒ

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening, in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended. Repeated therapy should only be undertaken with appropriate patient evaluation.

Contraindications: Known hypersensitivity to flurazepam HCl; pregnancy. Benzodiazepines may cause fetal damage when administered during pregnancy. Several studies suggest an increased risk of congenital malformations associated with benzodiazepine use during the first trimester. Warn patients of the potential risks to the fetus should the possibility of becoming pregnant exist while receiving flurazepam. Instruct patient to discontinue drug prior to becoming pregnant. Consider the possibility of pregnancy prior to instituting therapy.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase, and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg recommended initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

ROCHE®
Roche Products Inc.
Manati, Puerto Rico 00701

#1 FOR SLEEP

After more than 15 years of use, it's #1 for sleep that satisfies. Patients are satisfied because they fall asleep fast and stay asleep till morning.[1-8] And *you're* satisfied by the exceptionally wide margin of safety.[7-9] As always, caution patients about driving or drinking alcohol.

Please see references and summary of product information on reverse side.

DALMANE®

flurazepam HCl/Roche Ⓘⓥ

sleep that satisfies

ROCHE®

Copyright © 1985 by Roche Products Inc. All rights reserved.

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

AUGUST 1985 • Volume 85, Number 8

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
SEP 4 - 1985
M. D. S.

Contents

COMMENTARIES

Has breast self-examination had a fair trial? 479
GAIL DIEM, EdD, DAVID P. ROSE, MD, PhD

The current status of research in breast self-examination 480
ROGER S. FOSTER, Jr, MD, MICHAEL C. COSTANZA, PhD, JOHN K. WORDEN, PhD

Breast self-examination—a practice whose time has come? 482
JOSEPH FELDMAN, DrPH

THE CPC

A 77-year-old woman with giant cell arteritis, back pain, and shock 502

HISTORY

Medical education in New York City in 1866–1867: a student's notebook of Professor Charles A. Budd's lectures on obstetrics at New York University—Part I 488
BERT HANSEN, PhD

ESSAYS IN PEDIATRICS

Management of idiopathic thrombocytopenic purpura in childhood 499
JAMES B. BUSSEL, MD

CASE REPORTS

Suppurative pneumococcal inguinal adenitis 509
ARTHUR S. LEBOWITZ, MD, PHILIP M. TIERNO, Jr, PhD

Pseudoinfarction pattern associated with myocardial bridging of the left anterior descending artery 510
EDWARD T. DAVISON, MD, ROBERT I. HAMBY, MD

Uterine tumor due to placenta increta 512
SHAWKY Z. A. BADAWY, MD, JACK YOFFA, MD, BRENT FLETCHER, MD, THOMAS SIMON, MD

Cardiac siderosis in thalassemia major 513
EDWARD C. ZAINO, MD

Asymptomatic perforation of the small intestine by a Copper-7 intrauterine device 515
BURTON L. ROCHELSON, MD, WILLIAM J. MANN, MD, FRANK FROMOWITZ, MD, ROGER BAIM, MD

LETTERS TO THE EDITOR

Wounds of war 484
ROBERT E. ROTHENBERG, MD

Searching for alternatives to war 484
F. CRAIG FISHER, MD

Roswell Park 484
PAUL deR KOLISCH, MD
JOHN S. HALLER, Jr

Spontaneous dissection of the right coronary artery 485
JOSEPH E. DeJUNCO, MD, ROBERT I. HAMBY, MD, MANUEL VEGA, MD

Scalds of the lap 485
BENNETT M. DERBY, MD

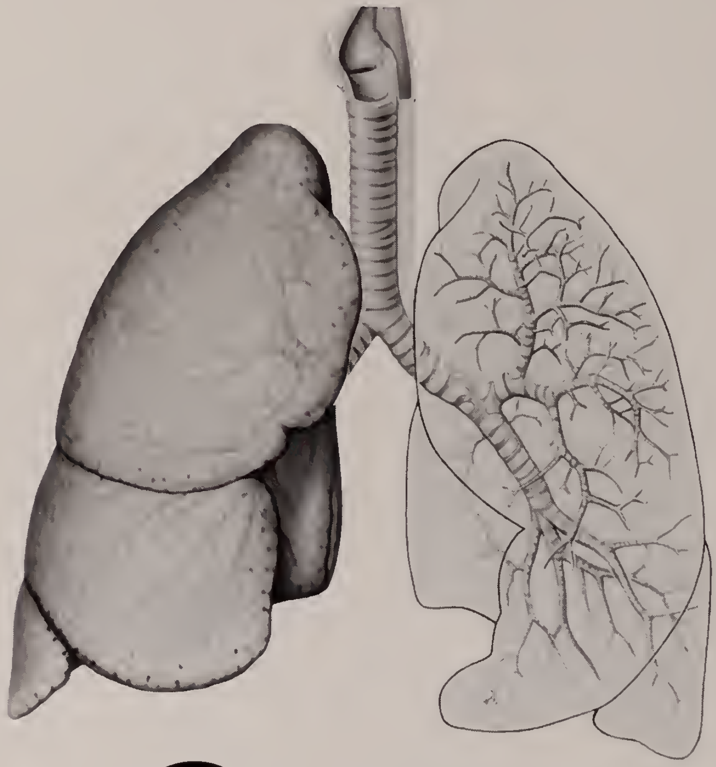
Medical approval for a driver's license 486
DAVID GREEN, MB, BS

Repair of inguinal hernia with Marlex mesh 486
VINAY K. KAPOOR, MS, MAHESH C. MISHRA, MS, TUSHAR K. CHATTOPADHYAY, MS, LALIT K. SHARMA, MS

BOOK REVIEWS 517
OBITUARIES 520
GUIDELINES TO AUTHORS 523
MEDICAL MEETINGS AND LECTURES 4A

Library of the College Physi
of Philadelphia   Dec. 85
19 S. 22nd St.
Phila, PA 19103

Consider the causative organisms...



Ceclor®
cefaclor
250-mg Pulvules® t.i.d.

offers effectiveness against the major causes of bacterial bronchitis

H. influenzae, *H. influenzae*, *S. pneumoniae*, *S. pyogenes*
(ampicillin-susceptible) (ampicillin-resistant)

Brief Summary Consult the package literature for prescribing information

Indications and Usage Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae)*, *Haemophilus influenzae* and *S pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor

Contraindication Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

Warnings IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS TO BOTH DRUG CLASSES

Antibiotics, including Ceclor should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins) therefore it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics Such colitis may range in severity from mild to life-threatening

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C difficile* Other causes of colitis should be ruled out

Precautions *General Precautions* — If an allergic reaction to Ceclor® (cefaclor, Lilly) occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e g , pressor amines, antihistamines, or corticosteroids

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms Careful observation of the patient is essential If superinfection occurs during therapy, appropriate measures should be taken

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

Ceclor should be administered with caution in the presence of markedly impaired renal function Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly)

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis

Usage in Pregnancy — Pregnancy Category B — Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor® (cefaclor, Lilly) There are, however, no adequate and well-controlled studies in pregnant women Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers — Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses Average levels were 0 18, 0 20, 0 21, and 0 16 mcg/ml at two, three, four, and five hours respectively Trace amounts were detected at one hour. The effect on nursing infants is not known Caution should be exercised when Ceclor is administered to a nursing woman.

Usage in Children — Safety and effectiveness of this product for use in infants less than one month of age have not been established

Adverse Reactions. Adverse effects considered related to therapy with Ceclor are uncommon and are listed below

Gastrointestinal symptoms occur in about 2 5 percent of patients and include diarrhea (1 in 70)

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment Nausea and vomiting have been reported rarely

Hypersensitivity reactions have been reported in about 1 5 percent of patients and include morbilliform eruptions (1 in 100) Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor Such reactions have been reported more frequently in children than in adults Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy No serious sequelae have been reported Antihistamines and corticosteroids appear to enhance resolution of the syndrome

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients)

Causal Relationship Uncertain — Transitory abnormalities in clinical laboratory test results have been reported Although they were of uncertain etiology they are listed below to serve as alerting information for the physician

Hepatic — Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40)

Hematopoietic — Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40)

Renal — Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200)

[0617B2R]

Note Ceclor® (cefaclor, Lilly) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever See prescribing information

© 1984 ELI LILLY AND COMPANY

Lilly

Additional information available to the profession on request from
Eli Lilly and Company
Indianapolis, Indiana 46285

Eli Lilly Industries, Inc
Carolina Puerto Rico 00630

400441

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
FRANK C. MAXON, JR, MD
ELIZA H. CALDWELL, MD
RICHARD B. NOLAN, MD
URSULA A. MATULONIS (Medical Student)
JOHN T. PRIOR, MD

Editor	ALAN BLUM, MD
Deputy Editor and Book Review Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Managing Editor	VICKI GLASER
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Administrative Assistant	IMELDA MORRISSEY
Editorial Assistant	JEANNE CONNERY
Librarian	ELLA ABNEY
Library Assistant	ELEANOR BURNS
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1985

RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MARK C. HOMONOFF, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. Synergistic Media Sales, 50 East 42nd St., Suite 504, New York, N.Y. 10017. Telephone: (212) 682-2200.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

DANIEL F. O'KEEFFE, MD, *President*
ALLISON B. LANDOLT, MD, *Past-President*
JOHN A. FINKBEINER, MD, *President-Elect*
SAMUEL M. GELFAND, MD, *Vice-President*
JOHN H. CARTER, MD, *Secretary*
GEORGE LIM, MD, *Assistant Secretary*
CHARLES D. SHERMAN, JR, , MD, *Treasurer*
MORTON KURTZ, MD, *Assistant Treasurer*
CHARLES N. ASWAD, MD, *Speaker*
SEYMOUR R. STALL, MD, *Vice-Speaker*

Councilors

Term Expires 1986
ROBERT A. MAYERS, MD, Westchester
THOMAS D. PEMRICK, MD, Rensselaer
RALPH E. SCHLOSSMAN, MD, Queens
STANLEY A. STECKLER, MD, Suffolk
ROBERT J. NEWBORN, Albany
(Councilor representing the medical student membership)

Term Expires 1987
DAVID M. BENFORD, MD, New York
DUANE M. CADY, MD, Onondaga
JAMES M. FLANAGAN, MD, Wayne
M. THEODORE TANENHAUS, MD, Brooklyn

Term Expires 1988
JAMES H. COSGRIFF, JR, MD, Erie
RICHARD A. HUGHES, MD, Warren
ANTONIO F. LASORTE, MD, Broome
SIDNEY MISHKIN, MD, Nassau

Trustees

G. REHMI DENTON, MD, Albany, *Chairman*
RALPH M. SCHWARTZ, MD, New York
EDGAR P. BERRY, MD, New York
RICHARD D. EBERLE, MD, Onondaga
JOSEPH F. SHANAPHY, MD, Richmond
VICTOR J. TOFANY, MD, Monroe
BERNARD J. PISANI, MD, New York

Executive Vice-President	GEORGE J. LAWRENCE, JR, MD
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Associate	IRMA A. ERICKSON
Director, Division of Scientific Publications	ALAN BLUM, MD
Director, Division of Management Services	GEORGE FORREST, JR
Assistant Director, Division of Governmental Affairs	JERRY S. HOFFMAN, ESQ
Director, Division of Communications	EDWARD HYNES
Director, Division of Insurance and Membership Benefits	BERNARD M. JACKSON
Director, Division of Membership	EUNICE SKELLY
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Scientific Activities	PETER B. FARNSWORTH, MD
Director, Division of Advertising-Sales and Meeting Services	ROBERT MILLER
General Counsel	DONALD R. MOY, JD
Director, Division of Computer Services	IVAN H. NAJMAN
Acting Director, Division of Medical Services	MARIO MENGHINI
Director, Division of Long-Range Planning	DEBORAH R. SIEGEL
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. Editorial material published in the *Journal* is covered by copyright. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

On nitrates,
but angina still
strikes...

After a nitrate, add ISOPTIN®

(verapamil HCl/Knoll)

To protect your patients, as well as their quality of life, add Isoptin instead of a beta blocker.

First, Isoptin not only reduces myocardial oxygen demand by reducing peripheral resistance, but also increases coronary perfusion by preventing coronary vasospasm and dilating coronary arteries — both normal and stenotic. These are antianginal actions that no beta blocker can provide.

Second, Isoptin spares patients the beta-blocker side effects that may compromise the quality of life.

With Isoptin, fatigue, bradycardia and mental depression are rare. Unlike beta blockers, Isoptin can safely be given to patients with asthma, COPD, diabetes or peripheral vascular disease. Serious adverse reactions with Isoptin are rare at recommended doses; the single most common side effect is constipation (6.3%).

Cardiovascular contraindications to the use of Isoptin are similar to those of beta blockers: severe left ventricular dysfunction, hypotension (systolic pressure <90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no artificial pacemaker is present) and second- or third-degree AV block.

So, the next time a nitrate is not enough, add Isoptin . . . for more comprehensive antianginal protection without side effects which may cramp an active life style.

ISOPTIN. Added antianginal protection without beta-blocker side effects.

Please see brief summary on following page.

ISOPTIN®
(verapamil HCl/Knoll)
80 mg and 120 mg scored, film-coated tablets

Contraindications: Severe left ventricular dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (except in patients with a functioning artificial ventricular pacemaker), 2nd- or 3rd-degree AV block **Warnings:** ISOPTIN should be avoided in patients with severe left ventricular dysfunction (e.g., ejection fraction < 30% or moderate to severe symptoms of cardiac failure) and in patients with any degree of ventricular dysfunction if they are receiving a beta blocker. (See *Precautions.*) Patients with milder ventricular dysfunction should, if possible, be controlled with optimum doses of digitalis and/or diuretics before ISOPTIN is used. (Note interactions with digoxin under *Precautions.*) ISOPTIN may occasionally produce hypotension (usually asymptomatic, orthostatic, mild and controlled by decrease in ISOPTIN dose). Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported. Such elevations may disappear even with continued treatment; however, four cases of hepatocellular injury by verapamil have been proven by rechallenge. Periodic monitoring of liver function is prudent during verapamil therapy. Patients with atrial flutter or fibrillation and an accessory AV pathway (e.g. W-P-W or L-G-L syndromes) may develop increased antegrade conduction across the aberrant pathway bypassing the AV node, producing a very rapid ventricular response after receiving ISOPTIN (or digitalis). Treatment is usually D.C.-cardioversion, which has been used safely and effectively after ISOPTIN. Because of verapamil's effect on AV conduction and the SA node, 1° AV block and transient bradycardia may occur. High grade block, however, has been infrequently observed. Marked 1° or progressive 2° or 3° AV block requires a dosage reduction or, rarely, discontinuation and institution of appropriate therapy depending upon the clinical situation. Patients with hypertrophic cardiomyopathy (IHSS) received verapamil in doses up to 720 mg/day. It must be appreciated that this group of patients had a serious disease with a high mortality rate and that most were refractory or intolerant to propranolol. A variety of serious adverse effects were seen in this group of patients including sinus bradycardia, 2° AV block, sinus arrest, pulmonary edema and/or severe hypotension. Most adverse effects responded well to dose reduction and only rarely was verapamil discontinued. **Precautions:** ISOPTIN should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacologic effects. Studies in a small number of patients suggest that concomitant use of ISOPTIN and beta blockers may be beneficial in patients with chronic stable angina. Combined therapy can also have adverse effects on cardiac function. Therefore, until further studies are completed, ISOPTIN should be used alone, if possible. If combined therapy is used, close surveillance of vital signs and clinical status should be carried out. Combined therapy with ISOPTIN and propranolol should usually be avoided in patients with AV conduction abnormalities and/or depressed left ventricular function. Chronic ISOPTIN treatment increases serum digoxin levels by 50% to 70% during the first week of therapy, which can result in digitalis toxicity. The digoxin dose should be reduced when ISOPTIN is given, and the patients should be carefully monitored to avoid over- or under-digitalization. ISOPTIN may have an additive effect on lowering blood pressure in patients receiving oral antihypertensive agents. Disopyramide should not be given within 48 hours before or 24 hours after ISOPTIN administration. Until further data are obtained, combined ISOPTIN and quinidine therapy in patients with hypertrophic cardiomyopathy should probably be avoided, since significant hypotension may result. Clinical experience with the concomitant use of ISOPTIN and short- and long-acting nitrates suggest beneficial interaction without undesirable drug interactions. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. *Pregnancy Category C:* There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor and delivery only if clearly needed. It is not known whether verapamil is excreted in breast milk; therefore, nursing should be discontinued during ISOPTIN use. **Adverse Reactions:** Hypotension (2.9%), peripheral edema (1.7%), AV block: 3rd degree (0.8%), bradycardia: HR < 50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%), elevations of liver enzymes have been reported. (See *Warnings.*) The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain: ecchymosis, bruising, gynecomastia, psychotic symptoms, confusion, paresthesia, insomnia, somnolence, equilibrium disorder, blurred vision, syncope, muscle cramp, shakiness, claudication, hair loss, macules, spotty menstruation. **How Supplied:** ISOPTIN (verapamil HCl) is supplied in round, scored, film-coated tablets containing either 80 mg or 120 mg of verapamil hydrochloride and embossed with "ISOPTIN 80" or "ISOPTIN 120" on one side and with "KNOLL" on the reverse side. Revised August, 1984. 2385

knoll® **KNOLL PHARMACEUTICAL COMPANY**
30 NORTH JEFFERSON ROAD, WHIPPANY, NEW JERSEY 07981

2406

Medical Society of the State of New York
ANNUAL CME ASSEMBLY
MARCH 7-9, 1986
The New York Hilton, New York City
Combined With The Annual Meeting of
The House of Delegates
MARCH 6-9

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meetings and lectures unless the information is submitted at least two months prior to the event.*

SEPTEMBER 1985

AROUND THE STATE

MANHATTAN

Sept 5-7. **Surgical Courses in Lens Implantation.** Info: Ruth Green, Administrator, Ophthalmological Foundation of America, 823 United Nations Plaza, New York, NY 10017. Tel: (212) 986-4848.

Sept 14. **Cataract and Implant Update.** New York. Info: Martha Klapp, Manhattan Eye, Ear and Throat Hospital, 210 E. 64th St, New York, NY 10021. Tel: (212) 838-9200.

Sept 18-22. **Seventeenth Annual Oculoplastic & Facial Surgery Dissection Course.** New York. Info: Congress Administrator, c/o Drs. Guibor, Hecht & Wiggs, 630 Park Ave, New York, NY 10021. Tel: (212) 734-1010 or (800) 223-4500.

Sept 20-22. **Computers/Medicine '85.** 21½ Cat 1 credits. New York Marriott Marquis Hotel. Info: Conference Management Corp, 17 Washington St, PO Box 4990, Norwalk, CT 06856.

continued on p 6A

Classified Advertising

PHYSICIANS WANTED

EXCELLENT OPPORTUNITY FOR READY MADE PRACTICE for General Practice Specialist or Internist willing to do a little general practice, available immediately, in an attractive Medical Building with in-house lab and x-ray facilities. Permanent relationship very desirable. Beautiful countryside-Cortland, NY. Phone collect (607) 756-9941 or submit c.v. with references to: Donald R. Gibbs, M.D., 110 North Main Street, Cortland, NY 13045.

NEW YORK, NEW PALTZ—Seeking full-time experience or residency trained physicians; ACLS certified; for state of the art FEC in beautifull Mid-Hudson Valley, 80 miles from NYC. Start date is July 15, 1985. Package includes Health, Life, Disability and Malpractice Insurance. Excellent salary, attractive scheduling and potential profit sharing. Send CV/Inquiries to Gary W. Greer, M.D., RD 3 Box 44A, Cogan Station, PA., 17728: phone 1-717-494-0420.

ARIZONA BASED PHYSICIAN RECRUITMENT firm has opportunites Coast to Coast. "Professionals working with Professionals". Over 13 years experience. Call (602) 795-7474; or send CV to: Mitchell & Associates, Inc., 2761 N. Country Club Rd., Suite 202, Tucson, AZ 85716.

PHYSICIAN-EMERGENCY ROOM BC or BE physician in primary care specialty to work with large group interested in ambulatory care medicine. Background in emergency medicine or family practice desirable. ACLS, ATLS required. Excellent salary and schedule. Upstate NY area. Send CV to Prospect Hill Emergency Care Physicians, P.C., c/o 961 Canal St., Syracuse, NY 13210.

FULL TIME AND PART TIME PRIMARY CARE PHYSICIAN NEEDED at Monroe Developmental Center, a 400 bed facility for the habilitation of multiple handicapped people in Rochester, New York. Excellent salary according to qualifications. Take advantage of regular negotiable working hours. No nights or weekend call required. Call or write Elmar H. Frangenberg, M.D., Chief of Medical Staff, Monroe Development Center, 620 Westfall Road, Rochester, New York 14620, (716) 461-2800 ext. 334. As an Affirmative Action Employer, we encourage minorities, women and handicapped to apply.

SURGEON—examine, diagnose & treat pts with heart, lung & vascular disease. Requires N.Y. license + Bd elig or cert in CVT surgery. Full time/on call emerg. Send c.v. James E. Lowe, M.D., c/o Lexington Prof. Ctr., 133 East 73rd Street, New York, N.Y. 10021.

PRACTICES AVAILABLE

SELLING PRACTICES IS OUR ONLY BUSINESS—Want to maximize the return on your investment? Before you buy or sell, call for a consultation to assure the best financial and transfer terms. We guide you through the entire sales process from initial meeting to closing. Serving 100 mile radius of New York City. Countrywide Business Brokerage, Inc., 319 East 24th Street, Suite 23-G, New York, NY 10010 (212) 686-7902.

FAMILY PRACTICE AVAILABLE WITH OFFICE BUILDING: Fully equipped. Located Ballston Spa, N.Y. near Albany, N.Y. Doctor retiring after 35 years. Information from Norman Fox, 404 Broadway, Saratoga Springs, N.Y. 12866. (518) 587-7778.

MEDICAL PRACTICE SALES and Appraisals. We specialize in the valuation and selling of medical practices. If interested in buying or selling a medical practice, contact our Brokerage Division at: The Health Care Group, 400 GSB Building, Bala Cynwyd, PA 19004. (215) 667-8630.

FOR SALE IMMEDIATELY—Lucrative Neurology Practice in Syracuse, New York, due to sudden death of practitioner. Includes office and hospital patients. Established over 17 year period. No surgery. Negotiable terms. Urgent need by patients to have dedicated physician. For further information call 315-457-9122 days and 315-451-4913 nights and weekends.

PRACTICES WANTED

OPHTHALMOLOGIST, BOARD CERTIFIED desires to purchase practice in New York City or suburban area. Confidence maintained. Please contact Dept. 168, c/o NYSJM.

REAL ESTATE FOR SALE OR RENT

FOR SALE ROSLYN-COUNTRY ESTATES large colonial. 5 bedrooms, 3½ baths, ½ acre, finished basement. Many Extras. (516) 621-1070.

SETAUKET, LONG ISLAND NORTH SHORE. Retiring doctor's office plus home near Stoneybrook University, 5 hospitals, new home developments. 3/4 acre corner, beautiful landscaping. Office wing of ranch choice of 3 or 4 rooms, panelled library, 1/2 bath. Living area has unique luxury features, sensational bathroom, large jalousie room professionally equipped for indoor grilling. Finished basement, gorgeous bedroom, bath, gym, huge cedar room. $225,000, original owner (516) 751-0001.

PROFESSIONAL OFFICE SPACE. New private building, A/C, soundproofing, 24 hour, 7 day access, 650 square foot floor or up to 1100 feet. Below market rent. In Port Washington, 5 minutes from Long Island Expressway, walk to L.I.R.R. station. Call (516) 944-6320.

REAL ESTATE FOR SALE OR RENT—CONT'D

BAY SHORE, Leasing new luxurious medical building. Located near Southside Hospital. Minimum suite 740 sq. ft. Large parking area, handicapped designed. Individual gas heat, a/c-electric. (516) 665-4064, 10 A.M. to 5 P.M., Monday to Friday.

WANTED: MEDICAL OFFICE BUILDINGS. As principals we are seeking "existing buildings"—fully tennanted by physicians, dentists and health care services. Contact: Paul Gellert, Gelco Associates, 155 West 68th St., N.Y., N.Y. 10023; (212) 724-7900.

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

MEDICAL OFFICE AVAILABLE in village of Syosset on the north shore of Long Island. 1800 sq. ft., prime location on first floor, for rent with option to buy in co-op ownership. Call (516) 921-6020.

HUNTINGTON VILLAGE, HUNTINGTON, LONG ISLAND available for immediate purchase home-office combination of recently deceased physician (psychiatrist). Please call (516) 427-9242 for further information.

YOU CAN CONQUER THE WORLD AND STILL ESCAPE FROM IT! Magnificent Georgian Colonial within walking distance to private beach; a splendid personal residence offering 4 bedrooms, 2 baths, living room with wood burning fireplace plus 4 room professional suite with bath. The best of both worlds is offered to you exclusively by Merrill Lynch Realty, Carll Burr, Inc. Previews by appointment only. $350,000. Agent: Rhonda Barmack (516) 872-8890.

LOOKING FOR A DOCTOR TO SHARE great location in the Village of Huntington. Building has its own parking, ground floor, more space available. Call (516) 673-2900.

MODERN-WELL FURNISHED MEDICAL OFFICE, East 67th Street, near Memorial and N.Y.H. Large reception area, 2 examination rooms, laboratory available. Hours to suit on Tuesday, Thursday and Saturday. Share with Internist/Oncologist on a fair rent and expense basis. Call Monday–Friday (10 AM to 2 PM) (212) 988-9773.

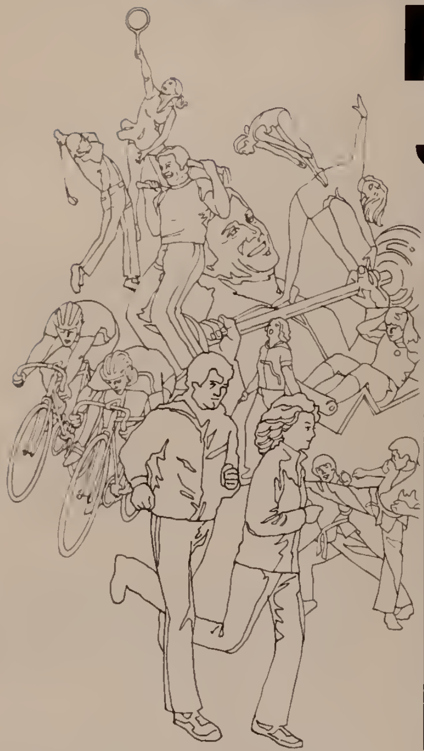
60's WEST *(LINCOLN CENTER)*

INVESTOR'S VACANT APTS

5% Down

Positive cash flow if you qualify. Convertible large 3 BR's, terrace. Views, from $385,000. Owner, 333-5199.

OVER 66,000
FAMILY PHYSICIANS
READ THIS
JOURNAL



the physician and sportsmedicine
A McGRAW-HILL PUBLICATION
FEBRUARY 1984
Profile of Youth Soccer Injuries
How I Manage Gout in Athletes
Heart Rate and PVCs During Exercise
Current Status of Meniscus Surgery
Exercise: An Alternative to Fad Diets for Adolescent Girls

Practical information on the medical aspects of fitness and exercise.

Tennis elbow: Joint resolution by conservative treatment.
Hypertrophic cardiomyopathy and the athlete.
Effects of sunscreen use during exercise in the heat.
Overuse injuries to the knee in runners.
How I manage ingrown toenails.

McGraw-Hill

---

continued from p 4A

NASSAU

Sept 11-13. **Fifth Annual Advanced Course in Lower Limb Amputation and Prosthetics.** East Meadow. Info: Lawrence W. Fridmann, MD, Physician Medicine and Rehabilitation, Nassau County Medical Center, East Meadow, NY. Tel: (516) 542-2525.

Sept 21. **The Poisoned Patient; Emergency Toxicology.** 7 Cat 1 credits. Info: Howard Mofenson, MD, Director—Poison Control Center, Nassau County Medical Center, 2201 Hempstead Turnpike, East Meadow, NY 11554. Tel: (516) 542-2323.

Sept 27. **Occupational Health: Issues in Environmental Health.** Garden City Hotel. Info: Ann J. Boehme, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

ALBANY

Sept 27-29. **Legal and Medical Aspects of Practicing Obstetrics and Newborn Pediatric Medicine.** Rensselaerville. Info: Herman M. Risemberg, MD, Professor of Pediatrics, The Albany Medical College of Union University, Albany, NY 12208. Tel: (518) 445-5122.

Sept 30 - Oct 1. **Genetic Disease Screening and Management.** Albany Hilton Hotel. 14 Cat 1 credits. Info: Thomas P. Carter, PhD, State of New York Department of Health, Wadsworth Center for Labs and Research, Rockefeller Empire State Plaza, Albany, NY 12201.

DUTCHESS

Sept 18. **Pulmonary Disease Update.** Poughkeepsie. Info: Catherine M. Tarella, Medical-Dental Staff, Saint Francis Hospital, Poughkeepsie, NY 12601. Tel: (914)471-2000.

SCHENECTADY

Sept 12-14. **Cerebrovascular Disease and Stroke Rehabilitation.** Schenectady. Info: Barbara G. Post, Sunnyview Hospital and Rehabilitation Center, 1270 Belmont Ave, Schenectady, NY 12308. Tel: (518) 382-4595.

ST. LAWRENCE

Sept 19. **Ileo-Anal Reservoir for Familial Polyposis and Ulcerative Colitis in Children.** Info: Henry Vinicor, MD, 53 South Main St, Norwood, NY 13668. Tel: (315) 353-2332.

continued on p 8A

EXCLUSIVE

NEWS FOR NEW YORK

TALWIN® Nx

C IV Each tablet contains pentazocine HCl, USP, equivalent to 50 mg base and naloxone HCl, USP, equivalent to 0.5 mg base.

NOW APPROVED FOR MEDICAID

T-540 NDC 0024-1951-04
100 tablets
C IV
Talwin Nx
Each tablet contains pentazocine hydrochloride, USP, equivalent to 50 mg base and naloxone hydrochloride, USP, 0.5 mg.
Caution: Federal law prohibits dispensing without prescription.
Winthrop

Talwin® Nx

C IV Each tablet contains pentazocine HCl, USP, equivalent to 50 mg base and naloxone HCl, USP, equivalent to 0.5 mg base.

Major Analgesic Gains Reimbursement Status

Albany—TALWIN Nx has recently been included on the New York State Medicaid list of reimbursable drugs.

Physicians can now make available to their Medicaid patients the many advantages of TALWIN Nx for moderate to severe pain conditions such as fractures, acute lumbosacral pain, severe sprains, joint pain, and acute trauma.

The newly listed TALWIN Nx is characterized by dependable efficacy and a proven record of safety.[1] One tablet provides pain relief equivalent to one grain of codeine, yet allows patients to function.* TALWIN Nx offers a 15 to 30 minute onset of action, and is logical therapy for persistent pain because tolerance buildup is rare. The product is especially appropriate for many elderly patients since it has a low risk of constipation.†

*Since sedation, dizziness, and occasional euphoria have been noted, ambulatory patients should be warned not to operate machinery, drive cars, or unnecessarily expose themselves to hazards.

†See important product information concerning contraindications, warnings, adverse reactions, patient selection, and prescribing and precautionary recommendations on following page.

1. Data from the Boston Collaborative Drug Surveillance Program, July 1984.

TALWIN Nx now approved for the Medicaid Drug List by New York State.

Winthrop-Breon

© 1985 Winthrop-Breon Laboratories

Talwin® Nx CIV

Each tablet contains pentazocine HCl, USP, equivalent to 50 mg base and naloxone HCl, USP, equivalent to 0.5 mg base.

Analgesic for Oral Use Only

Contraindications: Hypersensitivity to either pentazocine or naloxone.

> TALWIN® Nx is intended for oral use only. Severe, potentially lethal, reactions may result from misuse of TALWIN® Nx by injection either alone or in combination with other substances. (See **Drug Abuse and Dependence** section.)

Warnings: *Drug Dependence.* Can cause physical and psychological dependence. (See **Drug Abuse and Dependence**.) *Head Injury and Increased Intracranial Pressure.* As with other potent analgesics, respiratory depressant effects of the drug may elevate cerebrospinal fluid pressure due to CO_2 retention; these effects may be markedly exaggerated in the presence of head injury, other intracranial lesions, or a preexisting increase in intracranial pressure. Can obscure the clinical course of patients with head injuries; in such patients, use with extreme caution and only if deemed essential. *Usage with Alcohol.* Due to potential for increased CNS depressant effects, alcohol should be used with caution. *Patients Receiving Narcotics.* Pentazocine is a mild narcotic antagonist. Withdrawal symptoms have occurred in patients previously given narcotics, including methadone. *Certain Respiratory Conditions.* Should be administered with caution in respiratory depression from any cause, severely limited respiratory reserve, severe bronchial asthma and other obstructive respiratory conditions, or cyanosis. *Acute CNS Manifestations.* Therapeutic doses have resulted in hallucinations (usually visual), disorientation, and confusion which have cleared spontaneously within hours. Such patients should be closely observed and vital signs checked. Reinstitution of the drug should be done cautiously since acute CNS manifestations may recur.

Precautions: *CNS Effect.* Use cautiously in patients prone to seizures; seizures have occurred though no cause and effect relationship has been established. *Impaired Renal or Hepatic Function.* Decreased metabolism of pentazocine in extensive liver disease may predispose to accentuation of side effects; it should be administered with caution in renal or hepatic impairment. In long-term use, precautions should be taken to avoid increases in dose by the patient. *Biliary Surgery.* Some evidence suggests that unlike other narcotics pentazocine causes little or no elevation in biliary tract pressures; the clinical significance of these findings is not yet known. *Information for Patients.* Since sedation, dizziness, and occasional euphoria have been noted, ambulatory patients should be warned not to operate machinery, drive cars, or unnecessarily expose themselves to hazards. May cause physical and psychological dependence taken alone and may have additive CNS depressant properties in combination with alcohol or other CNS depressants. *Myocardial Infarction.* Use with caution in patients with myocardial infarction who have nausea or vomiting. *Drug Interactions. Usage with Alcohol:* See **Warnings.** *Carcinogenesis, Mutagenesis, Impairment of Fertility.* No long-term studies in animals to test for carcinogenesis have been performed. *Pregnancy Category C.* Should be given to pregnant women only if clearly needed. *Labor and Delivery.* Use with caution in women delivering premature infants. Effect on mother and fetus, duration of labor or delivery, need for forceps delivery or other intervention or resuscitation of newborn, or later growth, development, and functional maturation of the child is unknown. *Nursing Mothers.* Caution should be exercised when administered to a nursing woman. *Pediatric Use.* Safety and effectiveness in children below the age of 12 years have not been established.

Adverse Reactions: *Cardiovascular:* Hypotension, tachycardia, syncope. *Respiratory:* Rarely, respiratory depression. *Acute CNS Manifestations:* In rare instances, hallucinations (usually visual), disorientation, and confusion which have cleared spontaneously within a period of hours. Such patients should be closely observed and vital signs checked. Acute CNS manifestations may recur if the drug is reinstituted. *Other CNS Effects:* Dizziness, lightheadedness, hallucinations, sedation, euphoria, headache, confusion, disorientation; infrequently weakness, disturbed dreams, insomnia, syncope, visual blurring and focusing difficulty, depression; rarely tremor, irritability, excitement, tinnitus. *Autonomic:* Sweating; infrequently flushing; and rarely chills. *Gastrointestinal:* Nausea, vomiting, constipation, diarrhea, anorexia, rarely abdominal distress. *Allergic:* Edema of the face; dermatitis, including pruritus; flushed skin, including plethora; infrequently rash, rarely urticaria. *Ophthalmic:* Visual blurring and focusing difficulty. *Hematologic:* Depression of white blood cells (especially granulocytes), which is usually reversible, moderate transient eosinophilia. *Other:* Headache, chills, insomnia, weakness, urinary retention, paresthesia

Drug Abuse and Dependence: *Controlled Substance.* TALWIN Nx is a Schedule IV controlled substance.

Dependence and withdrawal symptoms have been reported with orally administered pentazocine. Patients with a history of drug dependence should be under close supervision. Possible abstinence syndromes in newborns after prolonged use of pentazocine during pregnancy have been reported. In prescribing for chronic use, the physician should take precautions to avoid increases in dose by the patient. Tolerance to the analgesic effect is rarely reported, there is no long-term experience with oral use of TALWIN Nx.

The amount of naloxone present (0.5 mg per tablet) has no action when taken orally and will not interfere with the pharmacologic action of pentazocine; however, this amount of naloxone given by injection has profound antagonistic action to narcotic analgesics. TALWIN Nx has a lower potential for parenteral misuse than the previous oral pentazocine formulation, but is still subject to patient misuse and abuse by the oral route.

Severe, even lethal, consequences may result from misuse of tablets by injection either alone or in combination with other substances, such as pulmonary emboli, vascular occlusion, ulceration and abscesses, and withdrawal symptoms in narcotic dependent individuals.

Overdosage: *Treatment:* Oxygen, intravenous fluids, vasopressors, and other supportive measures should be employed as indicated. Assisted or controlled ventilation should also be considered. For respiratory depression, parenteral naloxone is a specific and effective antagonist.

Please consult full product information before prescribing.

WIN 5-51296

Winthrop-Breon

Winthrop-Breon Laboratories
Division of Sterling Drug Inc.
New York, NY 10016

Buy Bonds

Take Stock in America.

continued from p 6A

AROUND THE NATION

CALIFORNIA

Sept 7-14. **Cardiology Tutorial in the Wilderness—Exploring the Colorado Plateau.** 24 Cat 1 Credits. Info: Office of CME, University of California San Diego School of Medicine, M-017, La Jolla, CA 92093. Tel: (619) 452-3940.

Sept 11-12. **Perspectives on Clinical Nutrition.** San Francisco. Info: American Society for Parenteral & Enteral Nutrition, 8605 Cameron St, Ste 500, Silver Spring, MD 20910. Tel: (301) 587-6315.

Sept 28-29. **The Skin From A to Z.** Napa Valley. Info: Extended Programs in Medical Education, Room 569-U, University of California, San Francisco, CA 94143. Tel: (415) 666-4251.

FLORIDA

Sept 4-7. **10th Annual Symposium on Cardiovascular Nursing.** Clearwater Beach. Info: American College of Cardiology, Extramural Programs Department, 9111 Old Georgetown Rd, Bethesda, MD 20814. Tel: (301) 897-5400, ext 230.

INDIANA

Sept 30-Oct 2. **Advanced Echocardiography.** Indianapolis. Info: American College of Cardiology, Extramural Programs Department, 9111 Old Georgetown Rd, Bethesda, MD 20814. Tel: (301) 897-5400, ext 230.

MASSACHUSETTS

Sept 28. **Cutaneous Laser Therapy.** 8

continued on p 14A

BALANCED CALCIUM CHANNEL BLOCKADE!

CARDIZEM
(diltiazem HCl)
balances potent coronary vasodilation with a low incidence of side effects

Low incidence of side effects
CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

Reduces angina attack frequency*
42% to 46% decrease reported in multicenter study.[1]

Increases exercise tolerance*
In Bruce exercise test,[2] control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes ($P<.005$).

CARDIZEM®
(diltiazem HCl)

**THE BALANCED
CALCIUM CHANNEL BLOCKER**

Please see full prescribing information on following page.

2/84

---

*Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

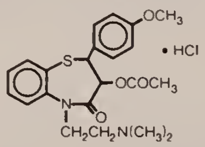
References:
1. Strauss WE, McIntyre KM, Parisi AF, et al: Safety and efficacy of diltiazem hydrochloride for the treatment of stable angina pectoris: Report of a cooperative clinical trial. Am J Cardiol 49:560-566, 1982.
2. Pool PE, Seagren SC, Bonanno JA, et al: The treatment of exercise-inducible chronic stable angina with diltiazem: Effect on treadmill exercise. Chest 78 (July suppl):234-238, 1980.

PROFESSIONAL USE INFORMATION

cardizem®
(diltiazem HCl)
30 mg and 60 mg tablets

DESCRIPTION

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one,3-(acetyloxy)-5-[2-(dimethylamino)ethyl]-2,3-dihydro-2-(4-methoxyphenyl)-, monohydrochloride,(+) -cis-. The chemical structure is:



Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform. It has a molecular weight of 450.98. Each tablet of CARDIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration.

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth muscle.

Mechanisms of Action. Although precise mechanisms of its antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways:

1. Angina Due to Coronary Artery Spasm: CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial and subendocardial. Spontaneous and ergonovine-induced coronary artery spasm are inhibited by CARDIZEM.
2. Exertional Angina: CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal and maximal exercise work loads.

In animal models, diltiazem interferes with the slow inward (depolarizing) current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Diltiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance.

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricular conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load. Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect; cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of diltiazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by diltiazem.

Intravenous diltiazem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Diltiazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, diltiazem significantly prolongs sinus cycle length (up to 50% in some cases).

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Diltiazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40%. CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 60 mg are given; a 120-mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem.

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm.** CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina).** CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS).

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia.
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing.
Cardiac Failure	Administer inotropic agents (isoproterenol, dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

The oral/LD_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort acute anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy**—CARDIZEM may be safely coadministered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

Cardizem 30-mg tablets are supplied in bottles of 100 (NDC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1771-49). Each green tablet is engraved with MARION on one side and 1771 engraved on the other. CARDIZEM 60-mg scored tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1772-49). Each yellow tablet is engraved with MARION on one side and 1772 on the other.

Issued 4/1/84

Another patient benefit product from

PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC
KANSAS CITY, MISSOURI 64137

NEW YORK STATE
JOURNAL OF MEDICINE

August 1985 • Volume 85, Number 8

COMMENTARIES

Has breast self-examination had a fair trial?

It has been estimated that in 1985 119,000 women will develop breast cancer and 38,400 will die from this disease.[1] While little can be done at present to prevent the onset of breast cancer, treatment of smaller, localized tumors generally yields better survival rates. Thus, early detection is critical for achieving a permanent cure. Of the three leading screening methods, breast self-examination (BSE), clinical palpation, and mammography, only BSE is routinely recommended for all women age 20 and older. With 25% of diagnosed breast cancers occurring in women under the age of 50 years,[2] and mammography contraindicated for most women in this age group, BSE is potentially an important method of early detection. Further, it is the only method that requires no physician appointment, cost, or special equipment, and it can be done in the privacy of a woman's home at her convenience. While this seemingly simple technique has been widely promoted of late, the assessment of its true effect on survival has been limited. Only one study seems promising; it showed an 18% increase in five-year survival among users of the technique.[3] Current research into efficacy is fraught with methodologic problems that need to be addressed to assess the procedure as a screening method.

The main clinical and pathological predictors of survival from breast cancer are tumor size and the presence or absence of axillary lymph node involvement. However, there are data that suggest that tumors of less than 4 cm in size are not in and of themselves predictive of treatment failure.[4] Without establishing such a standard, a comparison of tumor size based on practice groups or screening method may serve as an artificial variable. Also, other evidence indicates that even tumors too small to be detected by mammography may undergo systemic dissemination[5]; in such cases "early detection" becomes a misnomer. Considerations such as these suggest that effective BSE may prove to be selective for slow growing, histologically well differentiated tumors which inherently have a good prognosis. Therefore, any trial intended to evaluate the merit of BSE should take into account the histologic features of the identified tumor and should stratify outcomes by tumor type as well as by method of detection.

The issues of lead time and length bias were addressed in the study by Foster and Costanza.[3] Lead time relates to the fact that a patient may appear to have a longer survival simply because the diagnosis of her disease was made earlier; length bias refers to the likelihood of initial screening programs detecting a relative excess of slow-growing tumors resulting in an increased survival that is independent of any benefit from screening. Although BSE performers identified tumors of smaller size, with less axillary lymph node involvement and shorter patient delays in seeking medical consultation than non-performers, the most important finding was that 75% of the BSE performers were alive after five years, compared with 57% of the non-performers. By the statistical techniques used, this difference was attributed solely to BSE. However, an important question is raised by the way in which the women were grouped for analysis. While logic dictates that more frequent examination is better, there were no differences in mortality between women grouped as practicing BSE once a month and those doing so less than once a month. A significant effect appeared only when the groups were merged to create a never/ever dichotomy. Because of the range of practice behaviors in the "ever" group, it is not clear if the apparent benefits from BSE were due to the method, an increased health conciousness among performers, or the absence of psychological defense mechanisms such as denial.

An issue related to the frequency of practice is quality of performance. With the exception of Huguley and Brown,[6] no attempt has been made to define or evaluate BSE; yet standardization of method is a requirement for any well-designed clinical trial. Sheley[7] identified only 10% of his sample as able to apply an effective breast examination, and Celentano and Holtzman[8] confirmed this finding of inadequate knowledge of correct technique. Whereas 57% of Huguley's and Brown's sample of regular performers were judged competent by a nurse, 65% of those same women believed themselves to be performing BSE correctly.[6] Clearly, then, even among women who do practice BSE, variation in method may account for disparate research results. Thus, not only is compliance a problem,[9] but the nature of that compliance is critical to an evaluation of the method's effectiveness.

Another question to be addressed relates to the health care practices of participants. Perhaps the women who benefit from BSE are those who do not avail themselves of other forms of preventive health care, such as regular physical examinations by a physician.[10] These women may be a unique subpopulation and need to be identified as such. In addition, there are problems associated with self-reporting and the retrospective nature of the published studies: a potential combination of recall bias and social desirability ascribed to BSE may undermine the reliability of the data.

Before exploring further the application of BSE it is essential to define its role as a screening procedure. Studies that compare the effectiveness of BSE with other screening techniques might be misleading. If effective, BSE should be viewed as an adjuvant to cancer control—necessary for the detection of tumors that form between clinical examinations or mammography, or both, and, in some circumstances, as the only screening method available. It is because of the potential importance of BSE that careful assessment of the technique is necessary. As yet, a true evaluation has not been achieved. A prospective design is needed that incorporates a standardized method of BSE together with evaluation of identified tumors by their clinical and histopathological characteristics, and, perhaps, estrogen receptor status. Only then can we judge the effects of this important adjunct to cancer control and assess the true benefits accrued from its practice. Until that time, however, serious concern is raised with regard to any attempt to market the method as a fee-for-service educational program, rather than as an emerging practice in preventive health care. Without really knowing what earlier tumor identification, smaller tumors, or increased five-year survival rates mean in terms of ultimate survival following breast cancer, it is premature to market breast self-examination as one of the most reliable means of detecting breast cancer.

GAIL DIEM, EdD
DAVID P. ROSE, MD, PhD,
Naylor Dana Institute for Disease Prevention
American Health Foundation
Dana Rd
Valhalla, NY 10595

1. Cancer statistics 1985. *CA-A Cancer Journal for Clinicians.* 1985; 35:19-35.
2. Hicks MJ, Davis JR, Layton JM, Present AJ: Sensitivity of mammography and physical examination of the breast for detecting breast cancer. *JAMA* 1979; 242:2080-2083.
3. Foster RS, Costanza MC: Breast self-examination practices and breast cancer survival. *Cancer* 1984; 53:999-1005.
4. Fisher ER, Redmond C, Fisher B: Pathologic findings from the National Surgical Adjuvant Breast Project (protocol no. 4) VI. Discriminants for five-year treatment failure. *Cancer* 1980; 46:908-918.
5. Heuser L, Spratt Jr JS, Polk Jr HC, Buchanan J: Relation between mammary cancer growth kinetics and the intervals between screenings. *Cancer* 1979; 43:857-862.
6. Huguley CM, Brown RL: The value of breast self-examination. *Cancer* 1981; 47:989-995.
7. Sheley JF: Inadequate transfer of breast cancer self-detection technology. *Am J Public Health* 1983; 73:1318-1320.
8. Celentano DD, Holtzman D: Breast self-examination competency: an analysis of self-reported practice and associated characteristics. *Am J Public Health* 1983; 73:1321-1323.
9. Diem G: Barriers to the performance of breast self-examination among a select group of college women (Ed.D. dissertation). New York, Dept of Health Education, Columbia University, May 1982.
10. Cole P, Austin H: Breast self-examination: an adjuvant to early cancer detection. (editorial) *Am J Public Health* 1981; 71:572-574.

The current status of research in breast self-examination

We agree with the authors of the commentary "Has breast self-examination had a fair trial?"[1] that it is not possible to state that any cancer screening program, including breast self-examination (BSE), decreases the mortality from cancer. There are, however, different types of evidence that lead to varying degrees of certainty as to the efficacy of breast cancer screening programs.

There have been to date only two randomized prospective trials reporting the results of screening for early detection of breast cancer. The Health Insurance Plan of New York (HIP) trial utilized physical examination and mammography.[2,3] The HIP study suggests, with about 95% certainty, that for women over the age of 50 these two screening methods were efficacious in decreasing the number of deaths due to breast cancer. Two thirds of the benefit in the HIP study was attributed to the physical examinations and one third to the mammographic examinations. Tabár et al[4] have reported the results of a second randomized prospective trial carried out in Sweden.

Prospective randomized studies, while providing the greatest degree of certainty, still do not provide 100% certainty, are costly and time consuming, and would be difficult to control for a screening technique such as BSE. In the United States, contamination of a prospective randomized trial of BSE by information sharing would be a likely problem. A World Health Organization sponsored prospective trial of BSE to be carried out in Russia is being designed[5]; any data from that trial will be years away. In the Russian trial, randomization will be carried out at the health district or factory level. There is also a British trial of breast cancer screening underway with randomization at the community level.[6] In this trial there are four control populations, two populations in which the intervention is BSE instruction, and two populations in which the intervention is mammography or physical examination, or both.

What degree of certainty can there be that BSE is efficacious in reducing breast cancer mortality? There is evidence to suggest that early detection of breast cancer by physical examination reduces mortality rates from breast cancer: the HIP study, clinical stage survival data, and the improved survival rates for breast cancer that occurred in this country between 1900 and 1950 despite little change in the efficacy of therapy. Equating self-performed physical examination and physician-performed physical examination would imply that BSE should be effective in reducing mortality, but the efficacy of BSE was without any type of scientific validation until 1978.[7,8]

In a study of patients in a given geographic area who had breast cancer that was diagnosed on the basis of histologic evidence, there was a dose-response association between BSE performance frequency (determined retrospectively) and clinical and pathological stage. Subsequent prospective follow-up has demonstrated an association between periodic BSE and survival.[9] The survival advantage for BSE performers persisted after adjustment of the data for a number of covariates and for lead time bias. At five years of follow-up, there were no significant differences in the survival curves between the more frequent and less frequent performers of BSE. There were, however, dose-response differences in tumor size related to frequency of BSE. Five retrospective studies have reported an association between BSE and more favorable stage of breast cancer at diagnosis.[7-12] Three retrospective studies have failed to show an advantage for BSE.[13-15] These latter studies used different methods, and their patient populations were not geographically defined. A number of uncontrolled prospective studies have suggested a benefit for women performing BSE.[16-19] While there is no "proof" that periodic BSE reduces mortality due to breast cancer, there is evidence to support this theory. The degree of certainty that may be possible with randomized controlled trials would likely not be worth the cost of such studies.

Diem and Rose[1] are correct in stating that assessment of the magnitude of the effect of BSE on survival has been limited. In a study of Vermont women with breast cancer, the five-year survival of women who had performed BSE was 18% higher than that of women who never performed BSE. (This is equivalent to 42% fewer deaths in the BSE performers.) A number of factors such as poor proficiency in BSE performance, delay in reporting findings, and a probability that some women will claim more frequent BSE than they have actually carried out, would lead to an underestimate of the magnitude of the beneficial effect of BSE. The lead time bias phenomenon, which had a small effect on these observations, would tend to produce an over valuation of the effect of BSE on survival.

Diem and Rose are also correct that the studies to date have not controlled for the histologic grade of the breast cancer in the various BSE performance groups. While additional biologic information is always of interest, histologic grading (one presumed purpose of which would be to control for varying cancer growth rates) would probably not change the interpretation of the BSE data. The real issue is whether the BSE and survival data are subject to any significant length biased sampling error—whether the screening procedure tends to detect more slowly growing cancers (and thus more treatable cancers) than rapidly growing cancers. Length biased sampling error is a problem in evaluating the effects of a new screening procedure that is applied to a population. The observational studies on the relationship between BSE and survival have not included women who had just adopted the practice of BSE.

BSE is efficacious in decreasing breast cancer mortality. As noted by Eddy,[20] the reduction in mortality attributable to BSE as it is currently practiced by the average woman is about 15%. For the woman who is also undergoing periodic physical examination and mammography, the incremental effect of BSE on survival may be less. For the woman who is particularly competent in her technique of BSE, the benefit may be greater.

Among the numerous scientific questions related to BSE as a cancer screening methodology are the following: How is BSE currently performed? What would be an optimum technique of BSE? Are there trade-offs between the complexity of the technique that is taught to women and their compliance? What are the optimum teaching methods relative to proficiency and compliance? What are the economic costs of an effective BSE instructional, motivational, and reinforcement program? What is the relationship between BSE teaching and performance and any delay in seeking treatment? The answers to these questions should be obtained by testing specific populations of women rather than small groups of volunteers.

In addition to the many scientific questions relative to the role of BSE as a cancer screening program, there are public health policy questions. For example, should BSE be regarded as an alternative or an adjunct to other breast cancer screening techniques? Mammography may be a more efficacious screening technique than either professional physical examination or BSE, but it is also far more expensive. The American Cancer Society, which has taken a position advocating mammographic screening programs, has also stated that "at present there are insufficient facilities or personnel to accomplish this task."[21] At $100/mammogram, it would cost approximately three billion dollars annually to screen the 29,500,000 women in the United States between the ages of 40 and 64. Even with annual mammographic and physical examination screening, cancerous growths can appear between the scheduled screenings. In the National Institutes of Health/American Cancer Society Breast Cancer Detection Demonstration Projects, approximately 17% of the breast cancers were detected during the intervals between screening visits.[22] In a breast cancer detection program conducted in Louisville, KY, 27% of the breast cancers were detected in the interval between screenings.[23] What is the appropriate public health policy relative to promotion of BSE as an adjunct to or an alternative to other breast cancer screening programs? What is the appropriate public health policy relative to any breast cancer screening program versus alternative uses of health care resources?

In many parts of the world, populations of women at risk of developing breast cancer do not have available the health resources of periodic physical examination and mammography. Even in the United States most women do not receive periodic professional breast cancer screening examinations. The BSE efficacy studies already conducted suggest that BSE, even in the relatively unsophisticated form practiced by many women, can decrease breast cancer deaths. At least for women who do not have frequent professional examinations, BSE would appear to provide benefit. The chances that an expensive and lengthy randomized controlled trial, with mortality as the end point, would contradict this theory are small. Just as there has been development in mammography technology, there needs to be development and testing of methods for instruction and motivation in BSE performance. Health education research relative to BSE instructional methodology is in its infancy. Future trials of the efficacy of BSE should be built on the results of BSE educational studies that are designed to improve the proficiency and compliance of large numbers of women.

ROGER S. FOSTER, Jr, MD
Vermont Regional Cancer Center
One S Prospect St
Burlington, VT 05401
MICHAEL C. COSTANZA, PhD
JOHN K. WORDEN PhD
University of Vermont
College of Medicine
Burlington, VT 05405

1. Diem G, Rose DP: Has breast self-examination had a fair trial? *NY State J Med* 1985; 85:479-480.
2. Shapiro S: Evidence on screening for breast cancer from a randomized trial. *Cancer* 1977; 39:2772-2782.
3. Shapiro S, Venet W, Strax P, Venet L, Roeser R: Ten- to fourteen-year effect of screening on breast cancer mortality. *JNCI* 1982; 69:349-355.
4. Tabár L, Fagerberg CJ, Gad A, et al: Reduction in mortality from breast cancer after mass screening with mammography. *Lancet* 1985; 1:829-839.
5. Howard J (ed): *Breast Self-Examination: Issues for Research*. Proceedings of the Working Group Meeting to Explore Issues in Breast Self-Examination. Health Promotion Sciences Branch, Division of Cancer Prevention and Control, National Cancer Institute, 1984.
6. UK Trial of Early Detection of Breast Cancer Group: Trial of early detection of breast cancer: description of method. *Br J Cancer* 1981; 44:618-627.
7. Foster RS Jr, Lang SP, Costanza MC, et al: Breast self-examination practices and breast cancer stage. *N Engl J Med* 1978; 299:265-270.
8. Greenwald P, Nasca PC, Lawrence CE, et al: Estimated effect of breast self-examination and routine physician examinations on breast cancer mortality. *N Engl J Med* 1978; 299:271-273.
9. Foster RS Jr, Costanza MC: Breast self-examination practices and breast cancer survival. *Cancer* 1984; 53:999-1005.
10. Feldman JG, Carter AC, Nicastri AD, Hosat ST: Breast self-examination, relationship to stage of breast cancer at diagnosis. *Cancer* 1981; 47:2740-2745.
11. Huguley CM, Brown RL: The value of breast self-examination. *Cancer* 1981; 47:989-995.
12. Tamburini M, Massara G, Bertario L, Re A, Di Pietros: Usefulness of breast self-examination for an early detection of breast cancer. Results of a study of 500 breast cancer patients and 652 controls. *Tumori* 1981; 67:219-224.
13. Gould-Martin K, Paganini-Hill A, Casagrande C, Mack T, Ross RK: Behavioral and biological determinants of surgical stage of breast cancer. *Prev Med* 1982; 11:429-440.
14. Senie RT, Rosen PP, Lesser MI, Kinne DW: Breast self-examination and medical examination related to breast cancer stage. *Am J Public Health* 1981; 71:583-590.
15. Smith EM, Francis AM, Polissar L: The effect of breast self-exam practices and physician examinations on extent of disease at diagnosis. *Prev Med* 1980; 9:409-417.
16. Gastrin G: *Breast Cancer Control: An Early Detection Programme*. Stockholm, Almquist and Wiskell, 1981.
17. Gästrin G: Programme to encourage self-examination for breast cancer. *Br Med J* 1980; 281:193.
18. Gilbertsen VA, Nelms JM: Breast cancer. Improving long-term survival. *Minn Med* 1980; 63:160A,C.
19. Mahoney LJ, Bird BL, Cooke GM: Annual clinical examination: the best available screening test for breast cancer. *N Engl J Med* 1979; 301:315-316.
20. Eddy DM: Finding cancer in asymptomatic people: estimating the benefits, costs, and risks. *Cancer* 1983; 51:2440-2445.
21. American Cancer Society: ACS report on the cancer related health checkup. *CA* 1980; 30:194-240.
22. Baker LH, et al: Breast Cancer Detection Demonstration Project: Five-year summary report. *CA* 1982; 32:194-225.
23. Heuser L, Spratt JS Jr, Polk HC Jr, Buchanan J: Relation between mammary cancer growth kinetics and the intervals between screenings. *Cancer* 1979; 43:857-862.

Breast self-examination—a practice whose time has come?

In two previous commentaries in this issue on breast self-examination, Diem and Rose[1] raise the question as to whether breast self-examination (BSE) has had a fair trial, and Foster et al[2] respond by describing the current evidence that supports the practice of BSE.

Diem and Rose note that in a recent study by Foster et al,[3] 75% of BSE performers were alive after five years compared to 57% of the non-performers. But they question the validity of grouping women into only two categories: never practiced BSE or did practice BSE. While undoubtedly this a crude approach, the survival rates are similar to results that we found in Brooklyn.[4] The Brooklyn results indicate that four-year survival was 73% in women who practiced BSE monthly, 75% for those who performed BSE three to six times yearly, and 60% for those who practiced BSE less than once a year.

Thus, two studies have found that the practice of BSE is associated with longer survival. In both studies there was no difference in survival between women who practiced BSE monthly and those who practiced it less frequently.

The absence of a dose-response relationship between BSE frequency and survival could result from several possibilities. One of these is poor quality of BSE performance. Even though a woman may perform BSE frequently, she may not do it well. Neither study evaluated proficiency of performance, and poor performance of BSE has been reported by others.[5] Another possibility is a threshold effect: the benefit of doing BSE monthly may be comparable to doing it several times a year. Or it may be that the association between BSE frequency and survival is not a causal relationship.

Diem and Rose correctly point out that both lead time bias and length sampling bias can influence the association between BSE and survival. Lead time bias results from unequal lead time to appearance of disease. Breast cancer detected by BSE may have a shorter lead time and consequently appear to be diagnosed at an earlier stage and appear to be associated with longer survival, when in actuality this may not be true. Length sampling bias occurs because slower growing tumors are more likely to be detected by BSE. Foster et al[3] have considered these biases and conclude that they do not alter their findings. The only way to eliminate these biases is through a randomized trial. It is not clear whether a randomized trial is the "true evaluation" that Diem and Rose seek, or if they would accept the results of a prospective trial without random allocation.

Foster et al persuasively present the arguments against conducting a randomized trial of BSE. Instead they rely on Eddy's[6] concept of "degrees of certainty" and conclude that the weight of evidence indicates a beneficial effect of BSE. Additional evidence comes from a population-based (non-random) prospective study of BSE in England,[7] where women who discovered a breast mass during BSE had smaller tumors on initial finding compared to others. But, the authors noted that the evidence was not sufficient to conclude that a community-based BSE campaign would produce an overall improvement in stage at presentation. Herein lies the heart of the dilemma.

The evidence, although incomplete, favors a beneficial effect of BSE. The key issue is whether the evidence is sufficient to justify the cost of community education programs.

As Foster et al note, much needs to be learned about the best ways to motivate women and to teach the practice of BSE. Until more information is gathered about these aspects and the effectiveness of community education programs, large-scale efforts in this direction seem premature. However, physicians and nurses, at little additional cost, have the opportunity to instruct and encourage the practice of BSE in the context of general medical care. Therefore, the cost-benefit ratio is more favorable under these circumstances, even with the present incomplete evidence. The American Medical Association Council on Scientific Affairs recommends that physicians promote BSE and teach or reinforce it at regular examinations.[8] Such opportunities may be especially important for women who do not receive frequent examinations.[9]

In sum, BSE is a practice that should be encouraged as part of a woman's regular medical care, but evidence is not yet sufficient to justify large-scale community-based intervention.

JOSEPH FELDMAN, DrPH
Associate Professor
Department of Preventive Medicine
& Community Health
SUNY-Downstate Medical Center
450 Clarkson Ave.
Brooklyn, NY 11203

1. Diem R, Rose DP: Has breast self-examination had a fair trial? *NY State J Med* 1985; 85:479–480.
2. Foster Jr RS, Costanza, MC, Worden JK: The current status of research in breast self-examination. *NY State J Med* 1985; 85:480–482.
3. Foster RS, Costanza MC: Breast self-examination practices and breast cancer survival. *Cancer* 1984; 53:999–1005.
4. Feldman JG, Carter AC, Nicastri AD, Hosat ST: Breast self-examination, relationship to stage of breast cancer at diagnosis. *Cancer* 1981; 47:2740–2745.
5. Holtzman D, Celentano DD: The practice and efficacy of breast self-examination: a critical review. *Am J Public Health* 1983; 73:1324–1326.
6. Eddy DM: Finding cancer in asymptomatic people: estimating the benefits, costs and risks. *Cancer* 1983; 51:2440–2445.
7. Philip J, Harris WG, Flaherty C, Joslin CA, Rustage JH, Wijesinghe DP: Breast self-examination: clinical results from a population-based prospective study. *Br J Cancer* 1984; 50:7–12.
8. American Medical Association Council on Scientific Affairs: Early detection of breast cancer. *JAMA* 1984; 252:3008–3011.
9. Cole P, Austin H: Breast self-examination: an adjuvant to early cancer detection. (editorial) *Am J Public Health* 1981; 71:572–574.

FROM THE LIBRARY

CARCINOMA OF THE BREAST

Much attention has been attracted by the press by the reports coming from the Gratwick Laboratory, Buffalo, to the effect that cancer has been demonstrated to be a parasitic affection, and that a curative serum has been discovered (at least for mice). These findings have been in sharp contrast to the third report of the research committee of Harvard University, which holds a very negative view as to the parasitic origin of cancer and rather follows, it seems to me, the plausible pathological theory of Cohen Lewis, viz., that carcinoma is the result of misplaced embryomal cells, which later in life take on unusual activities. In passing, I would observe that it has seemed to me that if some of the pathologists would stop theorizing and attempt to unravel the many hidden mysteries which cell life contains, they would be the better able to interpret their findings, and the sooner, possibly, come to a solution of their problem.

E.C. THOMPSON, MD
(*NY State J Med* 1905; 5:324)

This section is designed to offer rapid publication (usually within 1–3 months) of new clinical, epidemiologic, and laboratory observations, as well as of comments on articles published in the Journal *or on any topic related to the role of the physician. Letters should be typed double-spaced and should include the professional affiliation, highest academic degree, address, and signature of each author. Preference is given to letters not exceeding 450 words. All letters are personally reviewed and acknowledged by the Editor. Address correspondence to Editor,* New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042.*

Wounds of war

TO THE EDITOR: WD Sharpe's reminder in "Wounds of war" (1985; 85:61–63) that war is hell presents little new to those who have experienced the hell. The 1864 excerpts quoted from the writings of Surgeon Chisolm could have been written in 1944, and would have been just as applicable. The harrowing sights of torn and ripped bodies never really leave one's psyche, even after the lapse of many years, nor does the memory of the nauseating, sweet smell of gangrene and decay leave one's nostrils.

Dr Sharpe's article is addressed to an audience of physicians, many of whom have served in the military. As one of that group, I find myself in disagreement with the inference that there should be no "satisfaction at having served in the armed forces or been in a war." Surely those who fought the war against Hitler have a right to a sense of satisfaction in having done their part to rid the earth of a monster. Nor do I agree that the satisfaction is a "male" satisfaction. Ask the nurses who operated with the surgeons and who cared for the wounded and dying whether they thought the role they played was masculine.

The "satisfaction" felt by those in the medical corps was not based on killing or on a desire to kill, but on the knowledge that they had saved lives. Their endeavors did not create any increased sense either of masculinity or feminity, but they did result in an ennobling feeling of having rendered a much-needed succor to the helpless and hapless victims of war.

ROBERT E. ROTHENBERG, MD
Cabrini Medical Center
227 East 19th Street
New York, NY 10003

Dr Sharpe did not wish to reply—Editor

Searching for alternatives to war

TO THE EDITOR: The cure for war[1] may already exist among us. Is there a case on record in which a modern elected government (democracy) has waged war on another? (England was not one until the reform act of 1832.)

Democracies have had to defend themselves against different versions of military dictatorships. Some dictatorships, through unconditional military defeat and effective occupation, have become democracies, and hence probably immune to starting wars (Germany, Italy, and Japan, for example). Impotent democracies (Czechoslovakia) have been forcibly converted to dictatorships. Ghandi's India has successfully defended itself against military dictatorship on its border. Military dictatorships within the defense perimeter of strong democracies, finding their form of government unworkable and maintenance of power deteriorating, have become democracies (Spain, Greece, Portugal, Argentina, indeed most of Latin America). As long as these countries are democracies, their initiating war seems improbable. Defenseless people, nonviolent and passive though they may be, are at great risk confronting the pathology of a military dictatorship (such as the European Jewish communities faced during World War II).

Democracy, probably the political equivalent of health in biological systems, should, in this context, be the subject of examination by epidemiologists, psychiatrists, and the hard sciences. Why is it that military dictatorships, with variable degrees of severity, are coincident with Islamic society? Why do terrorists want to oppose and disrupt the democratic system? Can a democratic system, incentive, and private property be separated? Are "political" and "economic" insufficient terms for these matters, and shouldn't we view them as some of the most basic frontiers in the prevention and cure of the disease of war? Is there a "goodness factor" indispensable for democratic institutions, and, hence, the substantial elimination of war? And so on.

Alternatives to war there may be. But until representative democracy has become the prevailing mode worldwide, defense against those who would make war looks inescapable.

F. CRAIG FISHER, MD
160 East Ave
Lockport, NY 14094

1. Engebretsen B: Searching for alternatives to war. *NY State J Med* 1985; 85:55–56.

Roswell Park

TO THE EDITOR: I am grateful to John S. Haller, Jr (1985; 85:207-209) for jolting me into looking up Roswell Park's biography. I knew that he was a great surgeon and that he was a Buffalonian. On the other hand, I did not know that he was born in Connecticut in 1852.

It is Haller's referring to him as "Roswell Park of Connecticut" that sent me to the library (the Reference Library of St. Bonaventure University). While Park was born in Connecticut, it would appear that he spent very little time there. His father, the Reverend Roswell Park, opened Racine College, Racine, Wisconsin, in 1852. It is from Racine College that the younger Park received his BA and MA degrees. He was awarded the MD degree from Northwestern in 1876.

Park taught at various medical schools in Chicago until 1883. He came to Buffalo in 1883 as Professor of Surgery, a post that he held for the rest of his life. It was from Buffalo that his fame spread. It was from Buffalo that he was elected President of the Medical Society of the State of New York and of the American Surgical Association. Need I go on? "Roswell Park of Connecticut" indeed!

PAUL deR KOLISCH, MD
6 Baxter Place
Friendship, NY 14739

In reply: Americans are a notoriously mobile people. In the past, as with today, the average American family moves every few years, often in a westerly direction, and a person's achievements may be noted as much by geographical location as by any other criterion. Perhaps the safest, if not the only, biographical placement is the area of one's birth. That, and that alone, cannot change. For all we know, one's very bones may be disinterred after death.

JOHN S. HALLER, JR
Associate Vice President/Dean of
Graduate Studies
California State University, Long Beach
Long Beach, CA 90840

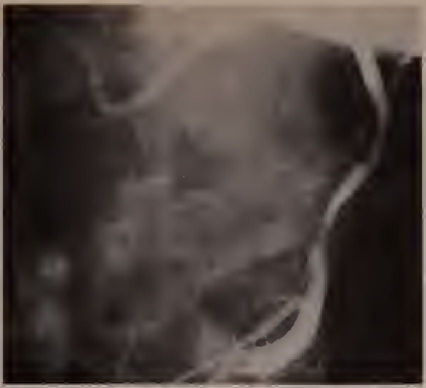
Spontaneous dissection of the right coronary artery

TO THE EDITOR: Spontaneous coronary artery dissection is a rare entity. To our knowledge, only three cases[1–3] have been diagnosed during life and documented by coronary arteriographic studies. The present report is unusual because the dissection occurred in the right coronary artery of a 52-year-old man who had unstable angina and no previous myocardial infarction.

Case Report: A 52-year-old man with a history of angina that was controlled with nitrates, beta blockers, and diuretics sought treatment in July 1983 when angina de novo developed. After increasing the dosages of his medications, a stress test was performed. The test was positive and demonstrated 2 mm ST segment depression. The patient had no history of hypertension or diabetes mellitus. There was a strong family history of coronary artery disease. The patient smoked cigarettes until the onset of symptoms.

The patient was admitted to the hospital, at which time there were no significant findings on physical examination. An electrocardiogram showed regular sinus rhythm with prominent R waves in V_1 and V_2. A chest film was clear, and the heart was not enlarged. Laboratory studies were remarkable only for an elevated cholesterol level of 351 mg/dL.

At cardiac catheterization, the right heart pressures were normal; the left heart pressure revealed an elevated left ventricular end-diastolic pressure at 18 mm Hg. A left ventricular cine angiogram revealed a normal contractile pattern with no evidence of mitral regurgitation.


Left anterior oblique view of the right coronary artery. The arrow points to the area where the dissection begins. The complete dissection is only visualized on a moving cineangiogram.

A selective coronary angiogram demonstrated a normal left coronary artery with small but patent left anterior descending and circumflex arteries. The right coronary artery was visualized using an 8 French right Judkins catheter. Cannulation of the orifice was accomplished without difficulty. Over 95% narrowing was observed in the mid portion. In the distal half of the right coronary artery there was simultaneous filling of both the true and false lumen, presenting characteristic angiographic features of a dissection. The dissection appeared to start just distal to the stenotic segment, included the acute marginal branch, and extended just proximal to the point of origin of the posterior descending artery.

Since the patient continued to have angina secondary to the coronary stenosis, the decision was made for him to go to surgery. At surgery a right coronary artery bypass was performed. The patient had an unremarkable postoperative course. He was seen six months later with no new symptoms; no further follow-up was indicated.

Discussion: Few cases of spontaneous dissection have been reported. A review of the literature reveals that most cases involve women who have dissections of the anterior descending artery, which are found at necropsy. The majority of these patients have eosinophilic infiltrates.[1] Even fewer cases have been reported in patients without prior history of cardiovascular disease or infarct, with the dissection being found at cardiac catheterization.[2]

This case is unusual because the dissection occurred in the right coronary artery of a man with unstable angina and no myocardial infarct. There was no history of trauma. The patient had no evidence of vasculitis, did not have characteristics of Marfan's syndrome and was not hypertensive. The risk factors present included cigarette smoking, hypercholesterolemia, and a family history of coronary artery disease. Cystic medial necrosis could not be ruled out.

Recognition of primary coronary artery dissection by angiographic studies is dependent on visualization of two lumens. In this patient, two distinct lumens were identified on selective injection of the right coronary artery.

JOSEPH E. DeJUNCO, MD
ROBERT I. HAMBY, MD
MANUEL VEGA, MD
The Heart Center
St. Francis Hospital
100 Port Washington Blvd
Roslyn, NY 11576

1. Robinowitz M, Virmani R, McAllister Jr, HA: Spontaneous coronary artery dissection and eosinophilic inflammation: a cause and effect relationship? *Am J Med* 1982; 72:923–928.
2. Palank EA, Dawson Jr JT, Cowen GD, Tysinger JR: Primary dissecting aneurysm of the right coronary artery. *Chest* 1977; 72:774–776.
3. Robinson PG, Mayers DL: Spontaneous dissection of left anterior descending coronary artery. (letter) *Am J Med* 1983; 74:496.
4. Forker AD, Rosenlof RC, Weaver WF, et al: Primary dissecting aneurysm of the right coronary artery with survival. *Chest* 1973; 64:656–658.
5. Claudon DG, Claudon DB, Edwards JE: Primary dissecting aneurysm of coronary artery. A cause of acute myocardial ischemia. *Circulation* 1972; 45:259–266.

Scalds of the lap

TO THE EDITOR: Recent analysis of accidents in a nursing facility by Jacob Dimant, MD (1985; 85:202-205) showed that the majority were from falls, and only six of 310 from burns. Burns were not further discussed, as they were of low incidence (6%), and were "usually" mild.

Of importance was that these were all from spilled hot liquids. Attention should be directed to the liability for serious burns from scalding of the lap area, seen infrequently but with devastating results. This occurs typically in a hemiplegic person with a degree of dementia who is seated in a chair or lying in a Gatch bed, folded at the waist. Despite instructions to wait until a nursing assistant returns to pour hot fluid from the metal thermos on the tray deposited on a wheeled table, the patient does not. Reaching to pull the table closer or to manipulate the flask, it overturns. The hinged top falls open and the full content gushes as a bolus toward the patient. The metal carafes, designed for maintaining temperature of water drawn boiling from an urn during transport from kitchen to ward, are efficient.

The table is next to or over the bed or chair, and the lap is drenched. Escape by the patient is impossible. High temperature fluid pools between the legs, over the genitalia and perineum, under the buttocks, and on the lowermost abdomen, and puddles in the groin flexure creases. There is not just initial transmission of heat from spilling but continued conduction of heat from immersion. With the relative thinness of the skin of the lower abdomen, inguinal line, and upper medial thighs, relatively short exposures produce full thickness injury. Beyond this is the cutaneous atrophy with increased fragility of aging.

Early appraisal has been misleading, as the initial appearance of the tissue is red and not blistered or weeping, erroneously suggesting only a superficial erythema. It is actually a deep wound whose red discoloration is due to hemoglobin fixed in the tissues.[1] Nursing requirements are multiplied in caring for a necrotic wound intimately exposed to fecal and urinary contamination, and grafting is ultimately called for. The outlook may be bleak given the stresses of thermal injury, infection, immobility, and surgery in an older patient. Recovery is long, and mortality is increased.

Analysis has shown that these accidents arise from impatience with the delay between the time the tray is delivered, and when assistance arrives to pour the hot water for tea or coffee. Reality dictates that some delay is inevitable, and that patients are not amenable to precautionary instructions even if repeated. The solution is that of keeping the mobile table well out of reach of all patients who require assistance, until the tray has been fully readied for the meal. This is taught to staff as a common sense parallel to the well understood dictum of leaving no matches with an immobilized patient who smokes.

Scalding to the nether regions from spills in a sense represents a disease of medical progress. As the leaking hot water bottle scalds of yesteryear[2] disappeared along with the poultice, modern-day central preparation of heated meals transported in carts and carafes appeared. It is a paradox that the more efficient the delivery, the hotter the water, and the more the likelihood of burn disaster with puddling. The counsel of Dimant for instructional investigation of each accident, even "near miss" mild burns, is soundly seconded.

BENNETT M. DERBY, MD
Department of Neurology
New York University School of Medicine
550 First Ave
New York, NY 10016

1. Artz CP, Moncrief JA, Pruitt BA Jr: *Burns. A Team Approach.* Philadelphia, W B Saunders, 1979, p 27.
2. Muir IFK, Barclay TL: *Burns and Their Treatment.* London, Lloyd-Luke, 1962, p 7.

Repair of inguinal hernia with Marlex mesh

TO THE EDITOR: Marlex mesh is a common prosthetic material used for hernioplasty. We present our experiences with its use in inguinal hernia repair.

Case Report: The study included 80 male patients, aged 12 to 80 years. Fifty-seven pa-

---

Medical approval for a driver's license

TO THE EDITOR: I believe my recent correspondence with the New York State Department of Motor Vehicles concerning the role and responsibility of a neurologist and neurosurgeon in supporting a patient's application for a driver's license would be of interest to many physicians in the state. (Permission to reprint the department's reply has been obtained.)

DAVID GREEN, MB, BS
Seton Hall
St. Mary's Hospital
Troy, NY 12180

Driver Improvement Bureau
Medical Review Unit
NYS Department of Motor Vehicles
Empire State Plaza
Albany, NY 12226

Dear [Sir]:

I have been aware of the fact that a neurologist can now provide a patient with a statement concerning his ability to drive a car, despite a recent convulsive disorder episode.

Some two or three years ago, the patient had to remark that he was convulsion-free for one year.

I would very much appreciate your sending me a letter concerning my legal responsibilities in this matter. I wish to learn the following from your legal department:

If I sign a letter stating that the patient can drive a car two or three or six months after a convulsion and then has an accident which causes bodily harm to the patient and to others, am I legally liable? Can the patient or the other individuals who are injured in the accident bring suit against me if I have signed such a letter as above?

Years ago there was a one-year limit and waiting period, and I felt reasonably confident that the administrative ruling of the Department of Motor Vehicles would protect the physician from suit. However, now that it is up to the neurologist to make up his or her mind, I wonder whether the neurologist isn't open to legal challenge and a suit on the basis of his misjudgment.

Whatever help you may be able to give me in this matter would be deeply appreciated. I have personally decided not to sign any letter for any convulsive disorder patient unless they state that they have been convulsion free for one year, and I am very hesitant on legal grounds to do beyond this. I am anxious to know whether my attitude in this matter is justified.

Sincerely yours,

Dear Dr Green:

I have received your letter of February 28, 1985, as to your liability if you sign a letter permitting a patient to drive two, three or six months after a convulsion, and he later has an accident which causes bodily harm to him and others.

There is no statute specifically protecting a physician from liability in these cases. Thus, it is possible he could be held liable under the laws of negligence. Nevertheless, I suggest that you direct your question to your own personal attorney.

Enclosed is form MV-80U.1. Please note "Request for Information from Physician." What you are asked to do is to render an opinion that the patient's medical condition is such that it would not interfere with his safe operation of a motor vehicle on the public highways.

I regret I am unable to give you a more favorable reply.

Very truly yours,
JOHN A. PASSIDOMO
Commissioner of Motor Vehicles
By: JOYCE M. WRENN
Assistant Counsel

tients had an indirect hernia, 16 patients had a direct hernia, and a combined indirect-direct hernia was found in seven patients. Seven hernias were irreducible and five were obstructed; 11 were recurrent hernias. Marlex mesh was used as an onlay graft after Bassini's repair. All patients received prophylactic antibiotics.

Wound infection and dehiscence occurred in one patient (1.25%). Serum accumulation responding to needle aspiration was observed in 12 patients (15%), however, it did not interfere with wound healing. In six of these 12 patients, various predisposing factors like obesity, diabetes, irreducibility, obstruction, and recurrence were present. In no case was the mesh rejected or required to be removed because of persistent infection. During a follow-up period of 12 to 24 months, two patients experienced a recurrence. Both these patients had already had recurrent hernias. No recurrence was seen in patients with primary (non-recurrent) inguinal hernias. Both the recurrences occurred within two months of the repair.

Discussion: Marlex—a knitted polypropylene mesh—was introduced by Usher and colleagues.[1,2] Its use is indicated in the repair of indirect inguinal hernia with a large internal ring and weak posterior wall, direct inguinal hernia with severely attenuated transversalis fascia, combined indirect-direct inguinal hernia, and recurrent inguinal hernia.[3] We have used Marlex mesh in all cases of inguinal hernia. Wound infection was the only significant post-operative complication and was seen in 1.25% of cases. (Seromas were more frequent but did not interfere with wound healing.) Usher[4] noted post-operative complications in 4.3% of 183 cases. Mesh rejection and removal due to persistent infection has been reported.[4] In none of the patients reported here was the mesh rejected or required to be removed.

The majority of recurrences after hernia repair with the use of prostheses occur during the first year after surgery, and there is no worsening of results thereafter.[5] Our observations agreed with this finding—all recurrences occurred within two months of the repair. During a follow-up period ranging from 12 to 24 months, an overall recurrence rate of 2.5% was observed—the same as that reported by Usher[6] (3% in a series of 286 cases). In our experience, all the recurrences were observed in patients who had had recurrent hernias; no primary (non-recurrent) inguinal hernia repaired with Marlex mesh recurred. Bassini's repair is an accepted and established technique of inguinal herniorrhaphy. Marlex mesh further strengthens the posterior wall of the inguinal canal. We have used Marlex mesh, with an acceptable morbidity rate and a lowered recurrence rate, and recommend it for the repair of various types of inguinal hernia.

VINAY K. KAPOOR, MS
MAHESH C. MISHRA, MS
TUSHAR K. CHATTOPADHYAY, MS
LALIT K. SHARMA, MS
Department of Surgery
All India Institute of Medical Sciences
New Delhi 110029 India

1. Usher FC, Gannon JP: Marlex mesh: a new plastic mesh for replacing tissue defects. Experimental studies. *Arch Surg* 1959; 78:131-137.

2. Usher FC, Fries JG, Ochsner J, Tuttle LL Jr: Marlex mesh: a new plastic mesh for replacing tissue defects. Clinical studies. *Arch Surg* 1959; 78:138-145.

3. Smith RS: The use of prosthetic materials in the repair of hernias. *Surg Clin North Am* 1971; 51:1387-1399.

4. Usher FC: Hernia repair with Marlex mesh. An analysis of 541 cases. *Arch Surg* 1962; 84:325-328.

5. Stoppa RE, Rives JL, Warlaumont CR, Palot JP, Verhaeghe PJ, Delattre JF: The use of Dacron in the repair of hernias of the groin. *Surg Clin North Am* 1984; 64:269-286.

6. Usher FC: The repair of incisional and inguinal hernias. *Surg Gynecol Obstet* 1970; 131:525-530.

ERRATUM

The cost of *The Heart: Physiology, Metabolism, Pharmacology, Therapy,* by Lionel H. Opie, MD, (1985; 85:231) is $55.00.

FROM THE LIBRARY

INGUINAL HERNIA

Regarding the cure of inguinal hernia by injection, much may be said for and against it. . . . The fact must not be overlooked that a large percentage of those having inguinal hernia are working people, and the question of being compelled to lose three or four weeks' time is a very important matter with them. By the injection method no time need be lost. You can treat the patient, and in an hour he can be at his work. . . .

I have used the following formula: Fluid extract quercus alba . . . 1½ ounce; solid extract quercus alba . . . 1½ drachm; carbolic acid crystal, iodine resublimed . . . āā 2 drachms; morphine sulphate . . . 10 grains. . . .

Placing the patient in a recumbent position and having reduced the hernia, the index finger is placed in the inguinal canal by invaginating the skin over the external ring and pushing the finger up the canal until the tip of the finger reaches the internal ring. By feeling over the site of the internal ring the tip of the finger in the canal can be felt, and the needle should be inserted at this point, directly over the finger tip, and pushed down quickly until it passes the end of the finger. The fluid should then be injected slowly, three to five minims being injected, and as the finger is withdrawn slowly the needle is made to follow it for about half an inch, during which time the fluid is injected slowly, and when the twenty minims have been injected the needle should be withdrawn suddenly so as to avoid depositing any of the fluid in the subcutaneous tissues, for if any fluid is deposited in the subcutaneous tissue it will cause intense burning for a short time. The truss should be placed in position immediately and the patient should lie down for about half an hour. . . . The injection should be repeated once a week for three or four weeks, when if the hernia does not come down after having taken the truss off and having tested it by the patient's coughing or stooping, you need not give any more injections. . . .

THOMAS P. SCULLY, MD
(*Trans Med Soc State* of *NY* 1900, pp 349-351)

Medical education in New York City in 1866–1867

A student's notebook of Professor Charles A. Budd's lectures on obstetrics at New York University—Part I*

BERT HANSEN, PHD

Because so much of the history of medicine must be written out of polished, public documents such as journal articles and textbooks, it is fortunate when an informal, unsanitized document is accidentally preserved. We know that students have been recording their teachers' explanations at least since Plato listened to Socrates; and after the 14th century, when paper came to replace the more expensive parchment, those notes have been produced year in and year out in countless numbers. But because these records seemed less important than formal presentations, and, in recent centuries, because they went out of date within the span of one's professional career, they have rarely been preserved. Even for the recent past, most medical school libraries and archives can display only a few notebooks, and these are often fragmentary or lacking adequate identification.

Considering the tens of thousands of medical alumni of American colleges—all of whom wrote out extensive lecture notes, lab reports, review sheets, problem sets, and so forth—this dearth of an historical record for such a formative experience as medical school is as remarkable as it is unfortunate.

Given this general situation, one encounters the notes that Charles Inches took in Dr Charles A. Budd's obstetrics course with special delight—for not only can we identify the student, the teacher, the college, and the date, but we have in them an apparently complete *verbatim* transcript of 26 full lectures given on Wednesday, Friday, and Saturday mornings from December 12, 1866 through February 15, 1867. Inches' report takes us into the 19th-century classroom almost as vividly as would a tape recording.

Course notes—whether compiled by the teacher or a student—allow historians to discern where teaching departs from the published textbooks of the time, and how the latest research might have been used in training the profession's new generation. This particular notebook from Budd's course at New York University gives even more, for in reporting every word it also includes those less formal aspects: the asides, dirty jokes, personal advice, judgments about colleagues and textbooks, and fatherly concern for the students' well-being and future success. Only rarely do historians have the opportunity to get so close to the lives of people who lived in the 19th century as to be able to eavesdrop on their speech, listening to every word, minute by minute. Through Inches' manuscript record, a modern reader can even feel the pulsing, driven style of narration as Budd recounts complicated deliveries, where human efforts, both maternal and medical, contend with unknown, uncontrollable biological forces: heart-rending dramas only some of which have happy endings. Even today, Budd's presentation holds the attention of historians and clinicians alike, for he dealt with many issues of perennial interest.

Recently, historians of the 19th century have devoted numerous studies to changing childbirth practices, to the increasing use of general practitioners instead of midwives—even for normal deliveries, and to the rise of obstetrics as a specialization within medicine.[1-13] Budd's lectures contain much material speaking to these important events. In particular, historians will want to observe the state of Budd's art with respect to medications (especially chloroform, ergot, and opium), the use of forceps and other instruments, abortion, the procedure of bloodletting, awareness of sepsis, the etiology of ophthalmia neonatorum, and Budd's attitudes toward his students, his profession, his fees, his patients, their supportive women friends, midwives, and perhaps women in general. Midwives, however, unlike the rest of this list, are conspicuous only by their absence. Nevertheless, some parts of this text are quite important for the history of midwifery, because Budd reports a number of specific practices among male doctors that one historian has attributed primarily to midwives.[14] From all these notes one can learn what a prominent member of New York City's medical elite deemed important for young practitioners to know.

Those young men, just out of their teens, were oblivious to our retrospective concerns; they wanted to know "how to do it." And this they had explained in clear and persua-

---

Address correspondence to Professor Hansen, Department of History, New York University, 19 University Place, New York, NY 10003.

Grateful acknowledgment is made to the Institute for Advanced Study, Princeton, NJ, for financial support of this research by a fellowship for 1984–85.

* This article is being published in two parts. Part II will appear in the September 1985 issue of the *New York State Journal of Medicine*. Several explanatory notes in Part I may refer to material that will appear in Part II.

sive phrases. Budd was well known among his colleagues for just that quality; according to James V. Ricci, the historian of obstetrics and gynecology in New York City,

> [Budd's] fame rests primarily on his fine qualities as a teacher. Both in the lecture room and before gatherings of the various medical societies, particularly the New York Obstetrical Society, he was always clear, methodical, analytical and enthusiastic.[15]

When Ricci published this statement in 1945 he knew Budd's lecturing style only by hearsay and reputation, possibly borrowing this evaluation from Budd's obituary in *The Medical Record*.[16] Today we are privileged to be able to consider Budd's style directly.

But before entering Budd's classroom to listen in, it is important first to familiarize ourselves with the medical community of his time, the persons involved, the character of surviving documents, and the range of subjects covered in Budd's course.

MEDICINE IN NEW YORK IN THE 1860S

The medical world of New York City in which Budd and his students circulated was large and diverse. The character of this world in 1866 has been deftly sketched by Rosenberg in "The practice of medicine in New York a century ago." [17] Among the estimated 1,500 practicing physicians in New York in the late 1860s, as in other major East Coast cities, it was hospital and dispensary service that marked off the leading physicians—who felt they contributed to the development of medicine—from those who simply practiced it.[18]

The more exclusive community constructed by the elite physicians was largely defined through their common linkages to the hospitals: teaching there or having consulting appointments, and more generally having had prior experience as residents.[19] New York's two teaching hospitals were already gigantic institutions: Bellevue had a thousand beds and nearly 8,000 patients in 1866; Charity Hospital on Blackwell's Island treated more than 7,500 patients. Such rich learning opportunities offered by so much clinical material did not go unnoticed or unexploited. The shared hospital connections were further reinforced by involvement in medical publishing, primarily as medical journal editors and contributors, and by participation in scientific and professional societies. Status and power, not to speak of income, were concentrated within this circle. These established physicians would almost all have been among the 273 members of the New York Academy of Medicine, and the most important among these were further distinguished by their participation in the 34-member Medical and Surgical Society of New York.[20]

In December 1866 the University Medical College was just 25 years old; it had then a tenuous and problematic association with the University of New York, later named New York University. A fire in May 1866, which destroyed the Medical College building on 14th Street, prompted plans to relocate to the Bellevue Hospital neighborhood, but from 1866 to 1869 college sessions took place downtown at Church Street and Broadway in the New York Hospital.[21]

The field of obstetrics was growing rapidly in America in the decades after 1850, and especially so in New York City.[9] While books in the field had been appearing for several decades, the new level of professionalization after midcentury is indicated by the establishment of the New York Obstetrical Society in 1863, *The American Journal of Obstetrics and the Diseases of Women and Children* in New York City in 1868, the American Gynecological Society in 1876, and the American Association of Obstetricians and Gynecologists in 1888.[22]

PROFESSOR BUDD

When this course began, Professor Charles Amos Budd was 35 years old;[23,24] his student Charles Inches had just turned 20 on December 7.[25] A native New Yorker, from a long line of physicians, and married to a doctor's daughter, Budd had travelled in Europe after earning his MD at the University Medical College in 1852, where he studied at lying-in hospitals in Dublin and Paris.[23] In 1855 he was elected a Fellow of the New York Academy of Medicine.[26] In the late fifties he published a few brief case reports.[27-32] From 1860 to 1863 he was a professor of obstetrics at the short-lived New York Medical College[33] (1850–1864, with a complete changeover of faculty members in 1860). Budd was one of the founders[26] of the New York Obstetrical Society in 1863 and served a term as its president in 1871.[34] In 1863, when Gunning S. Bedford resigned his professorship at the University Medical College due to ill health, the post was offered first to James D. Trask, who declined it,[35] and then to Budd, who accepted it, serving from 1863 to 1876 as Professor of Obstetrics, with Diseases of Women and Children, and Clinical Midwifery. For these years the college's catalog announced "*Budd Prizes.* Prof. Budd will give a prize to the member of the graduating class who shall pass the best examination in his department at the time of his final examination." [36] In 1876 Budd resigned due to ill health and was

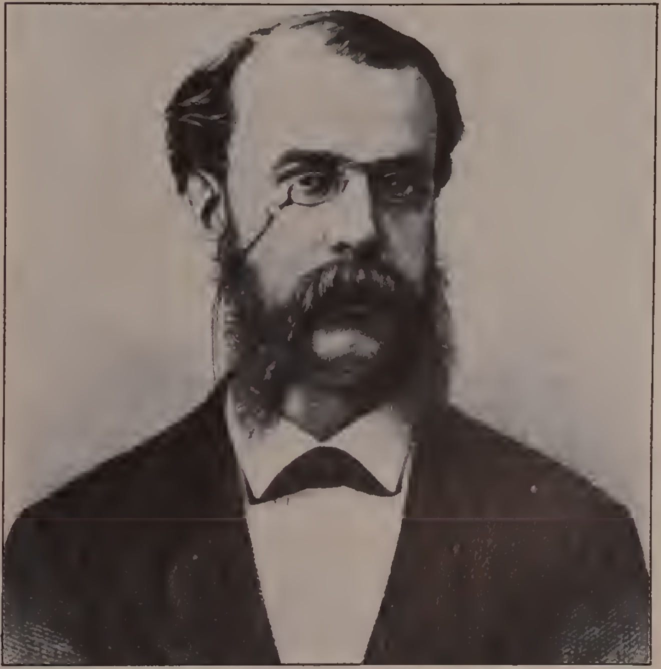


FIGURE 1. Charles Amos Budd, MD (1831–1877). Undated photograph, probably from early 1870s (courtesy of the New York Academy of Medicine Library).

appointed Professor Emeritus; he died a year later in May 1877 at the age of 46 (Fig 1).

Budd, like many of New York's medical leaders, had studied in Europe, an experience that was recognized as having pecuniary as well as intellectual advantages. In 1876, this fact was explained by the young William Welch to his father:

> The . . . culture and knowledge of the world to be derived from a year abroad are not so much to be considered as the actual addition . . . to my stock in trade, so to speak, [as] that it will yield a larger return. Even assuming that I do not obtain any position in a medical college, the prestige and knowledge which I should acquire by a year's study in Germany would decidedly increase my chances of success in a large city. The young doctors who are doing well in New York are in a large proportion those who have studied abroad.[37]

Budd's opportunities and achievements mark him as a typical member of this elite, except in one small regard: unlike most of the medical leaders, he was a native New Yorker.

CHARLES INCHES AND HIS BOOK

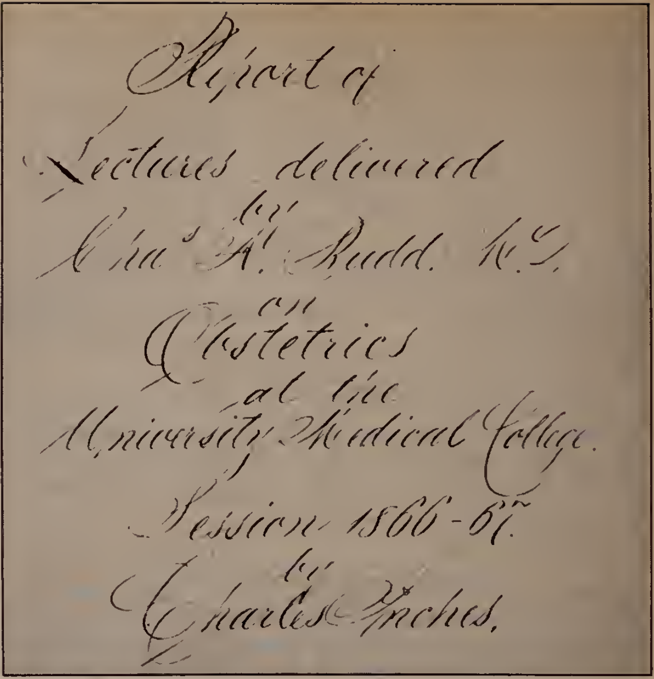
Since Budd published only a few short case reports, we would know little of his thinking but for the careful dictation of his young Canadian student Charles Inches (1846–1923). After graduating in 1868, Inches served on the staff at Bellevue Hospital. He later returned to New Brunswick (where he had been born in Fredericton), and he practiced in St. John. He also practiced for a while in Prince Edward Island, but then moved in 1880 to Scribner, Nebraska, where he settled permanently and kept up an active practice until a few years before his death in 1923.[25] He served as president of the Nebraska State Medical Association in 1891–1892; he was also a charter member of the Dodge County Medical Society, organized in 1901.[38-40]

There is hardly more evidence revealing Inches' role in this story than his name on the title page of this precious manuscript (Fig 2). None of his personal papers are known to survive. His life is recorded only in obituaries of the *Nebraska Medical Journal* and the local press; no earlier portrait than the photograph which accompanies his necrology in 1923 could be located (Fig 3). Circumstantial evidence which will be examined in the section on "The notebook and its history" even suggests that Inches probably parted with this book within a few years of its preparation, before he left New York City prior to 1872.

Although no document reveals Inches' motivations in creating this work, and while it is known that Budd's class had at least one printed textbook (see Excerpt 52), other information about the value attached to student notes in medical schools of that era suggests that Inches' fine copy may have been made for something other than personal study, review, and as a reference book to take along into practice.

OTHER STUDENT NOTEBOOKS

In the early 1860s at another of New York City's medical schools, The College of Physicians and Surgeons, there was a prize offered for the best written report by a senior student on clinical instruction. In 1861 this Harsen Prize was won by student John Shrady; his bound set of clinical notes survives today. In 1944, Butler's analysis of it was published in the *Bulletin of the History of Medicine*[41] as the essay that won the Osler Medal Contest for 1943. Whereas Shrady's handwriting is more ornate than that in Inches' book, and while the former's binding is far more sumptuous and its pages are gilt-edged, the overall resemblance between these two volumes suggests that Inches might originally have prepared his book for some similar



Report of
Lectures delivered
by
Chas A. Budd. M.D.
on
Obstetrics
at the
University Medical College.
Session 1866-67.
by
Charles Inches.

FIGURE 2. Title page (courtesy of Special Collections, The University Library, State University of New York at Binghamton).

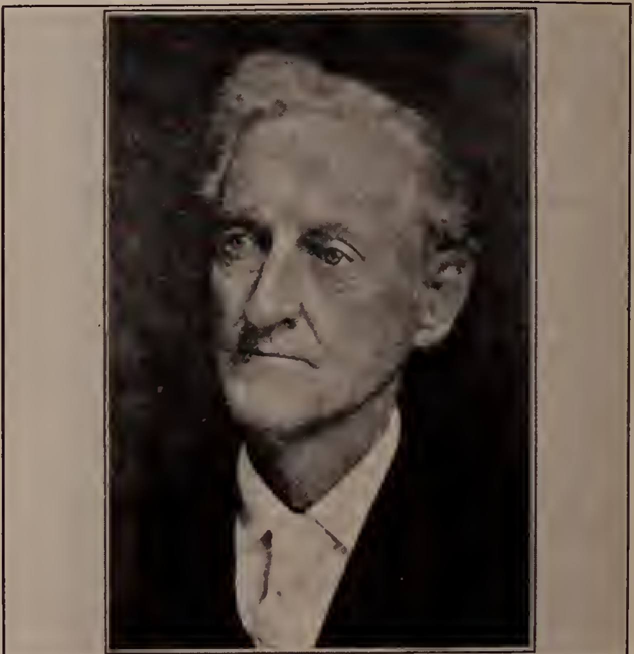


FIGURE 3. Charles Inches, MD (1846–1923). Undated photograph published in *The Nebraska State Medical Journal*, February 1923 (courtesy of the Nebraska State Medical Association).

purpose. But the crucial distinction between a record of course lectures and one of clinic cases must be kept in mind.

Clinical case reports taken down by students survive in far greater numbers than do lecture notes, probably because clinical cases were unique, being discussed by the professor spontaneously without written preparation. For these reasons, the transcript of clinical cases was more valuable both to the students and to their teachers.[42] For example, in the 1874–1875 session at New York University, new student prizes were introduced, of which the Lilienthal Prize was given to recognize the best written report of Dr Lilienthal's clinics.[43] Did the students seeking the prize receive their notebooks back after the competition, or did those notebooks—or at least the winning one—became the professor's property? When Budd's immediate predecessor, Gunning S. Bedford, published his *Clinical Lectures on the Diseases of Women and Children* from the clinic which he had opened in October 1850, the book acknowledged that these accounts had been transcribed by Doctors Woodcock, Cooper, and Gregory (perhaps while they had been students?). That in just 12 years this popular book of 653 pages was in its 7th edition and had already been translated into French and German[44] suggests the value that clinical case transcriptions had to a professor in the days before tape recordings.

William Henry Welch's student experiences in New York City in the 1870s give further indication of how valuable good note taking could be to students and others. As Welch recalled his second year of medical school (1873–1874) more than 50 years later, "I wanted a microscope more than anything else, and [Professor] Seguin offered a microscope for the best report on his clinical lectures on nervous diseases. Only a handful attended those lectures, because they came in the middle of Saturday afternoon. I regarded them as very remarkable . . . and I got that microscope by reporting those lectures." [45] Since Welch kept those notes until his death,[46] Professor Seguin probably received a transcription of them. Welch's notes on Professor Alonzo Church's lectures on the practice of medicine were regarded so highly that he was offered as much as $100 for them.[45]

Two other surviving manuscripts illustrate certain aspects of early medical instruction at New York University. For the session of 1842–1843 there survives "A report of cases attended by students of NYU Medical College and physicians of the NY Asylum for Lying-in Women." [47] One hundred five home deliveries are reported in detail, each of which was attended by one or two students from a group of nine, including Anthony T. Morris, to whom most of the writing in this manuscript is attributed. Its contents have not yet been printed or analyzed, although Heaton[48] published a very brief description of the reports.

For the following academic year, there is a student's notebook containing material from lectures by five of the school's founding faculty members during the 1843–1844 session: Professors Valentine Mott, Martyn Paine, John Revere, Granville Sharp Pattison, and Gunning Bedford. Unlike Inches' book, this one appears to be the bound notebook taken into class, rather than a later transcription. The author or owner is unknown, but may have been Lowell Holbrook, as an admission ticket for lectures of the 1848–1849 session with his signature was found in the book. Holbrook graduated in 1849. As this notebook, which is now in the Archives of the New York University School of Medicine, was acquired by purchase from a dealer in 1974, nothing is known of its history.[49]

The notes in it labelled "Midwifery" occupy only 20 pages (compared with the 366 pages of Budd's 26 lectures). The treatments of the topics are so brief that either the course was only a minimal introduction to the material, or that this student might have had to enter practice with little guidance for his first deliveries, since manual delivery, shoulder presentation, and face presentation are all covered in less than three pages.[*]

Two surviving notebooks from the same milieu, but not from NYU, highlight the typically fragmentary character of such documents, which makes Inches' book of nearly 400 pages so unusual and valuable. The first of these was written by John Van Alstyne in 1822, and the second by William T. Lusk in 1863. Van Alstyne's notes, though legible, are unfortunately sketchy or incoherent in many places, and there is no indication of what medical school, if any, Van Alstyne attended; he might simply have been apprenticed to the Dr White named in the text. A brief description of this manuscript with photographs of a few pages was published by Blinderman in this journal.[50]

For Lusk's notes, there is essential information about the professors, the courses, the date, and the school (Bellevue Hospital Medical College in 1863), but the notes themselves are incomplete.[†]

INCHES' NOTEBOOK AND ITS HISTORY

This manuscript consists of 366 pages of lined sheets (trimmed at the time of the original binding to 21 × 18 cm) bound in leather covers of blue-dyed half-calf over cardboard.[‡] The entire manuscript is done in a uniform, fine, "copperplate" handwriting, though most of the lettering is slightly less ornate than that on the title page. (Figs 4, 2).

---

[*] A listing of the topics with page numbers will easily provide a sense of the level of coverage and the subjects addressed and will also show that, although the notes are entitled "Midwifery" by the student, diseases of women and children are included in these 20 pages as well: manual delivery, p 267; shoulder presentation, 268; face presentation, 268; vaginal discharge, 268; menorrhagia, 269; polypus of womb, 270; ulceration stage of cancer, 271; cauliflower excrescence, 271; inversion of womb, 271; miscarriage, 271; watery discharges, 272; mucous discharges, 272; ascarides in rectum, 272; watery excrescences of the vestibulum, 273; bloody tumour of miatus [sic], 274; thickening of urethra, 275; carcinoma of rectum, 275; cancer of neck of womb, 275; prolapsus of womb, 277; prolapsus of bladder, 278; purulent discharge, 278; inflammation of mucous membranes of vagina, 279; amenorrhia [sic], 279; dysmenorrhia [sic], 280; puerperal fever, 280; diseases of children, 282; cholera infantium [sic], 282; purulent ophthalmia, 284; aphthous affections, 284; scarlatina, 284.

[†] Lusk divided his 400-page bound notebook into a 50-page section for each teacher period. But, for example, Prof Fordice Barker's lectures break on p 13, leaving 36 blank pages. Flint's section has 41 pages filled, but then Childs' has 46 pages blank.[51]

[‡] The front cover is gold stamped with a narrow surrounding pattern and with the title in gold letters. The binding on the spine has largely rotted away, but the pages are fresh and intact. Trimming of the edges cut away parts of some page numbers but no significant text. The front and back paste-downs and the first guard sheets (in each case, of one continuous double leaflet) are of unlined paper. There is a second sheet in the front only, also of unlined paper, glued to the first lined sheet, on which the title page is inscribed. This title page is a single, lined sheet; the rest of the book consists of stacked (not nested) double leaflets. Except for the guard sheets, the only blank page is the verso of the title page. Lecture I begins at the top of p 3 (the title being taken as p 1). Ensuing lectures begin on the same sheet as the previous one ends, except in cases in which a lecture ends at the bottom of a page.

Clearly the text was copied onto loose, folded leaflets, the title page prepared, and the pages trimmed before the leaflets were gathered, sewn, and bound. Whether written by Inches himself or by a copyist, the paper and handwriting indicate that this is not a notebook used in class, but a more leisurely copy from the live notes. That medical students might spend several hours a day transcribing their hurried notes is confirmed by the diary of Asa Fitch.[52]

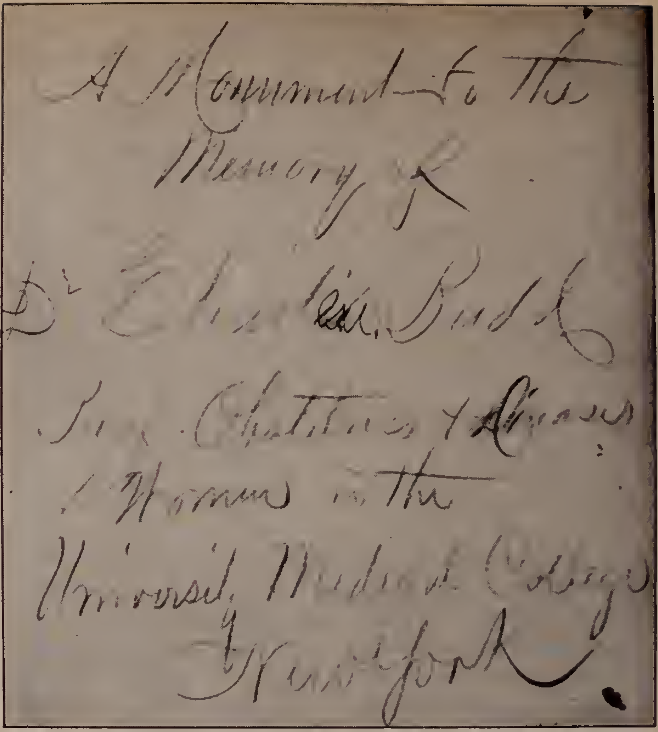
The writing is careful throughout, with few gaps or errors. Both *labor* and *labour* are used; a few words are regularly misspelled (dependant, Wedensday, seperate); and some words and names are spelled as they sound (eg, *savons* for *savants, Naegela* for *Naegele*). In the interest of clarity in the extracts published here, minor improvements in spelling and punctuation have been made (without identification), except for medical terms, which have been left in their original form.

On the first guard sheet of the book, there is an alternative version for a title page carelessly added later in green crayon (Fig 5). Does the phrase "A Monument to the Memory of" suggest that the fine copy was first made after Budd's death in 1877, or only that Inches (or a later owner) decided to try to have the previously completed book published as a memorial? The answer is not known. Knowledge about the book's earliest history is limited to what can be discerned from the text and from the codex itself.

Its later history—after a long gap—is known and must be reported, for it leads to the conjecture that Inches did not keep his book very long. In 1976, Helen Malia of Endicott, NY, "found the book again" when she sold her home. She consulted with a friend, Professor Walter Hugins of the history department at the nearby university, about giving it to the university. Hugins showed it to his colleague in history of medicine (myself), who arranged the donation to the university in memory of her late husband, John Gurnsey Malia, MD. The manuscript now resides in The Special Collections of the University Library of the State University of New York at Binghamton. In 1977, Mrs Malia answered an inquiry as follows:

> This book was among the many other books of my brother, Francis Kilbride, when he died. I understand that it was given to him by a doctor for whom he was caring as a private duty, registered nurse. The Doctor lived on Fifth Avenue in New York City and I am not certain of his name—I think it was LUTZ. He is also deceased. I am really very sorry that I am unable to give you concrete information, but all this happened many years ago and I have forgotten the details.[53]

Mrs Malia's memory may have been better than she realized, if her uncertain notion of "Lutz" is an error for "Lusk." Dr William Thomas Lusk (1838–1897) graduated from Bellevue Hospital Medical College in 1864 and returned there in 1871 to teach clinical midwifery and obstetrics and diseases of children. His own student notes from the fall of 1863 survive, but are poor and quite incomplete (see description above). Among other positions, he served as vice president of the New York Obstetrical Society in 1875 (where he would certainly have known Budd personally).[54,55] § Among the five children born to Lusk and his first wife (who died in 1871) was William Chittenden Lusk, who became a physician like his father and practiced in New York City.[54-61] This Dr Lusk lived at 47 East 34th Street (that is just off 5th Ave) from sometime before 1912[62] until his death in 1934.[63] Without specific evidence to confirm that Inches carried his book to


A Monument to the Memory of

Dr Charles A. Budd

Prof Obstetrics & Diseases of Women in the

University Medical College

New York

FIGURE 5. Sketch for an alternative title page on first guard sheet, written in green crayon, corrected in pencil (courtesy of Special Collections, The University Library, SUNY-Binghamton).

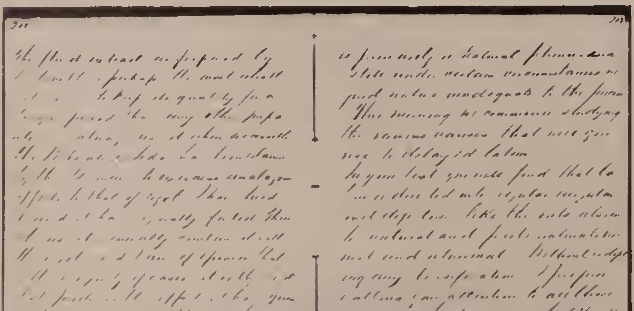


FIGURE 4. Notes on Prof Budd's lectures taken by Charles Inches, pp 212–213 (courtesy of Special Collections, The University Library, SUNY-Binghamton).

---

§ Budd and Lusk both served as physicians of the City Hospital (at that time named Charity Hospital) during the 1870s,[56,57] but whether simultaneously or successively, I am not sure. In 1882 Lusk published *The Art and Science of Midwifery*, which went through four editions in 10 years and was translated into French, Italian, Spanish, and Arabic.[54,58,59] In 1889 Lusk became president of the Bellevue Hospital Medical College, shortly before it suffered an uncomfortable merger with the Medical College of New York University.[60]

Canada and later to Nebraska, it is likely that it never left the city and was rather passed on by Inches to a colleague, perhaps to the first Dr Lusk, where it remained in his son's hands until it was given to Mr Kilbride in the early 1930s.

The Record of Budd's Lectures

The 26 lectures in the book take up an average of 14 pages each. And since each page has an average of about 115 words, the text of an average lecture runs about 1,600 words. Although the manuscript is clearly one complete volume with nothing missing, there may have been a second volume at some time. Even though nothing on the covers or title page suggests this, the course apparently continued beyond what is noted here. In Lecture XXVI on Friday, February 15th (p 365), Budd remarked, "... the time is determined by circumstances connected with those accidents upon which I shall dwell upon more in detail next week." (The awkward repetition of "upon" and the use of the phrase "more in detail" where one might expect "in more detail" are characteristic of the oral character of this verbatim transcript.) As no Budd manuscripts are known to librarians at the New York Academy of Medicine, the New York University School of Medicine, the New York Public Library, or the New York Historical Society, and as none are listed in the National Union Catalogue of Manuscript Collections, the continuation of Budd's lectures—if it was ever produced—may well be lost. (Mrs Malia is now deceased and correspondence with her surviving relatives has not turned up any record of a second volume.)

Budd's Topics

Budd used 26 lectures, recorded in 366 pages, to cover the following subjects: abortion, labor, mechanisms of labor, duties of an accoucheur, normal condition after delivery, causes of suspended foetal animation, face presentations, breech presentations, twin births, oxytocics, causes of delayed labor, prolapse of funis, shoulder presentations, version, use of forceps, and use of other instruments. (This contents list appears on the back cover of the notebook.)

My reading of Budd's lectures suggests that on technical points his views are generally in line with those published in the numerous textbooks of his era (for example, the works by Churchill,[64] Dewees,[65] Meigs,[66] and especially Ramsbotham[67] all of whom he cites). For this reason, attention here is directed primarily, but not exclusively, to his teachings on the less technical aspects of the subject, in short, to those passages that reveal the social interaction of mid-19th-century doctors with the patient, her family, and their concerns. This study therefore presents the two complete lectures on the doctor's activity in the lying-in room and 54 excerpts chosen to illustrate a variety of topics.

Budd's lectures about professional action, attitudes, and responsibilities in the birthing room make especially absorbing reading. Unlike some other parts of the course, in which the material is almost entirely concerned with the mechanics of difficult deliveries, the two lectures printed here reveal the human, and sometimes humane, aspects of the Victorian man-midwife at work dealing not only with labor and delivery, but also with his knowledge and its limits, his authority, his patients' concerned husbands, and various interferences. He is filled with warnings and advice for his students, referring several times to "young practitioners" and the mishaps to which they are especially susceptible. At many points he indicates how concerned a practitioner might be about his reputation in the community, often sneering at the parturient woman's friends also in attendance at the delivery who could criticize—or worse yet, gossip widely about—any difficulty or failure on his part. He also mentions the favorable reputation that can be secured by proper measures. The scene he sets in his lectures is always one of home birth, whether for the affluent or for the poor tenement dwellers, though in fact he and his colleagues attended numerous births in hospital settings.

If we now take ourselves back to the mornings of January 2nd and 4th of 1867, and join Charles Inches, who turned 20 years old just four weeks ago, to listen to Budd's lessons, we may not like all we hear, but we are likely to be fascinated by an extremely vivid portrayal of what must have been a common scene for parturient women in New York City just after the Civil War.

LECTURE VII. Wednesday, January 2nd [pp 89–106]

We commence studying this morning the duties of the accoucheur in the lying-in room. If I dwell rather long on this point, it is, I assure you, for your benefit, as the attention of them, one and all, are of the greatest importance to the accoucheur.

First. Always go without delay when you are called to a case of labour because in very many case[s] your presence is required from the very commencement of the parturient act; and lay little or no stress on what the messenger says with regard to the progress of the case. I advise you always, particularly those who are about to practice in the country, to take with you a lancet, female catheter, some preparation of opium and of ergot, also a small vial of chloroform.[‖]

Whether you should take your instruments is a question: some authors are in favor of doing so, while others say not, from the fact that you will be almost certainly induced to use them; and I think in the early years of your practice you are better off without them; for as I said, if the labour is slow and difficult and you are much wearied yourself, you will feel very much disposed to use them.[¶]

[‖] Budd suggests without any hesitation to use the catheter in difficult or extended labor (in Excerpt 53 below) and about 12 hours post partum if needed (near the end of Lecture VIII). For some doctors, however, the catheter was to be shunned as a violation of propriety, even if inserted under the bed covers without looking and certainly if done by exposing the patient's genitals to the doctor's glance. This controversy and the corresponding one over the use of specula and visual pelvic examinations (including statements by Budd's colleague Chandler Gilman) has been sketched by Donegan.[68] Note that Budd himself maintains the patient's "delicacy" by not looking at the genital area even when he needs to tie and cut the umbilical cord: "When you are cutting the cord be careful of what you are doing, as for the sake of delicacy you do it under the sheets; and if not careful, you are apt to cut off a finger or penis" (from Lecture VIII below). For an analysis of the public's and the profession's reactions to obstetrical anesthesia after its first use in 1847, see Duffy[69] and Cassedy.[70]

[¶] See also his advice about sending for the forceps, quoted below in Excerpt 22.

Similar advice, in more vigorous language, was offered by one British textbook republished in the United States: "I strenuously dissuade you from making familiar companions of your instruments because they are not wanted .... The very fact that an accoucheur, on all occasions, puts the lever into his pocket when he goes to attend a labor, proves that he is an officious, a meddlesome, and therefore, to my

On your arrival don't rush into the apartments, but have your arrival announced to the patient. Perhaps she is with her first child, sensitive and retiring in her disposition; and may never have seen you before. Conduct yourself gently and with dignity.

Another point, and I assure you these minutiae will be of the utmost importance to you, never enter the chamber with a long face, nor roll up your sleeves before you have notified her of an examination. Sit down alongside of her and attempt cheerfully to engage her in some general conversation and you will easily gain her confidence.

In this manner you notice the frequency, duration, strength, and character of the pain. If you are anxious to know more about the case, inquire of the nurse, [but] not by a whisper in the lady's presence.# You want to know: How long they last? Are they severe? Is there any show? If, when you are conversing with the patient, she suddenly puts on a wry face, feel her pulse and abdomen, whether it is firm or not.

By all this you are to form your conduct: if the pains are only trivial, you may retire into an adjoining room, if they are frequent and active, no time is to be lost in making a vaginal examination.

Dr. Dewees [William Potts Dewees, 1768–1841[74]] says an examination should never be made before your patient is announced of it.** If you say to her yourself that it is necessary to make an examination, that you will not hurt her etc. etc., or [you] take the nurse to a corner of the room and begin to tell her about it in the patient's presence, you excite and altogether disturb her. The easiest and by far most satisfactory method is while you are feeling her pulse and with her mind directed, to gently slip your hand down before she knows anything about it, you having had a napkin laid alongside of you.

Now what do you want to find when your finger is in? 1st, whether she is pregnant or not. Cases are by no means wanting, to demonstrate the need of this. Women thinking they are pregnant and labour coming on will send for their physician. In a case of this kind, where perhaps there is an ovarian tumour, unless you immediately reveal the truth, you will be the subject of not a little ridicule.

2nd, whether she is really in labour or not. We have already studied the signs of labour. Bear in mind an open os signifies nothing, as it may be so for a day or two before labour, but if in the interval it is soft and open and on the renewal of the pain it becomes rigid, then it is very significant.

The next thing is with reference to the condition of the soft parts whether they be patulous, dilated, and well lubricated, or undilated and tense; you should also feel if there is any tumour or malformation of the pelvis, also the situation of the orifice, obliquity of body and neck of the womb and the child's presenting part. The membranes are never to be ruptured for the sole purpose of facilitating an examination.

I generally select the time when a pain is just commencing to make an examination, and I do it as slowly and deliberately as possible. By a little experience you will satisfy yourself on all these points with very little motion.

Your opinions will be often sought for as to the time labour will occupy, and how soon she will be well. Never commit yourself on this point. I am always ready with an answer. "Doctor? Will I soon be well?" "I hope so!" "Doctor. How long will it be before I am well?" "That depends on circumstances. If the pains are regular etc., I am in hopes that such and such a time will do." If you carelessly name a time at the commencement of labour which you think will carry it through, hardly a moment will pass that period before the patient gets peevish and thinks something dreadful is going to happen. Therefore never say anything as positive and you will not incur any discredit.

There are certain circumstances by which you may estimate to yourself the duration of the labour. If you are called to a woman in labour with her first child and find the soft parts tense and rigid, the head just within reach, and so on, the probabilities are she will have a long labour. But if the pains are sharp and short, she will probably get through quicker than if they were long and easy. The cervix may be dilated in part and at the same time be undilatable. If you find a dilatable os, which you can determine by your finger in the early stages, it will in conjunction with some other points enable you to form a pretty good opinion. If the upper parts are soft and vulva, vagina, and perineum are hard and tense, the first stage and part of the second will be easy and the later parts difficult and extremely painful. The degree of lubricity, dilatability, and character of the pains tell a great deal.

Should you remain at the house or go away for a period? This is a point which requires a good deal of experience to judge aught. I would advise you at least in your early years of practice to remain throughout the entire labour. This will also go well towards your fee. Suppose you go away intending to be back in time and when you come find the child born. The nurse will make some comments, not I assure you in any degree complimentary.

Now these old nurses with side curls and carpet slippers, that are all terrible chatterboxes, have an antipathy to young practitioners and will not hesitate in saying any-

---

mind, a bad accoucheur. . . . 'Lead yourselves not into temptation'; if you put your instruments into your pocket, they are very apt to slip out of your pocket into the uterus. Patience and good nature, are two very useful obstetric instruments which may be fearlessly carried to every labor." [71]

Here and elsewhere, Budd acknowledges that instrumental interference is sometimes necessary, while he strongly reproves undue use of medications or instruments simply to hurry a delivery. (See also Lecture VIII and Extracts 21, 22, 25, 30, 33, 34, 36, 44–46, 51, and 53 below.) If Budd's attitudes should prove to be widespread among physicians, it may be necessary to reconsider some of the historical generalizations about men in midwifery being more regularly interventionist than their female counterparts. With respect to the activities of male and female physicians, Morantz and Zschoche have commented: "In some cases historians have merely assumed the accuracy of nineteenth-century statements when critics of male midwifery insisted that women were more willing to 'wait on nature' . . . . Occasionally even physicians deplored the enthusiasm of their colleagues for instruments. G. S. Bedford . . . warned his class not to take part in the 'indiscriminate and unpardonable use of instruments' which he believed 'generally prevailed.' " [72] In their study, Morantz and Zschoche review the 19-century debate on conservative versus activist (or "medicalized") obstetrical styles and also examine the actual records of delivery for two Boston hospitals in the 1880s and 1890s, one staffed by women doctors and one staffed by men. They find no significant statistical difference between the two hospitals in the interventions (anesthetics, forceps, or both). They note further that forceps deliveries increased in both hospitals toward the end of the century, showing that the professionalism of the practitioners, rather than their gender, may be the critical factor determining attitudes toward intervention in childbirth.[73]

# Advice not to whisper in the woman's presence is given again below and in Excerpt 35.

** I have not found this advice in Dewees (1832)[65]; perhaps Budd was using a different edition.

thing that will be detrimental to your standing. Always make your word law. Don't think of arguing with a nurse, but let her know her position from the very onset. Sometimes a kitchen servant, or friend, will be all the assistance and will enquire of you how to make the bed.[††]

In the country a straw mattress is chosen for the hips and a feather one for the back and head. In the city, an oil cloth coverlid is spread on the mattress, and several sheets over the oil cloth, which can be removed after labour, and she can have a dry bed to lay on. Some have a supplementary bed, to which the patient is transferred immediately after labour.

If you have anything to say, never recommend a feather bed. Some fourteen years ago, I was called to a woman and found labour just coming on. I ordered a dose of castor oil and sometime after took my seat by the bed which happened to be made with a feather mattress and was supporting the perineum with my hand and arm which made a true gutter from the woman's genitals down the bedside. The castor oil began to work just at a moment when I could not move, and the consequence was I received the contents of the rectum all over my clothes which were a new light summer suit.

Now as to the position of the patient. In Great Britain it is customary to place her on her left side, and sitting on the right side of the bed manipulate with the right hand. On this continent she is confined on her back and you may sit on either side of the bed. Among the common Irish, they will frequently get on their knees and elbows.[‡‡]

But this is altogether superfluous. If she prefers to be on her back, let her, and if on her side let her. Accustom yourselves to use either hand. I think it well in the early stages to walk about the room a little. If she won't walk, keep her off the recumbent position as long as possible. Generally by a little coaxing she will comply with your wishes.

With reference to the number of persons admitted into the lying-in room. There are generally a number of old women who through friendship tender their services and force themselves into the apartment. They do no good but greatly discommode you by their continual chattering and interference. They will also circulate all through the place how you conducted yourself etc. Now do not allow this to be the case, at any rate let no person stay whose presence is unpleasant to the patient. Especially would I say to you students who have your first cases in this city, when you go to a tenement house and find ten or fifteen women around the room, hustle them all out.

A certain doctor of Dublin, who is the author of a small book, pretty much on this point says that if the husband is a sensible man, he will not want to be in his wife's chamber, and if he is not sensible, you don't want him there.[§§]

Now I generally consult the wishes of the patient herself. If she is not anxious that he should be present, delicately put him out. Sometimes she will want her husband's hand to cling to and it is one of the greatest consolations to have him near her. Of course under such circumstances you would not think of dismissing him.[‖‖] If he is determined to stay when not wanted, send him three or four miles away for something you don't want. Get him out of the way at least.

In reality the active duties of the accoucher do not commence until the 2nd stage. Before that time they may be called only passive. If you are detained in the house for six or eight hours before the 2nd stage, see that the bladder and rectum be relieved.

As to the frequency of your examinations. Some say to make them once an hour. Now I [do not] look upon meddlesome midwifery as disgusting. It is not unfrequent to see a doctor make an examination, go out of the room and, [sitting back] with his heels higher than his head, smoke and spit. This is shilly shallying midwifery.[¶¶]

I advise you in your early years of practice to make frequent examinations; it is not only of great value to yourself but it impresses your patient with the idea that you are doing her some comfort. Do them gently and ascertain all you can. A delicate sense of touch is a great point in an obstetrician and is only to be gained by experience.

LECTURE VIII. Friday, January 4th, 1867. [pp 106–121]

I had finished in my last lecture the duties of the accoucheur prior to the 2nd stage and said that his active duties do not commence, in fact, before the head begins to descend.

These duties consist first in the support of the perineum. The chief [points] to be observed in accomplishing this are first the proper time. Young practitioners will commence to do this as soon as the head is near the perineum; but there is no need of it being done before the perineum is thoroughly distended; some authorities are in the habit of instructing their pupils that this supporting the perineum is all nonsense. But as a matter of comfort alone and of humanity, it is exceedingly proper. The head has now lost the support of the bony walls of the pelvis and it is now that the patient suffers the most agonizing pain.##

[††] Budd's antagonism towards the woman's female friends is expressed again several paragraphs below and in Excerpt 48. The commonplace character of his opinion is suggested by these remarks of a French doctor published in 1862: "Tell the crones who customarily occupy the bedchamber to leave. They befoul the air, make your client uneasy, torment her, and will get in your way during your maneuvers, only to criticize you afterward if you are unsuccessful." [75]

[‡‡] Note that this last position is specifically recommended by Budd to relieve cases of prolapsed cord; see Excerpt 42 below, where it is credited to the ingenuity of Dr T. Gaillard Thomas, not to the "common Irish." It seems likely that Budd got this idea from Ramsbotham, who wrote, "the peasantry of Ireland place themselves on their hands and knees." [76]

[§§] Clearly a reference to Henry Maunsell's book, *The Dublin Practice of Midwifery With Notes and Additions by Chandler R. Gilman*, where we read on p 122: "Never allow the husband to be present. If he is a man of sense and delicacy, he will not desire it, and if he is not, the more reason you should keep him out of your way." [77]

[‖‖] Suitor's research indicates that Budd's acceptance of the wife's preference on this is fairly typical for accoucheurs of that era; her study also claims that the presence of husbands in the birthing room was a new development of the 19th century.[12]

[¶¶] Note also Budd's published comments about one of his cases (where he was called in after the head "had become impeded at the inferior strait . . . for sixteen hours prior to my seeing her"): "Another practitioner had been called in some hours previously, who advised opium and patience. I found the woman much exhausted, the vagina and vulva dry, hot and sensitive, the abdomen tender to the touch, and the pulse rapid. . . . I subjected her to the influence of chloroform, and applied the forceps, delivering her of a son, which bore evidences of having been dead some time. The cord was around the neck. . . . I cite this case as an exemplification of Dr. Miller's recent aphorism: 'A meddlesome midwifery is bad, but a shilly-shally midwifery is worse.' " [78] It may not prove possible to identify this Dr Miller with certainty, but a likely candidate is Henry Miller, 1800–1874, professor of diseases of women at the Louisville Medical College from 1837 to 1858, who published *Theoretical and Practical Treatise on Human Parturition* in 1844, which was revised as *Principles and Practice of Obstetrics* in 1858. In 1859 he was elected President of the American Medical Association.[79,80] Budd reviewed the 1858 edition at length in the *American Medical Monthly*, concluding that "we feel confident [it] will prove a highly valuable addition to the library of any practitioner as a work of reference. As a text-book, however, for the student, we regard it as too meagre in detail, and not sufficiently explicit on many important point." [81]

Although a well supported perineum will occasionally rupture, a great deal of pain has been relieved and the passage of the head assisted by continuing the concavity formed by the sacrum and floor of the perineum. Now like everything else this may be done in a right and wrong way. If improperly, you cause more pain and retard the advancement of the head. Take a towel in your hand and with the radial border of your hand at the posterior commissure of the vulva in such a way that the curve is continued, make firm, equal pressure. The object of the towel is twofold: it is more cleanly and is more delicate.

The first thing to be done as the head is being disengaged is to pass up your finger along the neck of the child to ascertain if the cord is around it. Then put your other hand on the abdomen over the fundus uteri and make firm steady pressure. You want to follow the uterus down as it contracts and this is to be kept up until the placenta is removed and then apply your binder. When you feel the cord around the neck, put your finger under it and slip it over the head; if, as is sometimes the case, the parts are too tight in consequence of the contractions or from delivery being too quick, the child is born with a loop around the neck.

The next thing that is not to be done, and I wish you to bear this in mind as it is very important though apparently insignificant, is never make traction on the shoulders. There is a woman in labour 24 hours. Everyone in waiting on her is tired. After the head is delivered, you draw a long sigh of relief and with visions of good things awaiting you at home, you help the labour by pulling on the shoulders and making rapid delivery. In such a case there is strong liability of there being severe haemorrhage as the uterus is left in an uncontracted state. I constantly hear of cases of haemorrhage where it is necessary to introduce the hand and remove the placenta. This is nearly always dependent on neglect of pressure over the fundus or pulling on the shoulders.

If the uterus is sluggish and after a few minutes you notice the child's head becoming black, wipe out the mouth and blow on its face. When there is danger of asphyxia to the child, it is necessary to make some gentle traction on the child, but be sure to redouble your pressure over the fundus.

The shoulders delivered, there will be another suspension in the pain and again you might easily pull out the remaining part of the child, but be sure and do not, as in a few moments contraction again comes on and the body is delivered.

The child is now lying in the bed, kicking and squalling; do not yet take away your pressure on the fundus. If it is the nurse to whom you have entrusted that duty and she takes her hands away, standing gaping at you—blow her up. Be strict on this point. You are next called upon to separate the child from the mother. As to the material used for this purpose, I generally use a narrow tape. I find it best in many respects. Have your ligatures and scissors (which had better be blunt pointed) convenient to you. Some say never cut the cord so long as it pulsates, but there is no necessity for this. You may do it as soon as the child cries. Apply the ligature about two inches from the umbilicus, and tie it in a double knot. As to the propriety of two ligatures, I am decided, Cazeaux to the contrary, that one will suffice where there is not plural birth. The placenta is thereby unloaded of a great deal of blood and will come away much easier.***

When you are cutting the cord be careful of what you are doing, as for the sake of delicacy you do it under the sheets; and if not careful, you are apt to cut off a finger or penis.

The manner of handling the child is also important. Put one hand under the child's neck and the other under the feet with a finger between the two and you can easily and safely hand it to the nurse; otherwise it will be apt to slip out of your hands, not a very desirable object.

Some nurses will lay the child in an easy chair and hurry back to your assistance, and some Mrs. Jones or Smith who weighs not more than 500 will come in and sit down—in the easy chair! See that this neglect of the nurse never occurs.

The delivery of the placenta is your next duty, having satisfied yourself that there is not another child. I never wait more than five minutes. I have seen a case where the placenta was 36 hours in the vagina and three physicians in consultation waiting for its expulsion and afraid to touch the cord. Run your finger up along the cord and feel its attachment to the placenta. Hold the cord in the other hand and make gentle traction. You must bear in mind the diameters of the pelvis in this operation; when it arrives at the vulva, you take it and the membranes in your hand and rotate them two or three times, which will gather them all up into a lump. By this method they will come away entire and you are not liable to leave shreds of the membranes behind, which often cause after-pains. The placenta is to be delivered and put into the chamber underneath the bed clothes. Never be seen carrying the after-birth in your hand around the room looking for the chamber.

About the time you commence supporting the perineum you wrap a towel around your arm and have the nurse pin it tightly and safely. I generally use a pair of oil silk sleeves having elastic bands at each end; they are efficient and easily carried.

The next thing to be seen to is the application of the binder. Some of our modern savons [*viz.* savants] tell us that a binder is unnecessary. Now I think there would be reason enough if there was nothing else but the general symmetry of the female to be affected by it. It would not

---

With this practice, as with others cited above and below, Budd and his contemporary doctors might seem to merit the hostility Shorter has directed at traditional midwives, who, he claims, "intervened furiously in the natural process of birth. Constantly tugging and hauling at the mother's birth canal, at the infant's head, and at the placenta, they were captives of a folkloric view that the best midwife is the one who interferes most. Lest the reader be in doubt, massaging the area between the thighs, dilating the vagina, or attempting with one's hands to 'steer' the infant's head, are all pernicious, useless practices."[82] Readers will have to weigh for themselves the balance between Budd's 19th-century practical experience of "comfort alone and of humanity" and Shorter's 20th-century impatience with "useless practices."

*** Pierre Cazeaux, 1808–1862, published his major work in 1840, which appeared in English translation as *A Theoretical and Practical Treatise on Midwifery, Including the Diseases of Pregnancy and Parturition* in 1850 with further editions in 1857, 1863, 1866, 1868, 1875, and 1881. This was used as a textbook in Budd's course (see Excerpt 52).

be pleasant for any woman, now-a-days at least, to have her abdomen all wrinkled and flabby. The best material you can have for this purpose is a straight broad piece of muslin; sufficiently so to extend from over the ribs down over the trochanters. The object of having it so broad is to prevent its slipping out of place. If this can not be had, call for a long towel or pillow case. You roll it up in front and grasp the rest all in one hand and slip it under her hips stretching it out its whole breadth. Then tighten it up and pin from above downwards. Sometimes it is necessary to apply a compress over the fundus; a folded napkin assumes this purpose well. After the binding is completed call for a napkin, fold and warm it and apply it against the vulva.

We have now to deal with the child and first as to the washing of it. One thing I omitted: don't be too anxious about announcing the sex of the child. If the mother has been anxious for a girl and perhaps even allowed herself to become pregnant on that account, don't announce the fact too suddenly that it is a boy; if a girl, you may.

Now soap and water will not satisfactorily remove this ceruminous substance from the child. First smear it all over with a little lard; and then use your soap and water. Don't allow the nurse to rub the soap into the child's eyes, as I am satisfied this is not an unfrequent cause of opthalmia [sic] neonatorum. The best way to avoid this is not to allow any soap to go near the face at all.

Then take a piece of linen a few inches square, fold it and perforate; lay this on the child's abdomen and dress the navel through it and let it lay to the left side. Don't lay it on the right side anyway, for experience is against it. Some writer has explained this as acting as a compress on the biliary duct. Some sense may be in it. Then have a band two or three inches broad and lay enough to go around twice.

Never leave the house within an hour of the delivery of the child. This is a fundamental rule in the British school of midwifery. You make your first visit about twelve hours afterwards, or as near that as convenient. You then enquire if she has urinated, and if not, pass the catheter, which you should be sure and have; you also enquire if the child has made water or passed any meconium.[†††]

If the child is a male and is crying hard, examine the hypogastric region, and most likely you will find its bladder distended and in the prepuce you [will] find a little plug made of the ceruminous material in delivery. You can easily remove this by a probe.

Perhaps a few hours after labour, you will be called to see the child, which is in a constant fit of crying; in all probability you will find this dependent on a pin sticking in its body.

Acknowledgments. The author is especially grateful to Ms Marion Hanscom, Head of Special Collections at the University Library of SUNY-Binghamton, for invaluable assistance. The photographs are published here with the kind permission of the Library of the State University of New York at Binghamton, the Nebraska State Medical Association, and the New York Academy of Medicine Library. Several other institutions made their resources available to me in the course of this research: The College of Physicians of Philadelphia; the Health Sciences Library of Columbia University; The Dodge County (Nebraska) Historical Society; The Institute for Advanced Study; New York University Medical Center Library and Archives; Princeton University; and the library of the Research Laboratories of the E. R. Squibb Corporation.

For assistance in my research I wish to thank Van B. Afes of the Archives of the New York University Medical Center, Janet Brady Berk of the Miner Library of the School of Medicine and Dentistry of the University of Rochester, George Chauncey of Yale University, Gilbert J. Clausman of the Library of the New York University Medical Center, Anne P. Diffendal of the Nebraska State Historical Society, Inge Dupont of the New York Academy of Medicine Library, W. B. Eaton of the Dodge County (Nebraska) Medical Society, Janet Golden of the Wood Institute for the History of Medicine, Janet Kubinec of the Health Sciences Library of Columbia University, Sandy Lafferty of the Institute for Advanced Study, Susan Lawrence of Ball State University, Kenneth E. Neff and William L. Schellpeper of the Nebraska State Medical Association, Robert Sink of the New York Public Library, Gertrude Swanson of the Eastern Nebraska Genealogical Society at Fremont, Peg Van Sant of the Institute of Advanced Study, Nona J. Wiese of the Musbach Museum of Scribner, Daniel Williman of the State University of New York at Binghamton, and Helen Yam of the University of Nebraska Medical Center.

REFERENCES

1. Atwater EC: "Making fewer mistakes": a history of students and patients. *Bull Hist Med* 1983; 57:165–167.
2. Bogdan J: Care or cure? Childbirth practices in nineteenth century America. *Feminist Studies* 1978; 4 (June):92–99.
3. Donegan JB: *Women and Men Midwives: Medicine, Morality, and Misogyny in Early America.* Westport, Greenwood Press, 1978.
4. Drachman VG: The Loomis trial: social mores and obstetrics in the mid-nineteenth century, in Reverby S, Rosner D, (eds): *Health Care in America: Essays in Social History.* Philadelphia, Temple University Press, 1979, pp 67–83.
5. Ehrenreich B, English D: *For Her Own Good: 150 Years of the Experts' Advice to Women.* Garden City, Anchor Doubleday, 1979.
6. Leavitt JW: "Science" enters the birthing room: obstetrics in America since the eighteenth century. *J Amer Hist* 1983; 70:281–304.
7. Leavitt JW: Under the shadow of maternity: childbirth and death fears in nineteenth-century America. *Feminist Studies* (in press).
8. Litoff JB: *American Midwives, 1860 to the Present.* Westport, Greenwood Press, 1978.
9. Longo LD: Obstetrics and gynecology, in Numbers RL (ed): *The Education of American Physicians: Historical Essays.* Berkeley, University of California Press, 1980, pp 205–225.
10. Morantz RM, Zschoche S: Professionalism, feminism, and gender roles: a comparative study of nineteenth-century medical therapeutics. *J Amer Hist* 1980; 67:568–588.
11. Quiroga VA: Poor Mothers and Babies: A Social History of Childbirth and Child-Care Institutions in Nineteenth-Century New York City. PhD dissertation, Department of History, State University of New York at Stony Brook, 1984.
12. Suitor JJ: Husbands' participation in childbirth: a nineteenth-century phenomenon. *J Family History* 1981; 6:278–293.
13. Wertz RW, Wertz DC: *Lying-In: A History of Childbirth in America.* New York, The Free Press, 1977.
14. Shorter E: *A History of Women's Bodies.* New York, Basic Books, 1982.
15. Ricci JV: A glance into the history of the city hospital and into the lives of its gynaecologists and obstetricians. *Contributions from the Department of Gynaecology of the City Hospital New York [Blackwell's Island, later Welfare Island]* 1945; 1:23.
16. Obituary for Charles Arms [sic] Budd. *The Medical Record* 1877; 12:335.
17. Rosenberg C: The practice of medicine in New York a century ago. *Bull Hist Med* 1967; 41:223–253.
18. Vogel MJ: *The Invention of the Modern Hospital.* Chicago, University of Chicago Press, 1980, p 17.
19. Op cit, Atwater EC, pp 172–173.
20. Op cit, Rosenberg C, pp 225, 227, 228, 238, 252.
21. Walsh JJ: *History of Medicine in New York: Three Centuries of Medical Progress.* New York, National Americana Society, 1919; 2:458–459.
22. Op cit, Litoff JB, pp 20–21.
23. Atkinson WB: *A Biographical Dictionary of Contemporary American Physicians and Surgeons.* 2nd ed. Philadelphia, Brinton, 1880, p 127.
24. *The Medical Register of New York, New Jersey, and Connecticut for the Year Commencing June 15, 1877.* New York, GP Putnam's Sons, 1877, pp 199–200.
25. Obituary for Dr. Charles Inches. *The Scribner Rustler,* January 18, 1923, p 1.
26. Dupont I: Letter to the author from Rare Book Room of the New York Academy of Medicine Library, December 4, 1984.

[†††] Regarding catheterization, see the second paragraph of Lecture VII above and Excerpts 38 and 53.

27. Budd CA: A case of labor in which the foetus presented by the nucha and back of the head—demanding craniotomy. *Amer Med Monthly*, July 1858; 10:36-37.

28. Budd CA: Case of scirrhus of the cervix uteri, cured by the application of actual cautery. *The NY Med Times* June 1856; 5:300-302.

29. Budd CA: A case of unusually large development of the human foetus. *Amer Med Monthly*, March 1858; 9:192-193.

30. Budd CA: Curative treatment of prolapsus uteri, by the local application of tannin. *The NY Med Times*, February 1856; 5:156-158.

31. Budd CA: Select cases of midwifery. *Amer Med Monthly*, January 1859; 11:16-24.

32. Budd CA: Case of double footling presentation, reported at a meeting of the New York Obstetrical Society summarized in the *Amer J Obstet* 1875; 7:148.

33. Davis EH: *A Short Sketch of the New York Medical College with its Charter and a Complete List of its Officers and Graduates.* 1883, p 15.

34. Eliot WG: *Portraits of the Noted Physicians of New York, 1750-1900, with a Few Facts of the Life of Each.* New York, American Biographical Society, 1901, p 105.

35. Cutter IS, Viets HR: *A Short History of Midwifery.* Philadelphia and London, WB Saunders Company, 1964, pp 166-167.

36. *University of New York, Medical Department: Annual Announcement of Lectures, Session 1866-67.* New York, Russell's American Steam Printing House, 1866, p 8.

37. Flexner S, Flexner JT: *William Henry Welch and the Heroic Age of American Medicine.* New York, Dover Publications, 1966 (corrected reprint of 1941 ed), p 75.

38. Diffendal AP: Letter to the author from Nebraska State Historical Society, July 7, 1977.

39. Tyler AF (ed), Auerbach EF (comp): *History of Medicine in Nebraska.* Omaha, University of Nebraska Medical Center, 1977, pp 321, 423.

40. Necrology for Dr. Charles Inches. *The Nebraska State Med J* 1923; 8:74.

41. Butler BC: John Shrady's "Harsen Prize." *Bull Hist Med* 1944;16:131-153.

42. Lawrence S: Personal communication, December 1984, based on dissertation research in British medical archives.

43. Op cit, Walsh JJ, 2:541.

44. Bedford GS: *Clinical Lectures on the Diseases of Women and Children,* 7th rev ed. New York, William Wood, 1862.

45. Op cit, Flexner S, Flexner JT, pp 63-64.

46. Ibid, p 472.

47. Morris AT: *[Midwifery:] A Report of Cases Attended by Students of N.Y.U. Medical College and Physicians of the N.Y. Asylum for Lying-in Women.* New York, 1842-43. Manuscript Collection of the New York Academy of Medicine Library. Notebook of 71 pages.

48. Heaton CL: Early obstetrical teaching [in 1842-1843 in NYC]. *NYU Med Q* 1954; 10(2):12.

49. *Lectures by Professors Mott, Paine, Revere, Pattison, and Bedford: Session 1843 and 44 of New York University.* New York, 1843-1844. Archives of the New York University School of Medicine. Manuscript notebook possibly written by Lowell Holbrook (MD 1849).

50. Blinderman A: John Van Alstyne, his book: a medical student's lecture notes in 1822. *NY State J Med* 1975; 75:2577-2580.

51. Lusk WT: *Notes Taken at Bellevue Hospital Medical College in 1863 from Lectures of Fordyce Barker, Austin Flint Sr., Timothy Childs, Austin Flint Jr., George T. Elliot, Frank H. Hamilton, and Benjamin McCready.* New York, 1863. Manuscript Collection of the New York Academy of Medicine Library. Ruled notebook written in ink on 74 pages.

52. Rezneck S: A course of medical education in New York City in 1828-29: the journal of Asa Fitch. *Bull Hist Med* 1968; 42:559-560.

53. Malia HK: Letter to the author, August 5, 1977.

54. Smith AA: Address in memory of William Thompson Lusk, M.D., LL.D, in Lusk WT: *War Letters . . . 1861-1863.* New York, privately printed, 1911, pp 13-20.

55. Op cit, Walsh JJ, 4:28-29.

56. Op cit, Ricci JV, p 38.

57. Brown JH (ed): *Lamb's Biographical Dictionary of the United States.* Boston, James H. Lamb Co, 1900; 1:481.

58. Op cit, Ricci JV, pp 39-41.

59. Heaton CL: Obstetrics at the New York Almshouse and at Bellevue Hospital. *Bull NY Acad Med* 1940; 16:45-47.

60. Op cit, Walsh JJ, 2:481-482, 465-467, 486.

61. Ibid, 402-403.

62. Letter of presentation signed and dated 47 East 34 Street, January 23, 1912, by William C. Lusk, tipped into Princeton University Library copy of Lusk WT: *War Letters . . . 1861-1863.* New York, privately printed, 1911.

63. *Medical Directory of New York, New Jersey, and Connecticut,* Medical Society of the State of New York, Volumes 36 (1934) and 37 (1935-1936).

64. Churchill F: *On the Theory and Practice of Midwifery,* 3rd Amer. ed. Philadelphia, Lea and Blanchard, 1848.

65. Dewees WP: *A Compendious System of Midwifery,* 1st ed. Philadelphia, Carey and Lea, 1832.

66. Meigs CD: *Obstetrics: The Science and Art.* Philadelphia, Lea and Blanchard, 1849, 1852, 1856, 1863, 1867.

67. Ramsbotham FH: *The Principles and Practice of Obstetric Medicine and Surgery in Reference to the Process of Parturition Illustrated with 148 Figures.* 6th American ed. Philadelphia, Blanchard and Lea, 1851.

68. Op cit, Donegan JB, pp 155-157.

69. Duffy J: Anglo-American Reaction to Obstetrical Anesthesia. *Bull Hist Med* 1964: 38:32-44.

70. Cassedy JH: *American Medicine and Statistical Thinking, 1800-1860.* Cambridge, Harvard University Press, 1984, pp 89-90.

71. Blundell J: *The Principles and Practice of Obstetricity* (London, 1834) as quoted in Donegan JB: *Women and Men Midwives: Medicine, Morality, and Misogyny in Early America.* Westport, Greenwood Press, 1978, p 187.

72. Op cit, Morantz RM, Zschoche S, pp 570-571.

73. Ibid, pp 580-581.

74. Cutter IS, Viets HR: *A Short History of Midwifery.* Philadelphia and London, WB Saunders Company, 1964, pp 156-157, 212-213.

75. Munaret JM: *Le médicin des villes et des campagnes* (Paris, 1862) as quoted in English translation in Shorter E, *A History of Women's Bodies.* New York, Basic Books, 1982, p 54.

76. Op cit, Ramsbotham FH, p 113.

77. Maunsell H: *The Dublin Practice of Midwifery, With Notes and Additions by Chandler R. Gilman* (New York, William A. LeBlanc, 1842) as quoted in Suitor JJ, Husbands' Participation in Childbirth. *J Family History* 1981; 6:284.

78. Op cit, Budd CA, 1859, p 20.

79. Op cit, Cutter IS, Viets HR, pp 168-169.

80. Kelly HA, Burrage WL: *Dictionary of American Medical Biography.* New York and London, D. Appleton and Company, 1928, pp 844-945.

81. Budd CA: Review of *The Principles and Practice of Obstetrics* by Henry Miller. *Amer Med Monthly* May 1858; 9:363-366.

82. Op cit, Shorter E, p 59.

ESSAYS IN PEDIATRICS

The "Essays in Pediatrics" series includes articles that focus on clinical management of the diseases of children and adolescents and discusses recent advances in basic science, as they relate to the needs of the practitioner. The series emphasizes changing trends, and current controversies in diagnosis and treatment.

Proposals for manuscripts for "Essays in Pediatrics" should be submitted to Carl Pochedly, MD, Director of Academic Research, Children's Hospital Medical Center, 910 Blackford St, Chattanooga, TN 37403.

Management of idiopathic thrombocytopenic purpura in childhood

JAMES B. BUSSEL, MD

Idiopathic thrombocytopenic purpura (ITP) is a disease which, when it occurs in children, engenders much debate about whom to treat, when to treat, and what treatment is appropriate. This article reviews the natural history and pathophysiology of ITP, as well as treatment choices, with particular attention to areas of controversy.

ITP of childhood is a relatively benign disease.[1] Whereas the disease is often diagnosed because of extensive hemorrhagic symptoms with platelet counts less than 20,000/mm^3, even as low as 10,000/mm^3, less than 1% of patients with ITP will experience a serious hemorrhage, such as an intracranial hemorrhage.[2] Moreover, even after a hemorrhage involving the central nervous system, some patients will survive without serious neurologic sequelae.

During the six to 12 month period following the diagnosis of ITP, approximately 85% of children with this disorder will recover fully or improve sufficiently so as not to require further treatment.[1] The chance of spontaneous recovery decreases after six months from the time of diagnosis, but spontaneous recovery is always possible, and the long-term outlook is considered to be good despite insufficient data from longitudinal follow-up studies.[3]

Thrombocytopenia occurs in childhood ITP through rapid destruction of platelets by antiplatelet antibodies. The most direct evidence for this concept is derived from studies in which plasma from adults with ITP was infused into healthy volunteers who then became markedly thrombocytopenic.[4] Similar evidence comes from babies of mothers with ITP who develop transient neonatal thrombocytopenia via transplacental passage of antiplatelet antibody.[5] The factor that causes thrombocytopenia has been shown to be immunoglobulin G (IgG). Splenic lymphocytes from patients with ITP have been shown to synthesize antiplatelet antibody,[6,7] and platelet-associated immunoglobulins have been demonstrated in more than 90% of patients with ITP.[8]

Therapeutic interventions have been aimed at the antiplatelet antibody factor and include the following: inhibiting the ability of the antibody to opsonize platelets by interfering with the process of phagocytosis; dissociating the antiplatelet antibody from the platelet or preventing its binding to the platelet; and inhibiting the production of the antiplatelet antibody. No therapy is known to have an effect on bone marrow production of platelets, which is a poorly understood area in both the pathophysiology and treatment of ITP.

ACUTE ITP

The initial therapeutic dilemma in caring for a patient with ITP involves the decision of whether to treat the patient with acute ITP at the time of presentation. Based on early reports of intracranial hemorrhage in ITP which suggested that such an event tended to occur in the first month of disease,[9] treatment was often instituted for three to six weeks and then stopped regardless of whether the platelet count improved. This regimen indulged the desire of the physician to treat and the desire of the patient (and his or her parents) to be treated. Other physicians elected not to treat patients with acute ITP for three reasons: the generally good outcome associated with the disease; the ability of prednisone therapy to prevent central nervous system hemorrhage has never been established; and the incidence of central nervous system hemorrhage was thought to be lower than 1% (because of over reporting of incidence of this complication by major academic centers).

CHRONIC ITP

Regardless of whether immediate treatment was given, if the disorder did not spontaneously remit, the patient's clinical status would likely be similar two or three months after diagnosis. In patients with persistent disease, a similar dilemma occurs. Physicians who choose not to treat ITP at this point, regardless of whether they treated initially, do so for the following reasons: therapy has signifi-

---

Address correspondence to Dr Bussel, Assistant Professor, Division of Pediatric Hematology/Oncology, The New York Hospital, 525 E 68th St, New York, NY 10021.

cant adverse side effects; treatment is unlikely to affect the course of the disease; the danger of central nervous system hemorrhage is slight; and if thrombocytopenia persisted long enough to justify some form of definitive therapy, splenectomy would be the treatment of choice unless the patient was an infant. Physicians who opted to treat the disorder were concerned about quality of life and the persisting risk of hemorrhage.

An important factor is the emotional stress on the child and the family. Low platelet counts necessitate a restriction of activity. Most physicians, while emphasizing the serious aspects of ITP, usually stress the optimistic long-term outcome. Unfortunately, at present there are no reliable tests to determine in which child the ITP will become chronic and in which it will undergo spontaneous remission. Whether treatment is given immediately or not, the experience may be traumatic for the family—particularly so if the platelet count remains low.

TREATMENT OPTIONS

Corticosteroids. Studies on the use of prednisone in treating ITP yielded two conclusions: treatment with prednisone did not affect the course of the disease; and treatment with prednisone would usually elevate the platelet count only as long as prednisone therapy was continued at an adequate dose.[10,11]

In the past, the problem had been that the only treatment "safe enough" to be used in the majority of patients was prednisone. The side effects of daily prednisone are well known, not the least of which, from the point of view of the family and the child, is the disfigurement associated with developing features of Cushing's syndrome. The choice between persistently low platelet counts and continuous prednisone therapy is not a happy one for the child who does not exhibit at least an adequate platelet count ($30,000/mm^3$ to $40,000/mm^3$) in the first few months after the diagnosis of ITP.

Intravenous gammaglobulin. Much enthusiasm greeted the advent of intravenous treatment with unmodified gammaglobulin. Since no data exist to suggest that intravenous gammaglobulin has any consistent effect on the course of ITP, its use is based largely on its lack of toxicity.[12] In general, the toxicity of gammaglobulin, beyond a post-transfusion headache, has been minimal. Therefore, it is an excellent therapy with which to maintain a child's quality of life while waiting for a spontaneous remission. At least 75% of children with chronic ITP can be maintained with gammaglobulin therapy given less often than every two weeks. However, depending on the family, intravenous gammaglobulin can be an emotionally demanding treatment. The therapy requires about four hours, and its timing remains uncertain in that treatment is given only when the platelet count dips below a certain level ($30,000/mm^3$ to $40,000/mm^3$). Thus, the time interval between treatments may vary, and treatments can be difficult to schedule.

Splenectomy. If a patient is more than six years old (a somewhat arbitrary cut-off point in terms of the risk of postsplenectomy sepsis), and certainly if the child is more than 10 years old, splenectomy becomes a more rational alternative. Mortality due to splenectomy in children is extremely low, and morbidity includes wound infection, which occurs in only a small percentage of patients. The major reasons, therefore, to counsel against splenectomy are the possible lack of success in restoring a normal platelet count and the risk of overwhelming sepsis.

If a patient has had a good previous response to steroids (even if the dose of medication could not be tapered) or if there was a good, though non-sustained, improvement in the platelet count following use of intravenous gammaglobulin, the likelihood of success with splenectomy is 90%. If the patient had no response at all to steroids, the likelihood of success with splenectomy is probably lower, perhaps 60% to 70%. Other prognostic guides have not been shown to be of value in regard to predicting the possible success of splenectomy, although recent unpublished data from Schlicter et al and Rosse et al suggest that high platelet production by the bone marrow is a predictor of a good response. Improvement may follow splenectomy, but even if splenectomy is unsuccessful, removal of the spleen may allow other treatments to be more effective.

Recent innovations have probably decreased the risk of post-splenectomy sepsis. Widespread use of the polyvalent pneumococcal vaccine and prophylactic penicillin have probably decreased the occurrence of sepsis, and the new conjugated *Hemophilus influenzae* vaccine may also prove useful. Therefore, while the risk of post-splenectomy sepsis is an almost absolute contraindication to splenectomy in the child less than two years of age and a relative contraindication in a child less than four or five years old, other children probably have a small risk of post-splenectomy sepsis if appropriate precautions are taken. When there is uncertainty, antibody response to the pneumococcal vaccine can be evaluated to ensure that the patient has high titers of anti-pneumococcal antibodies prior to undergoing splenectomy.

An additional concern regarding splenectomy is the occurrence of a late relapse of ITP. In large adult series these relapses are often due to accessory spleens, which occur in 10% of patients but usually are removed at the time of splenectomy. Relapse may also be due to splenosis, and the patient can often be treated by repeating the splenectomy.[14] Late relapse may occur for other reasons,[15] although this is rare in children.

It is probably wise to avoid platelet transfusions if possible, particularly at the time of splenectomy, even though no studies have been done showing adverse effects of infused platelets.

Fortunately, patients in whom ITP persists even after splenectomy are rare. But when this situation occurs, management should probably be the same as was pursued before the splenectomy. Again, however, if daily corticosteroid therapy is required, then the use of intravenous gammaglobulin should be considered. If this treatment is also ineffective, even when combined with alternate dose of prednisone given on alternate days, immunosuppressive agents may be tried—despite the absence of data supporting their use in children, except for azathioprine. These agents include intravenous vincristine, 0.03 mg/kg weekly for several weeks; azathioprine, 50 to 75 mg/m^2 per day orally; and cyclophosphamide, two to three mg/kg/day orally. Danazol should be restricted to postpubertal males, but vinblastine, 0.1 mg/kg, may also be useful. Again, whereas attaining remission would be most desirable, a platelet count greater than 30,000/mm^3 to 40,000/mm^3 in a patient receiving no therapy is sufficient for a normal quality of life.

CHOICES FOR THE PHYSICIAN

Where does all of this leave the physician confronted by a child with ITP? A patient with acute ITP who at diagnosis has a platelet count less than 20,000/mm^3, and particularly a count less than 10,000/mm^3, and who has signs of active bleeding probably deserves treatment to avoid the risk of central nervous system hemorrhage. Either corticosteroids, or intravenous immunoglobulin, or both are likely to be effective. If the disease goes into remission or the platelet count rises to greater than 30,000/mm^3 or 40,000/mm^3, then no further treatment is required. However, a patient with persistent thrombocytopenia should not be maintained indefinitely on daily prednisone therapy, not only because there are substantial adverse side effects but also because prolonged corticosteroid therapy may suppress bone marrow thrombopoiesis.[16]

For the child with ITP who does not have a satisfactory response to therapy, who is old enough, and whose family is undergoing great stress in dealing with chronic disease management, a splenectomy is probably warranted. Otherwise, a patient with steroid-dependent, chronic ITP should receive periodic single intravenous infusions of maintenance immunoglobulin, in doses up to 1 gm/kg/infusion. These infusions should be given until the platelet count is stable at greater than 30,000/m^3 to 40,000/mm^3 without any therapy.

EMERGENCY MANAGEMENT

A patient with a bleeding problem—intracranial hemorrhage, for example—requires immediate platelet transfusion. Conventional therapy with intravenous dexamethasone or gammaglobulin may also be initiated, but splenectomy should probably be carried out within hours. A computed tomographic scan should be performed after surgery.

LABORATORY DATA

Laboratory tests aside from the platelet count are of limited benefit in the management of a patient with ITP. Platelet sizing, family platelet counts, and immunologic evaluation may help ensure that the child does not have Wiskott-Aldrich syndrome or another hereditary form of thrombocytopenia. May Hegglin anomaly as a cause of chronic thrombocytopenia can be excluded by scanning the peripheral smear. Platelet antibodies have never been shown to be specific for ITP and, therefore, are relatively useless in its diagnosis.[17] Serial platelet antibodies may help to determine that a given therapy, such as corticosteroids, has been effective in decreasing the platelet-associated IgG. Bleeding times may help to establish "safe" platelet counts in individual patients, since some patients will have normal bleeding times at platelet counts of 30,000/mm^3 and therefore not require therapy if the count is stable at this level. Tests for systemic lupus erythematosus are almost always fruitless in the absence of clinical symptoms and would probably not alter management even if positive, except perhaps for showing the need for more careful monitoring of renal function.

CONCLUSIONS

In summary, ITP of childhood is a relatively benign disease with many available treatments. In view of its favorable natural history with a strong tendency to spontaneous remission, it is particularly important that the treatments do not cause more harm than the disease; splenectomy should be postponed until at least one year from diagnosis if possible. The best maintenance therapy to "buy time" until such spontaneous improvement does occur is periodic infusion of unmodified intravenous gammaglobulin. However, especially in the growing child, quality of life, familial anxiety, and restriction of normal childhood activities are important issues which need to be addressed and, therefore, treatment decisions need to be tailored to the individual family.

REFERENCES

1. Stuart M, McKenna J: Diseases of coagulation: the platelet and vasculature, in Nathan K, Oski F (eds): *Hematology of Infancy and Childhood.* Philadelphia, Saunders, 1984, p 1234.
2. McClure PD: Idiopathic thrombocytopenic purpura in children: diagnosis and management. *Pediatrics* 1975; 55:68–74.
3. Ramos ME, Newman AJ, Gross S: Chronic thrombocytopenia in childhood. *J Pediatr* 1978; 92:584–586.
4. Harrington WJ, Minnich V, et al: Demonstration of a thrombocytopenic factor in the blood of patients with thrombocytopenia purpura. *J Lab Clin Med* 1951; 38:1–10.
5. Harrington WJ, Sprague CC, Minnich V, Moore CV, Aulvin RC, Dubach R: Immunologic mechanism in idiopathic and neonatal thrombocytopenic purpura. *Ann Intern Med* 1953; 38:433–469.
6. McMillan R, Longmire RL, Yelenosky R, Donnell RL, Armstrong S: Quantitation of platelet-binding IgG produced in vitro by spleens from patients with idiopathic thrombocytopenic purpura. *N Engl J Med* 1974; 291:812–817.
7. Karpatkin S, Strick N, Siskind GW: Detection of splenic anti-platelet antibody synthesis in idiopathic autoimmune thrombocytopenic purpura (ATP). *Br J Haematol* 1972; 23:167–176.
8. McMillian R: Chronic idiopathic thrombocytopenic purpura. *N Engl J Med* 1981; 304:1135–1147.
9. Komrower GM, Watson GH: Prognosis in idiopathic thrombocytopenic purpura of childhood. *Arch Dis Child* 1954; 29:502–506.
10. Simons SM, Main CA, Yaish HM, et al: Idiopathic thrombocytopenic purpura in children. *J Pediatr* 1975; 87:16–22.
11. Benham ES, Taft LI: Idiopathic thrombocytopenic purpura in children: results of steroid therapy and splenectomy. *Aust Pediatr J* 1972; 8:311.
12. Bussel JB, Schulman I, Hilgartner MW, Barandun S: Intravenous use of gammaglobulin in the treatment of chronic immune thrombocytopenic purpura as a means to defer splenectomy. *J Pediatr* 1983; 103:651–654.
13. Dunn NL, Maurer HM: Prednisone treatment of acute idiopathic thrombocytopenic purpura of childhood. Advantages. *Am J Pediatr Hematol Oncol* 1984; 6:159–164.
14. Verheyden CN, Beart Jr RW, Clifton MD, Phyliky RL: Accessory splenectomy in management of recurrent idiopathic thrombocytopenic purpura. *Mayo Clin Proc* 1978; 53:442–446.
15. Venetz U, Willi P, Hirt A, Imbach P, Wagner HP: Chronic idiopathic thrombocytopenic purpura in childhood. *Helv Paediatr Acta* 1982; 37:27–33.
16. Cohen P, Gardner FH: The thrombocytopenic effect of sustained high-dosage prednisone therapy in thrombocytopenic purpura. *N Engl J Med* 1961; 265:611–617.
17. Mueller-Eckhardt C, Kayser W, Mersch-Baumer K, et al: The clinical significance of platelet-associated IgG; a study on 298 patients with various disorders. *Br J Haematol* 1980; 48:123–131.

A 77-year-old woman with giant-cell arteritis, back pain, and shock

CASE PRESENTATION AND CLINICAL DISCUSSION

Antoinette M. Kuzminski, MD (Attending Physician, Department of Medicine): A 77-year-old Lebanese-born woman sought treatment for generalized fatigue, loss of appetite, and a 5-pound weight loss during an eight-month period. In addition, for about four months she had had severe left-sided headaches, bilateral ear and jaw pain with chewing and swallowing, and a bitter taste in the mouth. She had had fleeting pains in both shoulders several months earlier, but these had subsided. The erythrocyte sedimentation rate (ESR) was 99 mm/hr; giant cell arteritis was suspected, and biopsy of the temporal artery confirmed the diagnosis.

Approximately seven years previously, she had been seen in the Ambulatory Clinic with almost identical complaints, including anorexia, dysgeusia, night sweats, and fatigue, but without headache or jaw claudication. Two weeks thereafter, the complaints had subsided and no further investigation appeared indicated at that time.

She reported having had peptic ulcer disease in the remote past, and ulcer symptoms had recurred one year prior to presentation. She had had well-controlled hypertension for many years.

A 48-year-old son had died of an aortic aneurysm four years earlier.

Physical examination was remarkable for diffuse scalp tenderness, diffuse nodular breast tissue, and a stool positive for occult blood. Her pulses were strong, easily palpable, and of equal strength on both sides. She was treated with prednisone, cimetadine, antacids, and continued antihypertensive therapy. During the following week she showed marked improvement in all of her initial complaints except for the dysgeusia. Prednisone was continued at a dose of 40 mg daily for one month, with reduction of the ESR to approximately 20 mm/hr. Physical changes suggestive of Cushing's syndrome, fluid retention, and worsening hypertension led to a reduction of prednisone dosage to 10 mg/day and to an increase in the dose of antihypertensive medication during the following month, with good control of her blood pressure.

Three months after her initial presentation she complained of episodic tingling and numbness in her hands, feet, lips, and tongue. Her dysgeusia persisted, but her other initial symptoms and signs had resolved. Cardiovascular and neurologic examinations were within normal limits. The ESR was 38 mm/hr and, later, 45 mm/hr. The prednisone dosage was increased to 20 mg/day and was maintained at that level throughout the next two months. She felt well except for the persistent dysgeusia, and the ESR remained between 20 mm/hr and 40 mm/hr. The tingling and numbness in the hands persisted.

The patient then wished to discontinue the prednisone therapy. During the next six months, a progressive reduction of prednisone dosage by 10% increments was initiated, so that the dosage eventually had been reduced to 5 mg every other day. She was asymptomatic; the dysgeusia, tongue numbness, and tingling sensations in the hands and feet had resolved; the ESR remained between 30 mm/hr and 55 mm/hr.

After the one year of ambulatory follow-up, she was admitted to the hospital with complaints of acute onset of back and chest pain; when seen in the emergency department, she was found to be in shock, with electromechanical dissociation. Blood pressure was palpable in the carotid arteries and in the arteries of the left arm only. Dissection of the aorta was suspected based on clinical findings and on a chest film; pericardiocentesis on suspicion of cardiac tamponade was performed without benefit, and the patient expired.

Steven E. Szebenyi, MD (Attending Physician): How could she have palpable pulses and be in electromechanical dissociation?

Dr Kuzminski: When she was first seen in the emergency department, her blood pressure was unobtainable, and for the first 15 minutes of observation, she was treated as though she were in electromechanical dissociation. Then it was discerned by Doppler study that her blood pressure could be detected in the arteries of the left arm. I do not think that she was actually in electromechanical dissociation at that point, but late in the course of her stay in the emergency department, she was in true electromechanical dissociation, with no pulses palpable anywhere.

Dr Vaules performed an echocardiogram in the emergency department which demonstrated a grossly dilated aortic root, which also seemed to have an extra lumen—in confirmation of the diagnosis of aortic dissection.

RADIOLOGY PRESENTATION

Peter T. Wright, MD (Attending Radiologist): Only two sets of chest films are available. The earlier film taken during the year prior to the patient's final illness was an upright posteroanterior film: it shows a fairly normal cardiac silhouette, with a well outlined aortic knob; the descending aorta is less well seen. The lung fields are clear. There is an area of intimal calcification at the top of the aortic knob, which is perceived better on a closer view. The thickness of the aortic wall between the intima and the adventitia at this point is about 2 mm to 3 mm, which is within the normal range (Fig 1).

David W. Vaules, MD (Attending Cardiologist): Since

Based on the clinicopathologic conference (CPC) held at the Mary Imogene Bassett Hospital, Cooperstown, NY, on September 13, 1984.

Clinical Discussion: Antoinette M. Kuzminski, MD, Attending Physician, Department of Medicine; *Radiology Presentation:* Peter T. Wright, MD, Attending Radiologist; *Pathology Presentation:* Harlan D. Alpern, MD, Pathologist-in-Chief.

Editor: JOHN T. FLYNN, MD

Address correspondence to Dr Flynn, Consulting Editor, *New York State Journal of Medicine,* PO Box 5404, 420 Lakeville Rd, Lake Success, NY 11042.

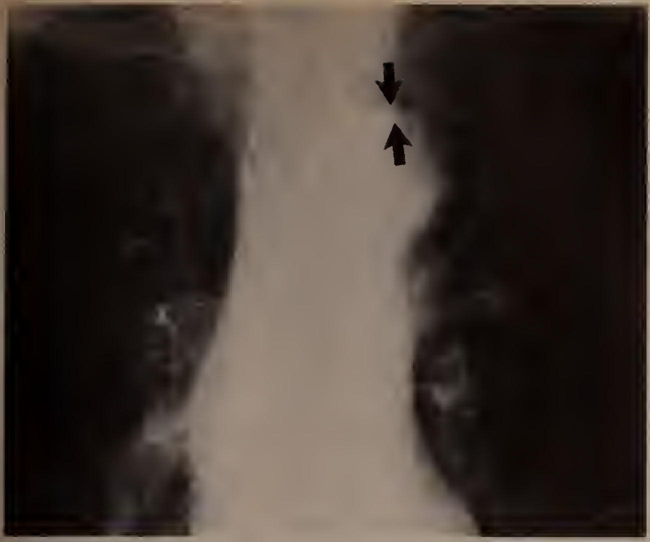


FIGURE 1. Chest film one year prior to death, showing intimal calcification at aortic knob, with normal aortic wall thickness (arrows).

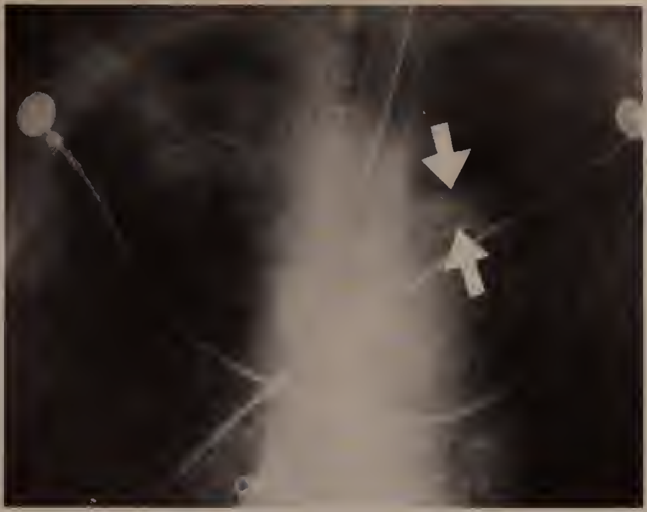


FIGURE 2. Emergency portable chest film on admission, showing mediastinal widening, with marked increase in thickness of aortic wall at level of intimal calcification (arrows).

the aorta is a circular structure, how can one be sure that this is not a tangential view?

Dr Wright: We know that the aortic knob is not really a single anatomic structure. It actually represents the transverse aortic arch, so that it is possible to see a small calcification in the region of the aortic knob, and superimposed on that we might see another portion of the aortic knob, either in front or in back of that point. That can lead to the false assumption that we are seeing either aortic wall thickening or even a normal thickness of the aortic wall. The only place where the aortic wall thickness measurement is reasonably reliable is at the top of the arch, because there is usually no aortic tissue superior to that point. Consequently, if we see the calcification at the superior aspect of the aortic knob, the thickening above that point should represent the true thickness of the aortic wall. If we see lateral or inferior calcification, we know that the arch at these points is extending either downward or upward both anteriorly and posteriorly; so that it is possible at these levels to misdiagnose aortic wall thickening.

Another useful source of information is the location of the calcification on the lateral chest film; there is a lateral film of this patient which shows that this calcification was at the top of the aortic knob.

The chest film taken in the emergency department on her final admission was an anteroposterior portable study, with the patient in the supine position. Again, we can see the intimal calcification, but now there is visible thickening of the aortic wall at this level, from 2 mm to 3 mm on the previous film to 11 mm to 12 mm on the current examination (Fig 2). The finding of abnormal pulses in the upper extremities, the difficulty in determining the blood pressure, and the earlier chest film which shows that the calcification was initially in a different location all strongly suggest the diagnosis of aortic dissection.

In general, the radiologic signs of aortic dissection on a plain chest film include aortic wall thickening or displacement of intimal calcification, which we are fairly certain is true in this patient; besides this, there may be successive change in the size or shape of the aorta on serial films, localized protrusions of the aortic knob distal to the brachiocephalic vessels, a large descending aorta with a small ascending aorta or vice versa, and the presence of blood in one of the hemithoraces.[1] Mediastinal widening is also a fairly good sign of aortic dissection, but in this patient it was difficult to assess change in the mediastinal width from one chest film to another, since we were trying to compare an upright posteroanterior film with a supine portable film. Admittedly, the mediastinum in this patient appeared widened on her final chest film, probably secondary to a mediastinal hematoma.

We felt that with the clinical history and the roentgenographic findings, an aortic dissection was present that involved the arch—a DeBakey Type I, which at times is amenable to surgical repair. Aortography or computerized tomography would have been useful confirmatory tests, but we did not have the opportunity to perform these tests on this patient. However, we felt confident that this was an aortic dissection.

Dr Vaules: An increase in the size of the cardiac silhouette might also suggest that a dissection has taken place.

Dr Wright: Yes, such a change would suggest hemopericardium secondary to rupture into the pericardial space. In this patient, the supine portable film was compared with the earlier upright film, and no gross change was noted. In situations in which a sequence of chest films permits comparison, an increase in the cardiac silhouette is a helpful sign.

Dr Kuzminski: This patient's several risk factors for an aortic dissection were long-standing hypertension, a family history of aortic dissection, and a history of vasculitis. The overwhelming majority of aortic dissections are related to chronic hypertension, or cystic medial necrosis, or both. It was thought that such was the situation in this patient.

I will focus my discussion on vasculitis as a cause of aortic dissection. Giant-cell arteritis is known to affect the large extracranial vasculature in about 10% of cases. Two studies originating in Scandinavia[2] in the early 1970s showed that five out of 61 patients in one series and 14 out of 93 in the other presented this less common distribution of vessel involvement in giant-cell arteritis. As criteria for involvement of the larger vasculature in this disease, these studies did not depend on any specific radiologic findings, but were rather based on such elements as complaints of claudication, the presence of vascular bruits, absent pulses, and the findings on aortography.

In 1975, Klein[3] at the Mayo Clinic reported on 248 consecutive patients with giant-cell arteritis, and observed that 23 had definite extracranial large-vessel involvement. The criteria in this study were more strict; a bruit alone was not accepted as evidence of local vascular involvement. Later, Healey and Wilske[4] found five such patients in a series of 50 patients with giant-cell arteritis. Although aortic or other large extracranial vascular involvement is relatively common, a catastrophic outcome such as a myocardial infarction or an aortic dissection seems to be rare in average patient populations with giant-cell arteritis. I have reviewed 11 major series of patients with giant-cell arteritis that have appeared in the literature since 1965. These describe 774 patients, nearly all of whose disorders were identified on biopsy and nearly all of whom were treated with glucocorticoids. Altogether there were three myocardial infarctions and three aortic dissections, which were attributed at post-mortem study to direct vasculitic involvement of the heart or aorta. There were no isolated infarctions of the extremities or viscera. In addition, numerous case reports of aortic dissection due to giant-cell vasculitic involvement are scattered through the medical literature.

It is difficult to assemble a profile of patients with giant-cell arteritis who are likely to develop aortic dissection. These reports range from patients who were not suspected of having vasculitis prior to death, to patients taking as much as 60 mg of prednisone daily. Some of the patients were symptomatic, with claudication or other suggestive symptoms; others were asymptomatic. Some patients were only in the initial phases of steroid therapy, some were tapering their steroid dosages, and some had recently discontinued steroid therapy and were thought to be in remission. In most of the case reports in which the patient was known to have giant-cell arteritis, the laboratory data had returned to normal levels at the time of the aortic dissection. The unpredictability of a catastrophic outcome raises many questions regarding the natural course of giant-cell arteritis and the guidelines that we use for its treatment.

The patient under discussion today seemed typical of the population of patients with giant-cell arteritis in almost every way. Her age and sex, her presenting symptoms, and her initial response to steroids were all classic for giant-cell arteritis. Later in the course of her illness, when her steroid dosage was gradually reduced, her ESR slowly increased, but not in correlation with any symptoms. One atypical finding was the complaint of paresthesias; I cannot attribute this to vascular insufficiency, since the complaint involved a diffuse symmetrical distribution, with improvement as steroid therapy was withdrawn. In fact, this patient is disturbingly typical of the majority of patients with giant-cell arteritis.

In 1983, a review of the clinical courses of 80 patients with giant-cell arteritis included an analysis of 678 outpatient visits[5]; the onset of symptoms and elevated ESR paralleled each other on only 23% of these visits. My own survey at this institution of 23 patients with long-term follow-up for giant-cell arteritis indicated approximately 50 clinical encounters in which the symptoms and ESR could not be correlated. Of the 29 patients in this institution with biopsy-proven temporal arteritis since 1978, 10% of the patients had erythrocyte sedimentation rates of less than 30 mm/hr at the time of the initial presentation, when vasculitis was presumably in its most active stage. Clearly, the ESR is a flawed tool; there is ample demonstration here today that normal sedimentation rates are not uncommon; we have no knowledge of the frequency of false positive test results.

With symptoms and laboratory evaluations constituting such poor measures of disease activity, there is no wonder that we know so little about this disease. Only serial biopsies of involved vessels will clarify this clinical picture; and even this would be compromised by the occurrence of "skip" lesions which might result in false negative biopsies.

A report in 1972 by Fauchald[6] included follow-up biopsies in 35 patients with proven giant-cell arteritis. Fifteen patients had biopsies while still undergoing treatment. Ten showed no clinical evidence of disease, but two of these asymptomatic patients had active arteritis at biopsy, and one showed residual infiltrates. One patient with clinical signs of continued disease activity had a negative biopsy.

Biopsies obtained after treatment showed the same incomplete correlation between clinical impression and tissue evidence of disease activity. Biopsies were taken in 20 patients at varying intervals following completion of therapy. Sixteen patients in this group showed no clinical signs of disease activity. At the time of biopsy of these 16, one had active arteritis and three had residual infiltration. The patient in whom active disease was observed had been asymptomatic for 10 years since the original diagnosis. Again, there were two patients in this group of 20 whose clinical symptoms suggested that an active arteritis was present, but whose biopsies were negative. This may reflect sampling error.

The tendency to relapse in temporal arteritis has been noted; this term implies that there has been at least a temporary cessation of disease activity before the recurrence. I think that in light of this material, the term "flare-up" would be more appropriate to use in this situation. There is substantial evidence that giant-cell arteritis may be present or even active without any clinical signs, symptoms, or laboratory evidence. I suspect that the patient described in this report was suffering from giant-cell arteritis in 1977, and it is even conceivable that it continued to smoulder ever since.

If indeed this patient's dissection was secondary to active giant-cell arteritis, what lessons can we learn from this case? We can be aware that a catastrophic outcome can occur, even when patients feel perfectly well and their laboratory tests are normal or not grossly altered. It flies in the face of common sense to continue to use a moderately toxic drug to treat an abnormal ESR; yet the ESR, nonspecific though it is, was the only indicator in this patient that the arteritis was active. If this patient had been given continuous treatment the final catastrophe perhaps could have been averted.

John S. Davis, MD (Attending Physician): Could this patient have had polymyalgia rheumatica seven years ago? Her symptoms, as described, do not sound as though she did; but she could have been started on low-dose prednisone by some other physician who was alert to the possibility of that diagnosis. The question then would be whether such treatment would have altered the subsequent course of events by affecting what might have been a precursor of her arteritis. There is presumably no way to have been sensitive to the question of giant-cell arteritis seven years ago, although, in retrospect, if such a diagnosis had been suspected, the physician would probably have wanted to treat the disorder with high-dose prednisone.

Dr Kuzminski: A sizeable number of people who have no symptoms of giant-cell arteritis and who appear to have polymyalgia rheumatica actually have positive biopsies of their temporal arteries. This patient did have symptoms suggestive of temporal arteritis; I think that the dysgeusia was due to ischemia of the tongue, since I could not explain it otherwise.

Nancy Merrill, MD (Attending Physician): Does giant-cell arteritis occur in families? Was an autopsy performed on the patient's son, who is reported to have had a dissecting aneurysm?

Dr Kuzminski: Yes, there are reports of the occurrence of dissecting aneurysm in families. The post-mortem examination of the patient's son did not show any vasculitis; he had cystic medial necrosis. I have seen the reports of the pathologic studies.

Dr Vaules: Pericardiocentesis should be approached with caution in patients who are suspected of having a dissection of the aorta with extension into the pericardium. If the patient is otherwise stable and alert, one may not want to do the pericardiocentesis immediately or at all; sudden relief of the pressure in the pericardium can facilitate further bleeding into the pericardial cavity. One has to make a judgment at the time as to whether to do the procedure or not, and one could be wrong either way. But one does not assume that all patients with such a dissection and with such a catastrophic presentation should undergo pericardiocentesis.

David Svahn, MD (Attending Physician): If, at some point in the patient's treatment her blood pressure had risen abruptly, what medications could have been used to control it, in order to avoid a dissection?

Dr Kuzminski: Her blood pressure was adequately controlled on aldactazide and nadolol, as I recall. The patient was resistant to taking medications, but her blood pressure was adequately controlled.

Dr Szebenyi: On the chest film, her lung volumes appear quite generous. Was she a cigarette smoker?

Dr Kuzminski: No, she was not.

CLINICAL DIAGNOSES

Giant-cell arteritis.

Dissecting aortic aneurysm, associated with giant-cell arteritis.

Hypertension.

Peptic ulcer disease, resolved.

Long-term glucocorticoid therapy.

PATHOLOGY PRESENTATION

Harlan D. Alpern, MD (Pathologist-in-Chief, Mary Imogene Bassett Hospital): The diagnosis of giant-cell temporal arteritis was made on the biopsy of the left temporal artery, a year prior to the patient's death (Figs 3, 4). An inflammatory process is present which involves the entire vessel wall. In the media, there is granulomatous inflammation with necrosis. A cellular infiltrate composed of histiocytes, fibroblasts, lymphocytes, and multinucleated giant cells is present along the margins of the necrotic areas. There are multinucleated giant cells in relation to the internal elastic lamina, the fragmentation and destruction of which is well demonstrated by the elastic tissue stain. There is fibrous intimal thickening with narrowing of the lumen and lymphocytic infiltration of the adventitia. Although the cause of giant-cell arteritis is unknown, it has been suggested that damaged vascular elastic tissue, especially from the internal elastic lamina, may act as an antigen to incite vascular inflammation. Granular IgM deposits have been found in affected arte-

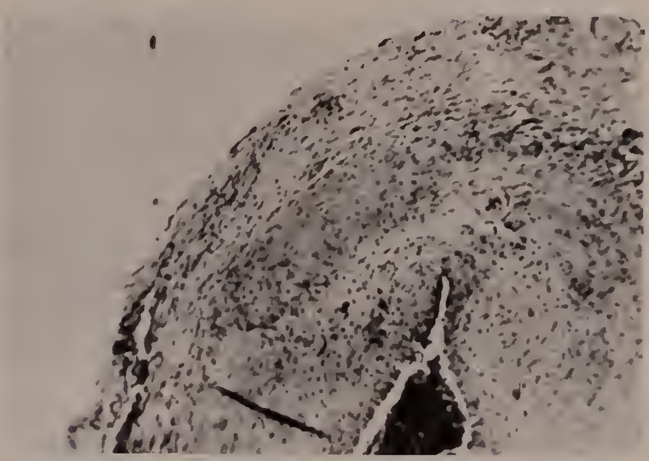


FIGURE 3. Biopsy of left temporal artery, showing granulomatous inflammation. (hematoxylin and eosin stain, original magnification X 40)

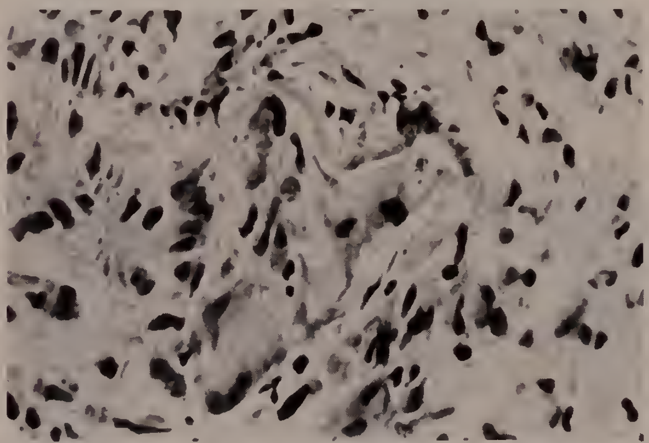


FIGURE 4. Higher power view of arterial biopsy, showing multinucleated giant-cells. (hematoxylin and eosin stain, original magnification X 400)

ries near the internal elastic lamina.[7]

An autopsy demonstrated that the patient died as a result of cardiac tamponade due to a hemopericardium, consisting of 150 mL of fresh blood and small clots. This hemopericardium was the result of a dissecting aneurysm within the media of the aorta, along its full length, with proximal dissection into the pericardium and distal dissection into the major aortic branches. The dissection produced a second lumen which contained blood (Fig 5). The dissection occurred as the result of an aortic tear in the intima and media at the root of the aorta (Fig 6). This tear was oblique and measured 2 cm in length. From this point the dissection extended both proximally and distally.

Cystic medial necrosis, which is usually associated with a dissecting aneurysm, was not found. Instead the aneurysm was the result of giant-cell aortitis. Histologic sections of the aorta demonstrated regions of intense granulomatous inflammatory infiltrations, composed of lymphocytes, histiocytes, and multinucleated giant cells (Fig 7). There was destruction of the elastic tissue in the region of the inflammation. Ruptured aortic aneurysm is a rare complication of giant-cell arteritis. In the series of 248 patients from the Mayo Clinic reported by Klein et al,[3] three died of aortic rupture.

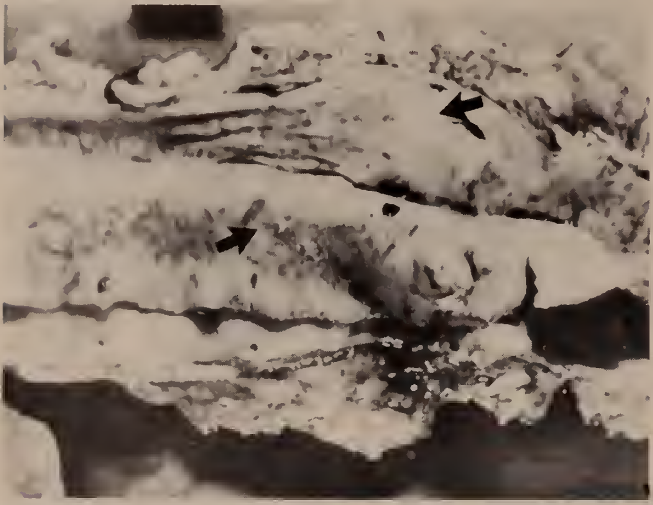


FIGURE 5. Outer layer of aorta (upper arrow) separated from inner layer (lower arrow) by intramural dissection.

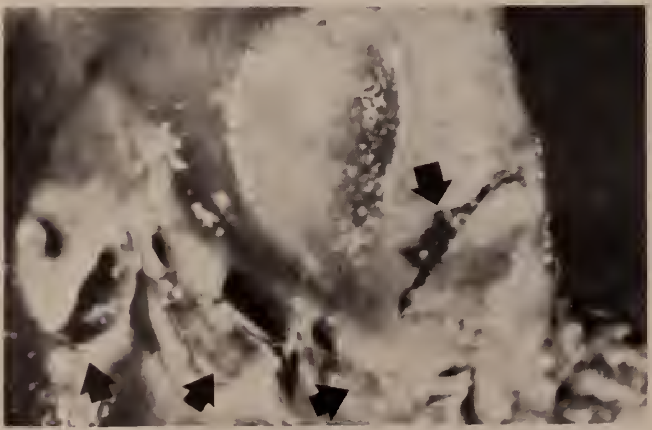


FIGURE 6. Root of aorta showing aortic valve cusps (lower arrows) and 2-cm aortic intimal tear (upper arrow).

The opposite (right) temporal artery was biopsied at the time of the autopsy and demonstrated active disease; there was intimal fibrosis and granulomatous inflammation in the media. Some calcifications were present. Multinucleated giant cells were numerous. Sections of the internal carotid artery showed evidence of giant-cell arteritis. There was no evidence of arteritis in the coronary, renal, or other abdominal branches of the aorta. The small and intermediate-sized vessels in all the organs showed no evidence of arteritis. There was coronary atherosclerosis.

The lungs were edematous and congested; they were approximately twice the normal mass. The patient had had a duodenal ulcer; at the time of death, the ulcer was healed, with re-epithelialization. In the stomach, there were two submucosal leiomyomata. There was diverticulosis of the colon, renal arterionephrosclerosis, and two small renal cortical adenomas. In addition, another nodule in one kidney proved to be a renal medullary interstitial cell tumor, which represents a proliferation of mesenchymal cells in the renal stroma. These tumors are relatively common in people over 50 years of age; it has been suggested that because of purported endocrine-like antihypertensive action of renal medullary interstitial cells, the tumor may represent a response to hypertension.

The clinical examination of the right breast had raised suspicions of a tumor; at autopsy, only fibrocystic disease was found.

This patient died of cardiac tamponade, following a massive hemorrhage into the pericardial cavity, which occurred as a result of a dissecting aneurysm not related to cystic medial necrosis or hypertension, but due to giant-cell aortitis. In this patient, this inflammation was a systemic disease, affecting multiple arteries, especially the aorta, producing a dissecting aortic aneurysm, and leading to her death.

Dr Davis: Did you have an opportunity to review the pathologic findings in the son? I find it incredible that the son would have died of an aortic dissection due to medial-cystic necrosis, and the mother died of an aortic dissection due to a different entity.

Dr Alpern: Cystic medial necrosis is generally found in cases of dissecting aneurysm, although the relationship between the two is unclear. The slides of the son's autopsy were reviewed, and I concur with the diagnosis made at the Albany Medical Center of dissecting aortic aneurysm associated with cystic medial necrosis. No aortitis was present.

Dr Davis: Is it possible that medial cystic necrosis of the aorta is relatively common, and most affected people are never harmed by it? Could that be because most such patients do not have the hypertension that might lead to a rent in the aorta?

Dr Alpern: That is the theory. However, the exact cause of dissecting aneurysm is not known, because cystic medial necrosis is found in some older persons who did not develop dissecting aneurysm. The relationship between cystic medial necrosis and dissecting aneurysm, be it causal or coincidental, is uncertain. Whether aortic dissection requires the additional element of hypertension, or whether

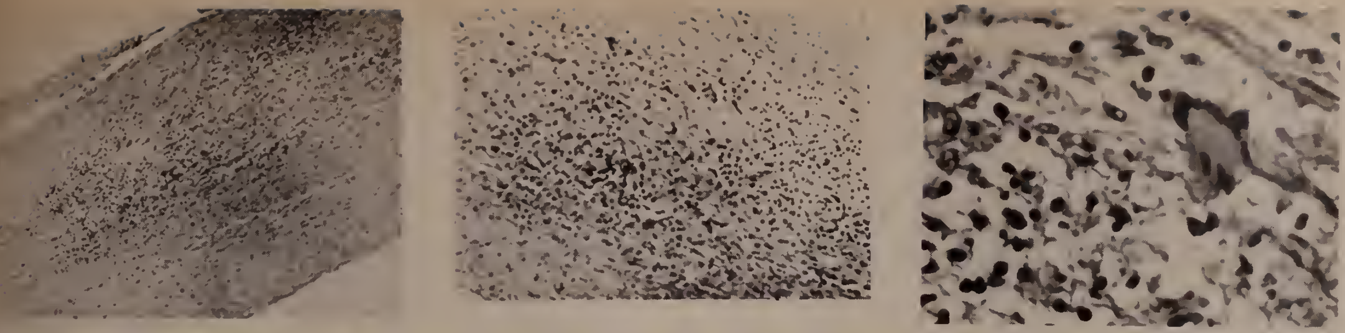


FIGURE 7. Left, granulomatous inflammation of aorta (hematoxylin and eosin stain; original magnification × 25); Center, original magnification × 100; Right, original magnification × 400.

there are other factors, is still unknown. Atherosclerosis is thought not to have a significant role.

Dr Vaules: Do you think that people with normal aortas develop small tears that never dissect and are never found?

Dr Alpern: No. The normal aorta does not tear like this. The aortic elastic tissue is very strong. The entire aorta is composed of very tough elastic tissues.

Dr Kuzminski: I take it that this kind of arteritic inflammatory change can go on for a very long time without the occurrence of a tear. Would it be possible that the combination of hypertension and the aortitis, and possibly even some cystic medial necrosis which was obscured by inflammation, could lead to the dissection?

Dr Alpern: I think that the hypertension is important in the predisposition to rupture. Seventy percent of patients who have the usual dissecting aneurysm have hypertension, and 90% have left ventricular hypertrophy, suggesting that previous hypertension had been present. Cystic medial necrosis was not seen in this case.

Dr Davis: What dosage of prednisone was she taking at the time of death?

Dr Kuzminski: Five milligrams every other day—not a significant dosage.

Alan J. Kozak, MD (Attending Physician): In patients who are successfully treated for temporal arteritis, is there generally any histologic residue from the original inflammatory process, which you might find at autopsy?

Dr Alpern: In some patients with giant-cell arteritis which has resolved, one may find residual intimal fibrosis.

Dr Kuzminski: But the studies that I discussed earlier indicate that active disease can persist.

Dr Alpern: Some patients who are thought not to have active disease based on clinical findings may have underlying disease; the opposite temporal artery studied at autopsy in this patient showed evidence of florid disease.

Gary Hoffman, MD (Attending Physician): As more information accumulates about giant-cell arteritis, we have to alter our concept as to how it may present. For example, there are reports of patients who present with confusing multisystem disease, and at some point in their illness may undergo a bone marrow study. Giant-cell arteritis has even been identified in the small vessels in the marrow, a very unusual event.[8] We have been rigidly adhering to the classification system described by Zeek[9] and promulgated by others since then, suggesting that one can readily distinguish many of the arteritides on the basis of the size and distribution of the vessels involved. Such schemes are at best generalizations and may not appropriately accommodate many clinical examples, in which symptoms and findings overlap different vasculitic categories.

Do you feel, Dr Kuzminski, that this case will affect the way in which you are going to treat the next patient you identify with this disease?

Dr Kuzminski: It is already affecting the treatment of two other patients under my care. I have continued their steroid therapy because of the changes in the ESR, even without symptoms.

Dr Vaules: Are you planning to have follow-up blood-vessel biopsies on these patients in the future?

Dr Kuzminski: That is a good idea.

Dr Hoffman: Are you going to continue daily dosages of steroids in such patients?

Dr Kuzminski: Yes, though I have had only a limited number of patients in this category so far.

Dr Hoffman: With regard to long-term steroid therapy in temporal arteritis, it is unclear as to how long such therapy should be continued; the symptoms tend to come and go. It would seem that steroids might be discontinued only if the patient was asymptomatic and the ESR had returned to normal; steroid therapy would ordinarily be reinstituted if the symptoms returned and the ESR again became abnormal.

Dr Kuzminski: The current recommendations for long-term management in giant-cell arteritis are largely aimed at prevention of blindness, which is by far the most common vascular complication in this disease. The incidence of this event is clearly dropping, largely, it seems, due to the more frequent recognition of the disease and its early treatment with high-dose steroid therapy. There can be little argument regarding the effectiveness of early dose-steroid therapy. The long-term management is much less clear cut.

There is sparse information in the literature regarding vascular catastrophes late in the course of giant-cell arteritis. The few patients described in detail in the literature seemed to have clinically quiescent disease, and normal sedimentation rates are the rule rather than the exception. Definition of an abnormal sedimentation rate is not clear. Some authors accept sedimentation rates up to 40 mm/hr. In the survey of 11 series that I mentioned earlier, no one specified what norm was used for sedimentation rate.

Richard Reese, MD (Attending Physician): Suppose the patient was asymptomatic but had an ESR in the 40 mm to 50 mm range, and after a week of steroid therapy the ESR was clearly much less. Would that mean that, despite the absence of symptoms, there was nevertheless some active arteritic inflammation going on in the patient, that it was responding to the steroid therapy, and that therefore the steroids should be continued?

Dr Hoffman: When the ESR decreases, one has reason to reduce the dosage. There are so many factors that can affect the ESR; in the absence of a paraprotein, red blood cell sedimentation varies directly with the level of serum fibrinogen, its coating of erythrocyte membranes, surface charge neutralization, and subsequent formation of red blood cell rouleaux. One must be careful to consider non-giant-cell-arteritis intercurrent events that may raise the sedimentation rate in these patients.

Dr Reese: But there aren't many diseases in the follow-up of which we depend so exclusively on the sedimentation rate as we do with this disease.

Dr Hoffman: I think that if the patient is feeling well and is either on low-dose steroid therapy or none at all, and if one finds that the ESR has increased, the test should be repeated. Often the repeat test will differ significantly from the earlier test, without any interval change in therapy. We have followed a number of patients with giant-cell arteritis with moderately elevated sedimentation rates, who have felt well and in whom steroids were not increased on the basis of the ESR alone. I have followed one patient with a moderately elevated ESR for eight years who had biopsy-proven disease and was quite ill when she first presented. After steroid-induced remission and subsequent discontinuance of steroids, she has done very well, even though the ESR would rise and fall. Am I doing her an injustice by not treating her when the ESR would rise? Is she insidiously developing aortitis and other vascular disease, or are these perturbations of the ESR unrelated to giant cell arteritis? I don't know. It is certain, however, that she would become quite toxic if given high doses of steroids. That fact is a given; the question of a possible aortic dissection on the other hand is uncertain, and I believe much less likely.

Thus I feel that in selected cases of proven giant-cell arteritis, it is justifiable not to raise the dose of steroids, and not to reinitiate steroids when a sedimentation rate rises, when the patient is asymptomatic and has no other features of reactivated vasculitic disease. The ESR rise should, however, be a signal to follow the patient more frequently for other clues of disease reactivation that would justify altering steroid therapy.

Dr Davis: In this patient, you described various events related to steroid therapy—facial changes suggestive of Cushing's syndrome, increase in blood pressure, fluid retention. She did not develop diabetes, compression fractures, or cataracts. I wonder whether the steroids may have been a factor in her death, such as by augmenting her hypertension. That tear in the aorta was the crucial event; she probably could have lived with her aortitis for an indefinite time.

Dr Hoffman: One type of compromise in the therapeutic approach here would be high-dosage alternate-day steroid therapy, once initial disease control is obtained with daily doses. Such modification of treatment does not avoid steroid-induced osteoporosis; however, it does minimize toxicity to many other organs that are the targets of steroid toxicity. The Mayo Clinic data that have promulgated the use of daily steroids for giant-cell arteritis, rather than doses every other day, have been misquoted. They address only *initial* steroid therapy for this condition, not chronic therapy.[10] For those patients who received high-dose daily steroids, the data show significantly fewer symptoms than those treated every other day during the first month of therapy (45 mg every day, versus 90 mg every other day). That was a short-term study dealing with initial therapy; we do not know how many of those patients might have improved, to become well after gradual tapering to an alternate-day program at a later date.

At this hospital we have treated some patients with giant-cell arteritis quite successfully with alternate-day therapy after initial control was evident following a daily dose. I believe that these patients were spared greater potential steroid toxicity.

Dr Kuzminski: There is probably a large number of patients with giant-cell arteritis who have active aortitis that we are not aware of. It is fortuitous as to whether such a patient will develop a dissection; there must be some other factor involved, since so many of the patients in the case reports of dissection were not experiencing florid reactivation of arteritis.

Dr Vaules: It is interesting to speculate as to how this happened, in terms of the location of the initial tear. It developed very close to the pericardial reflection, which in terms of trauma to the heart and great vessels is one of the areas most susceptible to tears. If this patient had sustained some type of chest trauma, which to someone else might have been insignificant, such injury alone might have been sufficient to start the dissection.

Dr Kuzminski: This patient happened to be at rest when the dissection developed.

Final Diagnoses

Dissecting aneurysm, secondary to giant-cell aortitis, involving the ascending aorta, with full-length proximal and distal dissection; dissection of the major aortic branches; and hemopericardium (150 mL).

Giant-cell arteritis, right temporal and internal carotid arteries.

Edema and congestion of the lungs.

Moderate atherosclerosis of coronary arteries.

Healed ulcer in first part of duodenum.

References

1. Abrams HL; Dissecting aortic aneurysm, in Abrams HL (ed): *Abrams' Angiography Vascular and Interventional Radiology*. Boston, Little Brown and Co, 1983, pp 441-466.
2. Hamrin B: Polymyalgia arteritica. *Acta Med Scand* (suppl) 1972; 533:1-131.
3. Klein RG, Hunder GG, Stanson, AW, Sheps SG: Large artery involvement in giant cell (temporal) arteritis. *Ann Intern Med* 1975; 83:806-812.
4. Healey LA, Wilske KR: Manifestations of giant-cell arteritis. *Med Clin NA* 1977; 61:261-270.
5. Ellis ME, Ralston S: The ESR in the diagnosis and management of the polymyalgia rheumatica/giant-cell arteritis syndrome. *Ann Rheum Dis* 1983; 42:168-170.
6. Fauchald P, Rygvold O, Øystese B: Temporal arteritis and polymyalgia rheumatica. *Ann Intern Med* 1972; 77:845-852.
7. Bonnetblanc JM, Adenis JP, Queroi M, Rammaert B: Immunofluorescence in temporal arteritis. *N Engl J Med* 1978; 298:458.
8. Enos WF, Pierre RV, Rosenblatt JE: Giant cell arteritis detected by bone marrow biopsy. *Mayo Clin Proc* 1981, 56:381-383.
9. Zeek PM: Periarteritis nodosa and other forms of necrotizing angitis. *N Engl J Med* 1953; 248:764-772.
10. Hunder GG, Sheps SG, Allen GL, Joyce JW: Daily and alternate-day corticosteroid regimens in treatment of giant-cell arteritis. *Ann Intern Med* 1975; 82:613-618.

Suppurative pneumococcal inguinal adenitis

ARTHUR S. LEBOWITZ, MD, PHILIP M. TIERNO, JR, PHD

Infections with *Diplococcus pneumoniae* continue to be common, primarily involving the upper and lower respiratory tracts. Secondary involvement of other structures such as serous cavities, meninges, joints, endocardium, and blood stream may occur as complications of the primary infection. To our knowledge, however, pneumococcal infection of lymph nodes has not previously been described. We report on a patient with suppurative pneumococcal inguinal adenitis, probably sexually transmitted, occurring in the absence of a primary respiratory disorder, meningeal infection, or overt bacteremia. In this era of increased attention to infections of sexual transmission, pneumococcal infection is an additional consideration in the differential diagnosis of acute inguinal adenitis.

CASE REPORT

A previously healthy 30-year-old homosexual man sought treatment for a fever of 100°F, malaise, and an erythematous tender area in the right groin of two days' duration. He denied urinary or anorectal symptoms. Physical examination was within normal limits except for tender, non-fluctuant grouped nodes, 1.5 to 2.0 cm in diameter, just below the right inguinal ligament. The genital and rectal examinations were within normal limits except for a 4 × 2 mm shallow, clean perianal ulcer. The patient stated that the perianal lesion was an infrequent, although recurring problem, thought previously to be of herpetic origin. The patient's most recent sexual encounters had been five days and 18 days prior to the onset of the illness. Each was with a different, anonymous partner, involving mutual anal and oral penetration without condoms. He had undergone resection of an anal fistula one year previously and had been diagnosed as having pityriasis rosea four months earlier.

Laboratory findings included normal liver function tests, antibodies to hepatitis A and B, and a titer of antibodies to lymphogranuloma venereum of 1:8 by complement fixation. Herpes antibody was present at a 1:4 dilution, and a complete blood cell count revealed a white cell count of 5,300/mm^3 with 65% polymorphonuclear leukocytes, 35% lymphocytes, and an erythrocyte sedimentation rate of 43 mm/hr. A presumptive diagnosis of lymphogranuloma venereum infection was made, and treatment with tetracycline, 500 mg every six hours, was begun. It was thought that the perianal ulcerative lesion represented recurrent herpes simplex infection.

The patient was seen six days later complaining of increasing pain in the right inguinal region; he had a temperature no higher than 100°F. The perianal cutaneous lesion had resolved; again, genital and rectal examinations were unremarkable. On this visit, however, larger, matted right inguinal lymph nodes above and below the right inguinal ligament were noted. Tetracycline therapy was continued. Four days later, the patient's temperature had risen to 103°F, and he had marked pain in the right inguinal region with fluctuant, matted lymph nodes and a prominent groove sign. Needle aspiration yielded 35 cc of cream-colored, nonfoul pus.

The patient's therapy was changed, pending the Gram stain and culture results, to oral dicloxacillin and trimethoprim and sulfamethoxazole. Cultures for *Chlamydia* were obtained as well as for bacterial infection. Twenty-four hours later there was heavy growth of a Gram positive lanceolate coccus; on serologic testing, by the Neufeld (quellung) reaction, the infecting agent was identified as *Streptococcus pneumoniae* type 3. It was highly susceptible to the penicillins, but resistant to tetracycline. Therapy was changed to ampicillin, and the infection resolved. Seven days after aspiration, the white blood cell count was 4,800/mm^3, with 56% polymorphonuclear leukocytes, 44% lymphocytes, and an erythrocyte sedimentation rate of 36 mm/hr. A follow-up titer for lymphogranuloma venereum was unchanged at 1:8, and the chlamydial culture remained negative. The patient's recovery was uneventful.

DISCUSSION

Although a variety of infections caused by *S pneumoniae* has been described, no report of sexually transmitted disease or of suppurative inguinal adenitis of pneumococcal origin has been found. Duff and Gibbs[1] described two cases of acute intra-amniotic infections due to pneumococci, which were thought to be transmitted iatrogenically. In addition, *S pneumoniae* was isolated from the vaginas of two of 1,083 women cultured in a systematic attempt to study maternal and fetal morbidity and mortality in relation to bacteria found in the mother's vagina at the time of admission for labor.[2] The healthy vagina is an unlikely reservoir for pneumococci, because these organisms survive only within a pH range of 6.5 to 8.3.[1] The acidic conditions within the vagina of a non-menstruating woman would provide an unfavorable environment for either colonization or survival. In another study, the etiology of idiopathic epididymitis was investigated by culturing urethral washings and epididymal aspirates from 20 patients.[3] In one patient a pneumococcal organism was isolated from the urethra but not from the epididymis. Although the significance of this finding was not determined, transient colonization of the male urethra has, nevertheless, been reported.[3]

Pneumococci may reach the inguinal lymph nodes by at least two routes. First, the organism may disseminate from a primary site of infection, usually the respiratory tract. Overt signs of focal respiratory tract infection may not be present when bacteremia occurs.[4] Second, because the inguinal nodes drain the ipsilateral lower extremity as well as the genitalia and perineum, infection of these structures could lead to

---

From the Departments of Medicine (Dr Lebowitz) and Microbiology (Dr Tierno), New York University School of Medicine, New York.

Address correspondence to Dr Tierno, Associate Professor, Department of Microbiology, New York University School of Medicine, 560 First Ave, New York, NY 10016.

regional inguinal adenitis. Cutaneous infection with *S pneumoniae* has been described in two patients in whom the serotype matched that of the pulmonary isolate. The cutaneous locus was the site of intramuscular injection in both.[5] Although this patient had a perianal lesion, the lesion was clean, shallow, and resolved spontaneously; it was thought to be herpetic on the basis of intermittent and sporadic past occurrences. Cultures of this lesion for bacteria and herpes virus were not done, however.

At the time of presentation and at subsequent visits the possibility that the patient may have had early symptoms of acquired immune deficiency syndrome was entertained. This conclusion was thought to be unwarranted, however, because of an absence of any accepted clinical and laboratory criteria. The patient denied any of the usual prodromal symptoms such as weight loss, unexplained fever, diarrhea, or oral candidiasis. He had no other lymphadenopathy, and the presumed herpetic perianal lesion was not chronic, necrotizing, or invasive. Lastly, he manifested an adequate percentage of lymphocytes in a peripheral smear. A T-lymphocyte subset analysis was not performed.

A recent report[6] describes three homosexual patients who developed acute inguinal lymphadenitis due to group A beta-hemolytic streptococci while being followed for chronic lymphadenopathy. In two of the three patients, treatment was initiated with tetracycline for a presumptive diagnosis of lymphogranuloma venereum. The correct diagnosis was established, as with this patient, when worsening symptoms and physical findings led to surgical drainage. The authors postulated a genital infection as the primary focus.

Apparent differences, when compared with the patient described in this report, are the absence of chronic lymphadenopathy or clinical or laboratory evidence of acquired immune deficiency syndrome, and the absence of chronic herpes simplex infection.

It seems most likely that the patient described in this report had a primary asymptomatic genital infection—particularly in light of the patient's history of two recent oral-genital contacts—which spread to regional nodes. An asymptomatic upper respiratory primary infection with bacteremia and secondary infection of previously scarred inguinal lymph nodes can not be totally excluded. In conclusion, pneumococcal infection should be considered in the differential diagnosis of infectious inguinal adenitis.

REFERENCES

1. Duff P, Gibbs RS: Acute intraamniotic infection due to *Streptococcus pneumoniae. Obstet Gynecol* 1983; 61:25S-27S.
2. Beargie R, Lynd P, Tucker E, et al: Perinatal infection and vaginal flora. *Am J Obstet Gynecol* 1975; 122:31-33.
3. Furness G, Kamat MH, Kaminski Z, et al: The etiology of idiopathic epididymitis. *J Urol* 1971; 106:387-392.
4. Austrian R. The pneumococcus at Hopkins: early portents of future developments. *Johns Hopkins Med J* 1979; 144:192-201.
5. White B: *Biology of* Pneumococcus. *The Bacteriology, Biochemical and Immunological Characters and Activities of* Diplococcus pneumoniae. New York, Commonwealth Fund, 1938.
6. Ho DD, Murata GH: Streptococcal lymphadenitis in homosexual men with chronic lymphadenopathy. *Am J Med* 1984; 77:151-153.

Pseudoinfarction pattern associated with myocardial bridging of the left anterior descending artery

EDWARD T. DAVISON, MD, ROBERT I. HAMBY, MD

The role of transient systolic arterial narrowing due to myocardial bridging in producing ischemia is not clear. Because the coronary blood flow occurs predominantly in diastole, a transient systolic narrowing of the coronary artery would not be expected to impede flow. However, myocardial bridging and systolic milking have been reported to produce myocardial ischemia, myocardial infarction, and sudden death.[1-3]

We describe a patient with typical symptoms of angina, an anteroseptal wall myocardial infarction pattern on an electrocardiogram in association with myocardial bridging, a normal left ventricular angiogram, and a normal thallium-201 stress scintigram.

CASE REPORT

A 66-year-old man was asymptomatic until 1979, when symptoms of angina developed described as pressing pain on exertion and relieved by rest. He did not consult a physician until approximately six months after the onset of symptoms, at which time he saw his family doctor, who prescribed sublingual nitroglycerin and propanolol; the clinical diagnosis was angina pectoris. An electrocardiogram revealed the presence of a QS pattern in V_1-V_3 (Fig 1) suggestive of an old anteroseptal wall myocardial infarction. There was no previous electrocardiogram for comparison.

He remained on the medication until 1983, at which time he underwent cardiac cathe-

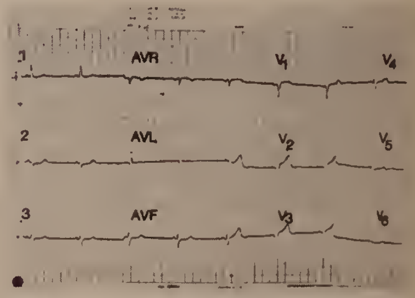


FIGURE 1. Twelve-lead electrocardiogram with QS pattern in V_1-V_3 suggestive of old anteroseptal wall myocardial infarction.

From the Department of Cardiology, Franklin General Hospital, Valley Stream (Dr Davison), and The Heart Center, St. Francis Hospital (Dr Hamby), Roslyn, New York.

Address correspondence to Dr Davison, Associate Chief, Department of Cardiology, Franklin General Hospital, 300 Franklin Ave, Valley Stream, NY 11580.

terization and angiography because of increased frequency and intensity of chest pain. Pressures in the heart chambers were normal. Pull through pressures across the outflow tract of the left ventricle did not demonstrate any evidence of aortic or subaortic disease. A selective right and left coronary arteriogram in multiple projections showed a segment of the proximal mid-left anterior descending coronary artery—about 4-cm in length—with severe systolic narrowing (more than 75% reduction of cross-sectional area) (Fig 2), but with a normal lumen during diastole (Fig 2). The other coronary arteries were normal. A left ventricular angiogram revealed normal wall motion. No evidence of perfusion deficit was seen on a stress thallium-201 scintigram. In view of the symptoms of angina and the presence of myocardial bridging, he received verapamil in addition to propanolol, and there has been significant improvement of his condition.

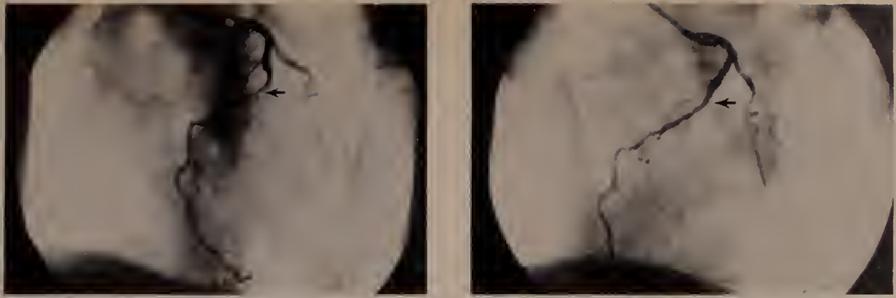


FIGURE 2. Single-frame sequential coronary angiogram of the left coronary artery in the left anterior oblique projection systole (left) and diastole (right). Note significant systolic narrowing of the mid portion of the left anterior descending coronary artery observed during each systolic phase (arrows).

DISCUSSION

The reported incidence rate of myocardial bridging has ranged from 5.4%[3] to 85.7%[4] in autopsy studies. Irvin[5] reviewed 465 consecutive coronary angiograms in a retrospective study and found that myocardial bridging was present in 35 (7.5%). Bridging was found exclusively in the left anterior descending coronary artery; the junction of the proximal and middle sections of the artery was by far the most common site. In the past, bridging was not considered significant except for a possible effect on the development of atherosclerosis in the bridged vessels.[3] However, recent evidence suggests that bridging may lead to myocardial ischemia and ischemic symptoms,[1,6] sudden death,[2] and paroxysmal atrioventricular block.[7]

Observations by Noble et al[6] in 11 patients with angina pectoris and muscular overbridging of the left anterior descending artery suggest that cyclic compression of a mural LAD artery produces ischemia. Ischemic electrocardiographic changes and abnormal lactate production or extraction with tachycardia developed in four out of five patients with 75% systolic narrowing of the left anterior descending artery. Increased circulatory catecholamines may heighten a patient's vulnerability to ventricular fibrillation[1-3,8] or may precipitate coronary spasm through an alpha-adrenergic receptor mechanism.[9] Ahmad et al[1] demonstrated exercise-induced perfusion abnormality in the thallium-201 scintigram and an impaired left ventricular ejection fraction response during exercise in patients with grade III constriction (greater than 75%). Recently, surgical intervention has proven successful in improving the symptoms of myocardial bridging.[10,11]

The presence in this patient of an infarction pattern, with a normal thallium stress test and a normal left ventricular angiogram, is of interest in view of the morphologic findings described by Morales et al[2] in three ostensibly healthy persons whose deaths occurred incidental to strenuous exercise. An unobstructed left anterior descending artery, diminished vascularity to the posterior left ventricle and ventricular septum, and morphologic evidence of patchy ischemic necrosis of the ventricular septum in different stages of healing were found. Cellular injury and tissue fibrosis were found in the anteroseptal region of the ventricular septum in two patients, consisting of fragmentations of myocardial fibers, proliferation of fibroblasts, round cell infiltration, dense collagen, and fibrous tissue deposits in some areas. It is likely that the loss of R-wave in V_1-V_3 in this patient represented myocardial fibrosis in the absence of infarction. The absence of a perfusion deficit on a thallium scan taken at rest and during stress, as well as normal wall motion on a ventriculogram and a gated pool study would suggest against a diagnosis of myocardial infarction.

Preliminary data regarding thallium-201 myocardial imaging in patients with myocardial bridging has been reported by two different groups of investigators. Thallium-201 scintigrams were reported to show normal findings in these patients in one study,[12] and objective evidence of myocardial ischemia was demonstrated in the other study.[1] The possible presence of exercise-induced coronary spasm in association with rapid systolic coronary compression at high heart rates cannot be excluded, and it may be that at the time this patient underwent testing, a spasm was not precipitated.

The effectiveness of verapamil in this patient was quite remarkable. Its negative inotropic action in reducing the force of systolic compression and its potent vasodilatory effect may be the reasons for its beneficial effects in patients with myocardial bridging. Verapamil therapy should be considered before surgical intervention by supra-arterial myotomy in patients with symptoms of myocardial bridging.

Acknowledgement. Ms Arlene Arama-Davison assisted in the preparation of this paper, and Miss Emma Hertlein and Mrs Margaret Holleran provided secretarial assistance.

REFERENCES

1. Ahmad M, Merry SL, Haibach H: Evidence of impaired myocardial perfusion and abnormal left ventricular function during exercise in patients with isolated systolic narrowing of the left anterior descending coronary artery. *Am J Cardiol* 1981; 48: 832-836.
2. Morales AR, Romanelli R, Boucek RL: The mural left anterior descending coronary artery, strenuous exercise and sudden death. *Circulation* 1980; 62:230-237.
3. Poláiek P, Králové H: Relation of myocardial bridges and loops on the coronary arteries to coronary occlusions. *Am Heart J* 1961; 61:44-52.
4. Edwards JC, Burnside C, Swarm RL, Lansing AL: Arteriosclerosis in the intramural and extramural positions of coronary arteries in the human heart. *Circulation* 1956; 3:235-241.
5. Irvin RG: The angiographic prevalence of myocardial bridging in man. *Chest* 1982; 81:198-202.
6. Noble J, Bourassa MG, Petitclerc R, Dyrda I: Myocardial bridging and milking effect of the left anterior descending artery, normal variant or obstruction? *Am J Cardiol* 1976; 37:993-999.
7. Den Dulk K, Brugada P, Braat S, Heddle B, Wellens HJJ: Myocardial bridging as a cause of paroxysmal atrioventricular block. *J Am Coll Cardiol* 1983; 1:965-969.
8. Liang B, Verrier RL, Melman J, Lown B: Correlation between circulating catecholamine levels and ventricular vulnerability during psychological stress in conscious dogs. *Proc Soc Exp Biol Med* 1979; 161:266-269.
9. Levene DL, Freeman MR: Alpha-adrenoceptor-mediated coronary artery spasm. *JAMA* 1976; 236:1018-1022.
10. Farugui AMA, Maloy WC, Felner JM, Schlant RC, Logan WD, Symbas P: Symptomatic myocardial bridging of coronary artery. *Am J Cardiol* 1978; 41:1305-1310.
11. Noble J, Grondin P, Bourassa MG: Successful periarterial muscle resection for myocardial bridging and milking effect of the left anterior descending artery. (abstract) *Am J Cardiol* 1977; 39:267.
12. Greenspan M, Iskandrian AS, Catherwood E, Kimbiris D, Bemis CE, Segal BL: Myocardial bridging of the left anterior descending artery: evaluation using exercise thallium-201 myocardial scintigraphy. *Cathet Cardiovasc Diagn* 1980; 6:173-180.

Uterine tumor due to placenta increta

SHAWKY Z.A. BADAWY, MD, JACK YOFFA, MD, BRENT FLETCHER, MD, THOMAS SIMON, MD

The phenomenon of adherent placenta can be divided into three categories: placenta accreta, in which the chorionic villi are in direct contact with the myometrium; placenta increta, in which the villi invade part of the myometrium; and placenta percreta, in which the chorionic villi reach the serosa and sometimes penetrate through it, causing internal hemorrhage.[1] The diagnosis of such conditions is usually apparent in the delivery room during the delivery of the placenta. Alternatively, the patient may seek treatment at a later date with complications related to an adherent placenta. The patient presented in this report had placenta increta that was diagnosed seven months after a premature delivery.

CASE REPORT

A 29-year-old housewife (gravida 3 para 0) was referred to the Reproductive Endocrinology Unit at Upstate Medical Center for evaluation of a history of spontaneous abortions. The first pregnancy, in 1979, occurred after three years of unprotected sexual relations and ended in spontaneous abortion at 12 weeks. The second pregnancy occurred eight months later, in 1980, and ended in spontaneous abortion at 11 weeks. The third pregnancy occurred in 1981. She experienced spotting on and off during the pregnancy. She sought treatment because of premature labor at 21 weeks gestation. She was given 10 mg of isoxsuprine intramuscularly, 12 mg of bethamethasone intramuscularly, and 100 mg of meperidine intramuscularly. In addition, she was given 1 gram of ampicillin intravenously after cervical cultures were obtained.

The uterine contractions continued in spite of maximum ritodrine therapy. An ultrasound examination showed a fetus of 21 weeks gestation. The placenta was located anteriorly and to the right of the midline. The level of amniotic fluid was diminished, but no uterine abnormalities were noted. The patient delivered a stillborn infant weighing one pound seven ounces. The placenta failed to be delivered spontaneously for 35 minutes. The patient was then given general anesthesia, and attempts to deliver the placenta failed. The placenta was then removed piecemeal by curettage, thus suggesting a diagnosis of placenta accreta. The patient suffered no complications after the procedure. A review of her condition seven months later revealed no abnormalities. She had no history of diethylstilbesterol exposure. Her cycles had been regular—every 28 days, but the periods were associated with right lower quadrant pain for one day. Physical examination revealed a healthy woman in no distress. Blood pressure was 120/80 mm Hg; height, 5ft 4in; and weight, 177 pounds. Chest, heart, and abdominal examinations did not reveal any abnormalities. She had normal thelarche and pubarche. The pelvic examination showed a normal vulva, vagina, and cervix. The body of the uterus was anteverted and mobile, with a firm well-defined mass measuring 5 × 5 cm arising from the posterior aspect of the right cornu.

The laboratory findings were as follows: tests for venereal disease, negative; fasting blood sugar, 80 mg/dL; total thyroxine, 8.3 μg/dL (normal, 2.5-12 μg/dL); T_3 resin uptake, 28.8% (normal, 25%-35%); and T_7, 2.4 μg/dL (normal, 1.2-4.2 μg/dL). Karyotyping and banding using peripheral lymphocyte cultures showed normal chromosomal components of 46XX for the patient and 46XY for the husband. Hysterosalpingogram with fluoroscopy revealed a T-shaped uterine cavity and patent fallopian tubes. The dye was seen diffusing from the right cornu of the uterus to outline a semilunar cavity (Fig 1). Ultrasound examination confirmed the presence of an echogenic mass near the fundus of the uterus and to the right of the midline. The lower pole of the mass was in contact with the upper part of the uterine cavity; it was surrounded by uterine musculature (Fig 2). The diagnosis of leiomyoma was entertained.

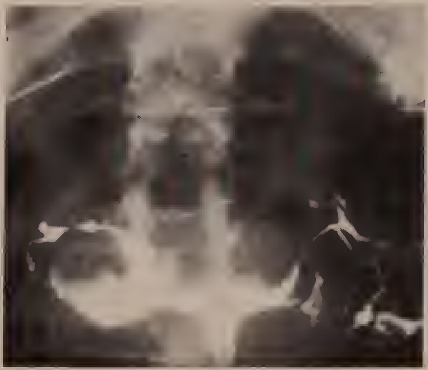


FIGURE 1. Hysterosalpingogram showing T-shaped uterine cavity and semilunar diffusion of dye from right cornu.

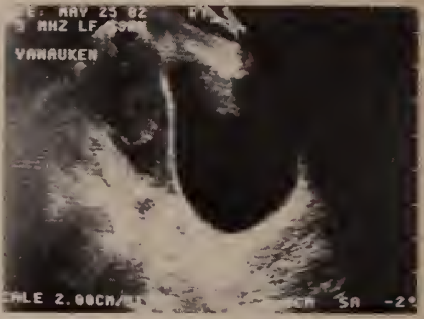


FIGURE 2. Pelvic ultrasound showing a fundal mass to the right of midline.

On November 19, 1982, the patient underwent laparotomy under general anesthesia. The right cornu of the uterus showed that the mass was well encapsulated, measuring about 5 × 5 cm, partly intramural and partly subserous on the posterior aspect of the uterus, and distinct from the course of the fallopian tube. There were no other pelvic abnormalities. After injection of diluted vasopressin solution (1 U/mL) into the uterine wall surrounding the mass, the overlying peritoneum was cut using microcautery. The mass was dissected sharply from the surrounding uterine muscle and removed. The uterine cavity was entered at the lower part of the mass. Exploration of the uterine cavity with a hemostat did not show any anomalies. The incision was closed in layers using 3-0 vicryl sutures. The patient had an uneventful recovery.

Pathologic findings. The mass was nodular and firm, with grey and yellow discoloration, weighed 13 grams, and measured 4.5 × 3.0 × 2.5 cm. The mass was softer than the usual leiomyoma, and no swirling pattern was seen. The center of the mass appeared necrotic. Microscopic examination showed smooth muscle with a localized area of necrotic tissue, within which there were ghost-like-outlines of villous structures. There was focal calcification. This was surrounded with dense fibrous tissue which revealed foreign body reaction and focal deposits of hemosiderin laden macrophages (Figs 3, 4).

DISCUSSION

The condition described in this pa-

From the Reproductive Endocrinology Unit, Department of Obstetrics and Gynecology (Drs Badawy, Yoffa, and Fletcher), Upstate Medical Center, and the Department of Pathology (Dr Simon), Crouse-Irving Memorial Hospital, Syracuse, NY.

Address correspondence to Dr Badawy, Director, Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology, SUNY-Upstate Medical Center, 750 E. Adams St, Syracuse, NY 13210.

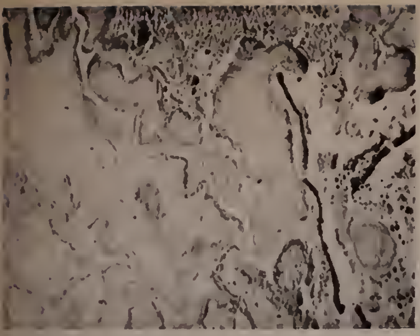


FIGURE 3. Ghost-like villi surrounded by uterine muscles. (Original magnification × 250)

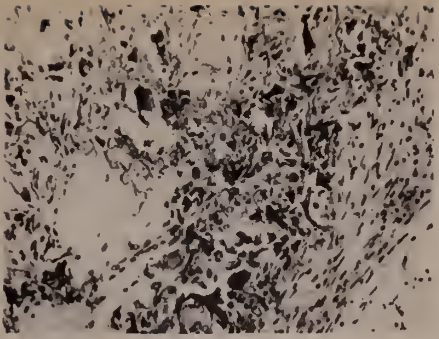


FIGURE 4. Foreign body reaction with focal calcification. (Original magnification × 250)

tient represents a rare example of calcified placenta increta, which was diagnosed by histopathologic evaluation of a circumscribed mass in the uterine fundus seven months following premature delivery. This disorder is probably a complication of the last pregnancy, in which the abnormally adherent placenta failed to separate spontaneously and was removed by curettage, suggesting a diagnosis of placenta accreta. The incidence of placenta accreta depends on whether the diagnosis is made on clinical findings or on pathologic studies and thus varies between 1 in 2,000 to 1 in 4,000 deliveries.[2] Placenta accreta results from an abnormal decidua with partial or complete absence of Nitabuch's stria, or a myometrial insult due to previous injury, or both.[3] The patient in this study had had two post abortal curettages that might have caused the insult to the endometrium, the myometrium, or both. Intrauterine synechiae have been reported following post abortal curettage.[4] These synechiae may lead to abnormal placenta formation due to deficient decidual reaction. Pregnancies that occurred following treatment of intrauterine synechiae have been associated with a high incidence of spontaneous abortion and abnormal placenta formation. Another possible cause of the disorder in this patient is the abnormal uterine configuration, namely a T-shaped uterus.[5] Patients with this abnormality have had a higher incidence of spontaneous abortion than the general population.[6]

From the hysterosalpingogram, it was documented that the dye exuded from the uterine cavity to outline the fundal mass. This might suggest either a diverticulum or an area of adenomyosis in the uterine wall in which part of the placenta developed.[7] A diagnosis of interstitial pregnancy has been excluded in this case because of the demonstration of intact fallopian tubes both by hysterosalpingogram and by examination of the adnexa during surgery.

This case illustrates one type of abnormally adherent placenta, in which the intrauterine portion was removed by curettage but the intramural portion was left untreated. The study of beta subunit human chorionic gonadotropin levels in similar patients enables the treating physician to monitor the patient and to test for foci of placenta increta and placenta percreta. In addition, the use of chemotherapy may lead to resolution of such foci of abnormally adherent placenta in the uterine wall, as has been used in the treatment of ectopic pregnancy.[8] In conclusion, the diagnosis of placenta increta leading to a uterine mass should be considered in patients with a history of placenta accreta.

REFERENCES

1. Bromham DR, Joyce DN: Antepartum haemoperitoneum due to placenta increta. Case report. *Br J Obstet Gynaecol* 1980; 87:75-76.
2. Read JA, Cotton DB, Miller FC: Placenta accreta: changing clinical aspects and outcome. *Obstet Gynecol* 1980; 56:31-34.
3. Teteris NJ, Lina AA, Holaday WJ: Placenta percreta. *Obstet Gynecol* 1976; 47:15S-18S.
4. March CM, Israel R: Intrauterine adhesions secondary to elective abortion. Hysteroscope diagnosis and management. *Obstet Gynecol* 1976; 48:422-424.
5. Jewelewicz R, Khalaf S, Neuwirth R, et al: Obstetric complications after treatment of intrauterine synechiae (Asherman's Syndrome). *Obstet Gynecol* 1976; 47:701-705.
6. Barnes AB, Colton T, Gundersen J, et al: Fertility and outcome of pregnancy in women exposed in utero to diethylstilbesterol. *N Engl J Med* 1980; 302:609-613.
7. Myerhardt MH: Spontaneous rupture of the uterus in late pregnancy accompanied by placenta accreta. *J Missouri Med Assoc* 1949; 46:177.
8. Tanaka T, Hayashi H, Kutsuzawa T, Fugimoto S, Ichinoe K: Treatment of interstitial ectopic pregnancy with methotrexate: report of a successful case. *Fertil Steril* 1982; 37:851-852.

Cardiac siderosis in thalassemia major

EDWARD C. ZAINO, MD

Thalassemias are congenital hemoglobin disorders in which there is defective production of hemoglobin chains. In homozygous beta thalassemia there is increased fetal hemoglobin with little or no A hemoglobin,[1-6] severe anemia, and eventual cardiac failure. The many blood transfusions required and increased intestinal iron absorption result in hemosiderosis.

To my knowledge there has been no previous report of the ultrastructure of the heart in such a patient in heart failure, nor has there been correlation of the effect of iron chelation with deferoxamine. In the patient described in this report, electron microscopy of the surgical myocardial biopsy was performed.

CASE REPORT

A 19-year-old man with beta thalassemia major sought treatment for swelling in the legs and shortness of breath. He had received periodic blood transfusions from the

---

Address correspondence to Dr Zaino, Director, Cooley's Anemia Transfusion Center, Mercy Hospital, 1000 North Village Ave, Rockville Centre, NY 11570.

age of three years. A splenectomy had been performed at eight years of age because of hypersplenism and increased need for blood transfusions. At 15 years of age he was receiving transfusions of two units of packed cells every four weeks whenever the hemoglobin level dropped below 10 g/dL. At 17 years of age he had signs of congestive heart failure, with dyspnea on exertion and edema of the extremities. He showed some improvement following treatment with oral diuretics and digoxin. One year later, while still on these medications, tachycardia and marked pericardial effusion developed. After multiple pericardiocenteses were unsuccessful in containing the recurring excessive pericardial fluid, a pericardectomy and left ventricular cardiac biopsy were performed. A percutaneous liver biopsy showed marked hemosiderosis within the reticuloendothelial cells and the hepatocytes.

He had been receiving continuous 12-hour subcutaneous deferoxamine therapy (2 grams daily) given with an automatic dispensing syringe for two years in an attempt to reduce the iron deposition. The initial course of treatment consisted of continuous 24-hour intravenous deferoxamine therapy at a dose of 10 grams a day for seven days. The urinary iron excretion was 75 mg on the first day and 55, 40, 35, 25, 20, and 15 mg per day on the subsequent days, producing negative iron balance. After he was discharged from the hospital following the pericardectomy, he was found to be taking the deferoxamine appropriately.

Following pericardectomy at age 18, the patient's condition improved, and he was able to return to normal physical activity. One year later marked cardiomegaly, dyspnea, cyanosis, and peripheral edema developed, prompting the current admission. A heart block was noted, and an intrathoracic pacemaker was inserted. The patient went into intractable heart failure with tachyarrhythmia and pulmonary edema, and he died.

At autopsy the heart weighed 400 grams; it was enlarged and hypertrophy was evident in all four chambers. The heart was a brown-red color and showed marked diffuse hemosiderosis under light microscopy. The Figures are electron micrographs of the heart taken a year prior to the patient's death, at the time of surgical biopsy. They show abnormalities of the mitochondria, lysosomes, and myofibrils. The mitochondria are unevenly dispersed and not present in their usual even columns between the myofibrils. They are pleomorphic: some are plump, others are elongated, narrow, and angulated. The myofibrils are frayed, fragmented, disintegrated, diminished in number, and absent in some areas. There are many abnormal giant lysosomes filled with electron-dense iron deposits within the cytoplasm.

DISCUSSION

In idiopathic hemochromatosis there is myocardial iron deposition in one third of the patients who die of congestive heart failure.[7] The iron is in the atrial and ventricular muscle[8] and in the conduction system.[9] There is 2 to 19 times the normal average amount (38 mg/100 g of dry weight) of inorganic cardiac iron.[10]

Patients with chronic anemia, leukemia, or aplastic anemia, who have received 100 units or more of blood, have shown increased cardiac iron levels with supraventricular tachycardia[8] occurring with atrial iron deposition. Premature ventricular contractions and paroxysmal atrial tachycardia are common. The electrocardiogram in cardiac siderosis usually shows low voltage and non-specific T-wave changes.[11-14]

In hemochromatosis there may be a globular heart with right and left ventricular enlargement.[7,15] The cardiac histology is similar in both primary and secondary hemochromatosis. In primary idiopathic hemochromatosis, light microscopy shows muscle hypertrophy, fragmentation and separation of fibers, disproportion of sarcoplasm to myofibrils, pyknosis, karyorrhexis, and hydropic degeneration.[10] The iron deposition has been found to be greatest in the subepicardium, subendocardium, and papillary muscles, and less marked in the mid-portion of the ventricle.[8] The iron is mainly perinuclear and yields positive results on a periodic acid-Schiff stain.[16,17] There are no reports of electron microscopic evaluations of these patients.

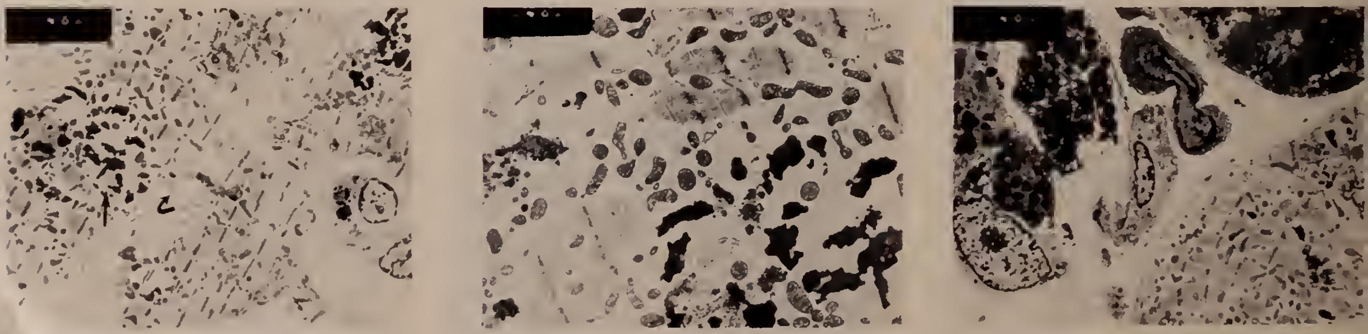
This patient showed marked cardiac siderosis on electron microscopic studies (Figs), with abnormal giant iron-laden lysosomes.

Massive iron deposition in abnormally large lysosomes within the cardiac cells, in patients with thalassemia major, is the most likely cause of heart failure. These giant inelastic metallic bodies may lead to mechanical impairment of cardiac cell contractility and function, as well as myocardial myofibril disruption and destruction. Iron-laden lysosomes have been reported in the spleen and bone marrow in thalassemia.[18]

Throughout the years of increasing positive iron balance in a child with homozygous thalassemia, the lysosomes became abnormally large and laden with iron, ballooning out into the myocardial cell. The lysosomal enzymes are dispersed into the cell causing autodigestion.

The ideal treatment of cardiac siderosis is iron removal. The only available effective iron chelator is deferoxamine. It must be given parenterally over a prolonged period of time, daily and continuously over 10 to 12 hours with a mechanical syringe strapped to the body.[19] In this patient the iron load was too large to allow sufficient iron removal in the short period of chelation therapy and to prevent progressive cardiac siderosis and death from intractable heart failure.

Acknowledgments. A grant from the Cooley's Anemia Foundation supported this study. Dr Adam Weslowski, Dr Delfin San-



Electron micrographs of the left ventricle of the heart. Left, There are large dense lysosomal hemosiderin deposits displacing myocardial fibers (straight arrow). The myofibrils are fragmented and there is mitochondrial pleomorphism (curved arrow) (original magnification × 6,000); Center, Displacement of myofibrils by dense hemosiderin bodies (original magnification × 15,000); Right, Histiocytes laden with many large iron inclusions (original magnification × 6,000).

tos, and Dr Alfred Marks helped with the clinical management of the patient.

REFERENCES

1. Bank A, Marks PA: Excess of chain synthesis relative to beta chain synthesis in thalassemia major and minor. *Nature* 1966; 212:1198.
2. Benz EJ Jr, Forget BG: The biosynthesis of hemoglobin. *Semin Hematol* 1974; 111:463–523.
3. Clegg JB, Naughton MA, Weatherall DJ: Abnormal human hemoglobin. Separation and characterization of the alpha and beta chains by chromatography and the determination of two new variants. *J Mol Biol* 1966; 19:91.
4. Heywood JD, Karon M, Weissman S: Amino acids: incorporation into alpha and beta chains of hemoglobin by normal and thalassemic reticulocytes. *Science* 1964; 146:530.
5. Heywood JD, Karon M, Weissman S: Asymmetrical incorporation of amino acids into the alpha and beta chains of hemoglobin synthesized in thalassemic reticulocytes. *J Lab Clin Med* 1965; 66:476.
6. Weatherall DJ, Clegg JB, Naughton MA: Globin synthesis in thalassemia: an in vitro study. *Nature* 1965; 208:1061.
7. Finch SC, Finch CA: Idiopathic hemochromatosis: an iron storage disease. *Medicine* 1955; 34:381.
8. Buja LM, Roberts WC: Iron in the heart. Etiology and clinical significance. *Am J Med* 1971; 51:209–221.
9. James TN: Pathology of the cardiac conduction system in hemochromatosis. *N Engl J Med* 1964; 271:92–94.
10. Keschner HW: The heart in hemochromatosis. *South Med J* 1951; 44:927–931.
11. Lewis HP: Cardiac involvement in hemochromatosis. *Am J Med Sci* 1954; 227:544.
12. Petit DW: Hemochromatosis with complete heart block with a discussion of the cardiac complications. *Am Heart J* 1945; 29:253.
13. Swan WGA, Dewar HA: The heart in hemochromatosis. *Br Heart J* 1952; 14:117.
14. Schreiber AW: Hemochromatosis and the heart. *Ann Intern Med* 1957; 47:1015.
15. Littmann D, MacMahon HE, Tussel D: Clinicopathologic conference: Cardiac manifestations in hemochromatosis. *Bull Tufts-New Eng Med Cent* 1958; 4:159.
16. Richter GW: A study of hemosiderosis with the aid of electron microscopy. With observations on the relationship between hemosiderin and ferritin. *J Exp Med* 1957; 106:203.
17. Richter GW, Bessis MC: Commentary on hemosiderin. *Blood* 1965; 24:379.
18. Zaino EC, Rossi MB, Tuan DP, Azar H: Gaucher's cells in thalassemia. *Blood* 1971; 38:457.
19. Zaino EC: *CIBA Symposium on Desferal—Desferrioxamine and Trace Metal Excretion.* New York City, New York, May 19, 1977.

Asymptomatic perforation of the small intestine by a Copper-7 intrauterine device

BURTON L. ROCHELSON, MD, WILLIAM J. MANN, MD, FRANK FROMOWITZ, MD, ROGER BAIM, MD

Perforation of the uterus is a potentially serious, albeit relatively unusual complication of the intrauterine device (IUD). Incidence of perforation ranges from 1 in 350 to 1 in 2500.[1] Actual incidence is probably higher, as many perforations may be unnoticed or unreported.

Most perforations are asymptomatic, and perforation of the uterus itself is not usually a cause of complications. However, perforation of the uterus followed by perforation of another viscus may represent significant danger to the patient, including gangrene and obstruction of the bowel. Other reported complications include dense peritoneal adhesions, appendicitis, volvulus, herniation of bowel through a closed device, bladder perforation, and uterocutaneous fistula. Presented here is a case of a Copper-7 IUD (Cu-7) perforating the uterus, lodging both within the myometrium and the lumen of the small intestine; the patient was asymptomatic. Diagnosis was made prior to laparotomy, enabling adequate psychological and medical preparation of the patient.

CASE REPORT

A 32-year old woman (gravida 3 para 2) was seen in the emergency room with heavy vaginal bleeding and passage of tissue. She was eight weeks after her last menstrual period. The patient reported having had a Cu-7 IUD inserted 18 months prior to admission, five weeks after a normal vaginal delivery. The insertion had been painful, followed by heavy vaginal bleeding. At the time of the insertion the patient was observed for an hour and was sent home on analgesic therapy. Until three weeks prior to admission she was asymptomatic except for mild constipation. At that time, the patient was one week late for her period and a pregnancy test was positive. The IUD string was visualized and removal was attempted. However, the string broke, and the IUD could not be removed.

On examination in the emergency room, the os was open and the IUD string was not visible. A diagnosis of spontaneous incomplete abortion was made. A suction curettage was performed without complications. The uterine cavity was smooth and regular, and no IUD could be palpated. A cervical dilator was placed in the uterus and a plain film of the abdomen was taken. The IUD was seen to be above the tip of the dilator.

Laparoscopy was then performed, and no IUD could be seen. What appeared to be a segment of small bowel was visualized adjacent to the posterior fundus of the uterus.

After the procedure the findings were explained to the patient. A sonogram was performed which showed the IUD partially extruding into an echogenic area, probably bowel, and perhaps partially within the myometrium (Fig 1). The bowel was prepared for surgery, and the patient was advised as to the possibility of small bowel or large bowel resection. As the area posterior to the adhesions could not be visualized, involvement of the large intestine could not be ruled out.

An exploratory laparotomy was performed. The small bowel was adherent to the posterior fundus of the uterus, with the

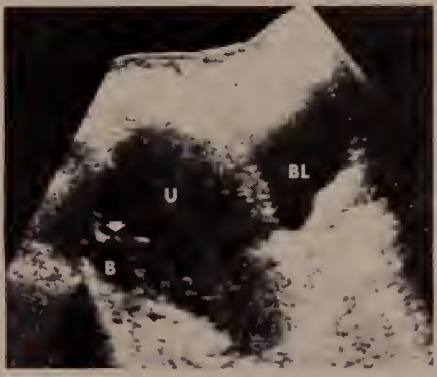


FIGURE 1. Ultrasound of IUD (arrow) perforating posterior wall of uterus: U, uterus; B, small bowel; BL, bladder.

Address correspondence to Dr. Rochelson, Fellow, Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology, SUNY at Stony Brook, Stony Brook, NY 11794.

copper of the IUD seen in between (Fig 2). On dissecting the adhesion, the tip of the long limb of the IUD was seen to come out of the fundus of the uterus. The short limb of the IUD was completely within the lumen of the small intestine with only the long limb sticking out, with surrounding inflammation of the bowel (Fig 3). The small bowel was resected and side-to-side anastomosis performed using an automatic stapling device. Figure 4 shows the Cu-7 in the lumen of the resected small intestine. The uterine perforation was identified, but there was no bleeding and no sutures were required.

The patient was treated with antibiotics and was discharged from the hospital on the sixth day after surgery without complications.

DISCUSSION

When an IUD perforates a uterus, partially or completely, it may on occasion also perforate a neighboring viscus, such as the large bowel or small bowel.

In concomitant intestinal perforation, the clinical picture varies considerably, from the complete absence of symptoms, to acute symptoms of peritonitis and intestinal obstruction, to more chronic complaints of vague abdominal pain, diarrhea and mild fever. In six reported cases of small bowel perforation by an IUD, only one patient was asymptomatic,[2] as was the woman described here. This was probably due to the dense attachment of bowel to uterus, preventing egress of the contents.

An IUD string that is not visible at the external os of the cervix suggests uterine perforation, but more often reflects upward retraction of the string or unnoted spontaneous expulsion of the IUD. However, it may on occasion be associated with uterine perforation or even intestinal perforation, as in this patient.

Insertion of an IUD should be relatively painless. Pain such as that suffered by this patient may serve as a warning sign of perforation. The breaking or tearing of the string on attempted removal should make one suspect perforation and institute immediate diagnostic tests. Sometimes, even when the string does not break, with undue traction, an IUD—which has acted as a "stopper" closing a uterine and an intestinal perforation—may be dislodged with resultant peritonitis. In this patient, uterine perforation was suggested by tearing of the string on attempted removal, unanticipated pregnancy, and absence of the IUD in the uterine cavity at curretage.

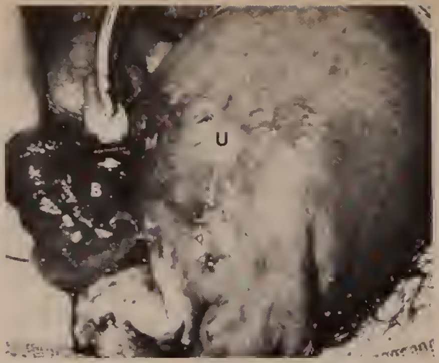


FIGURE 2. At laparotomy the Cu-7 IUD (arrow) is seen between the uterus and small intestine: U, uterus; B, small intestine.

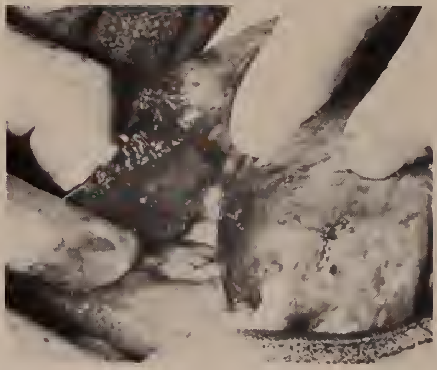


FIGURE 3. The tail of the Cu-7 emerging from the resected small intestine, with surrounding inflammatory reaction.

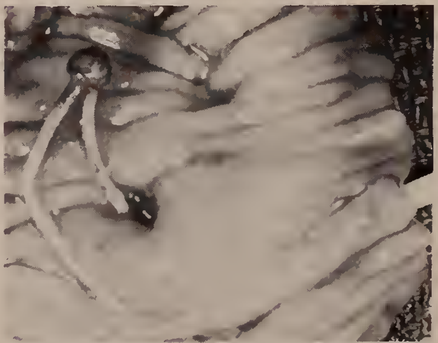


FIGURE 4. Cu-7 within lumen of small intestine.

When the string can not be seen, laparoscopy and ultrasound may be valuable aids in localizing the IUD and in preparing the patient for laparotomy and possible bowel resection. Laparoscopy could not demonstrate the exact location of the IUD in this patient partly because the IUD was within the intestinal lumen and partly because of the adhesions, which were quite dense, as is usually the case with copper-bearing devices. Sonography indicated uterine perforation and was suggestive of intestinal perforation as well. Other methods that enable an even more exact diagnosis are hysterography, barium enema examination, and small bowel studies. A plain film of the abdomen is more informative with a uterine sound in place.

Of paramount importance are a high index of suspicion and the institution of suitable diagnostic measures. Knowing the exact location of the ectopic IUD prevents potentially dangerous attempts at removal through the vagina or via surgery on an unprepared patient. Although there is no clear consensus on this point, puerperal insertion should probably be avoided, as perforation may be more likely during this period.

REFERENCES

1. Zakin D, Stern WZ, Rosenblatt R: Complete and partial uterine perforation and embedding following insertion of intrauterine devices. I. Classification, complications, mechanism, incidence, and missing string. *Obstet Gynecol Surv* 1981; 36:335-353.

2. Key TC, Kreutner AK: Gastrointestinal complications of modern intrauterine devices. *Obstet Gynecol* 1980; 55:239-244.

THE AMA AND US HEALTH POLICY SINCE 1940

By Frank D. Campion. 301 pp, illustrated. Chicago, Chicago Review Press, 1984. $25.00 (hardcover)

Over the past years I have had occasion to refer back to *A History of the American Medical Association*, published in 1947 and compiled by Morris Fishbein, MD, for years the editor of the *Journal of the American Medical Association*. When I became aware of the publication of *The AMA and US Health Policy Since 1940*, written by Frank D. Campion, I hesitated reading it because of a perception that this book would continue a chronology of events from the 1940s and not bring into focus the underlying forces and interrelated complexities of this controversial period. Being somewhat familiar with the structure, activities, and philosophies of the American Medical Association (AMA) over these past decades, I did not want a chronology, but rather hoped for a readable analysis of the times, decisions, policies, and interrelationships that have brought the AMA federation to the decade of the 1980s. I was not disappointed.

I believe the most obvious contribution of this book is simply that its style, format, readability, and fascinating overview of the four decades of American medicine are readily understandable to anyone interested in health care delivery. One does not have to possess a basic knowledge of the AMA, as such; its influence, achievements, and opinions vividly come to life within the parameters of the social, economic, and political undercurrents of the period. It should become a standard text for all students of the evolution of health care delivery in the United States since 1940. For others interested in broader concepts of social change, it fulfills a need for some understanding of why approximately 10% of the federal budget is devoted to health care costs.

Chapter 1 sets the stage for change and presents those push-and-pull forces that have changed the American health care delivery system from a predominantly private one, where only about 20% of the cost of the national medical care bill was financed by government, to a system in which private control is rapidly shrinking and governmental control proportionately increasing. The tremendous medical advances that occurred starting with World War II, surgical developments, and an analysis of forces leading to the beginning of the trend away from generalization to specialization are also presented. Most of these issues are familiar to physicians who have been practicing since the 1940s, but can now be better understood by laymen as well. The first four chapters are easy reading for anyone.

Part II (chapters 5–8) can be skimmed, but remains a good reference source for the internal organization of the AMA.

Part III forms the core of this book. It covers many of the dynamic social and medical events of the decades under discussion. While this section provides a broad sense of chronology, this is not carried out sequentially, but allows for a theme presentation of situations that developed over a relatively long period of time. Although there is considerable overlapping, the author chose to sacrifice historical chronology to some degree for the sake of clear and accurate theme presentation. This is an excellent approach, since one does not have to look back continually and piece together related happenings in varying time frames. Key discussions of great political change, choices by the people and government, the beginning of real political action, education under federal aid, Medicare, Medicaid, and finally the increasing emphasis on cost controls are brilliantly analyzed and put into perspective. The period of great AMA financial problems starting in the early 1970s and the new (and old) problems faced by the pragmatists assuming control in the late 1960s are expertly handled. This discussion culminates in chapter 20, which discusses the emergence of processes for solving these problems.

Part IV recognizes many of the events of somewhat lesser importance, but nevertheless of significance in the overall picture. They are considered chronologically and encompass such issues as medical education, scientific applications, and the general delivery of medical care.

The clearness, credibility, logic, and readability of this book greatly impressed me. It is a valuable contribution to the history of medical care and will serve both the student and casual observer of health care. It is recommended reading, for it answers the inevitable question of "What is the AMA doing for me?"

ROBERT J. O'CONNOR, MD
Deputy Executive Vice President
Medical Society of the State of New York
Lake Success, NY 11042

MAINGOT'S ABDOMINAL OPERATIONS

Eighth edition, volumes 1 and 2. Edited by Seymour I. Schwartz, MD, Harold Ellis, DM, MCH. 3,301 pp, illustrated. E. Norwalk, CT, Appleton-Century-Crofts, 1985. $195.00 (hardcover)

Rodney Maingot's book on abdominal operations has long been popular with both practicing surgeons and those in training. The eighth edition of this work only carries the Maingot name in its title, as Dr Maingot passed away two years ago. The book has developed over the decades, first as the work of one man—a British surgeon, and later with added chapters by others. Maingot's work is now mainly an American production, as the majority of the authors are from the United States. The meaningfulness of the title of the book is fading a bit with the passage of time. For example, in this edition, the recommended operation for achalasia is performed through the chest.

The contents of the book cover most abdominal surgery as practiced nowadays. Quite properly, some subjects receive less attention than before, such as gastric reservoirs, the benefit of which is now somewhat in doubt. On the other hand, prominence is given to highly selective vagotomy and operations for obesity; these are popular at present, but their future value is uncertain. The presence of multiple authors inevitably causes some degree of overlap. Overall, however, the editors have done a good job of assembling pertinent material. It is curious that in dealing with hernias, the most common ones—inguinal and femoral—have been omitted.

The work is now in two volumes (as were two previous editions). However, these volumes are heavier in weight and have smaller legends than previous editions. This makes for more difficult reading because of crowded print on the pages. In addition, the illustrations and reproductions do not seem to be as clear as in the prior editions. For the most part, the illustrations are helpful aids in learning the techniques. Few chapters have too many illustrations, but a few would benefit from more, such as the chapter on the problem of hiatus hernia and reflux.

The new editors have undertaken a difficult task in continuing the updating of a personal textbook on surgery. Only time will tell whether the passing of the original editor will influence the book's popularity. The new authors have assembled a respectable list of contributors, who are for the most part well known and highly respected surgeons. I do sorely miss the contributions from the past. The surgeons who have taken over do their best, but somehow it is not quite the same.

There is need for a book such as this, and the new editors deserve praise for their efforts in continuing the Maingot tradition, in adding new and promising areas of surgery, and in preserving descriptions of time-tested surgical procedures. The price of these volumes is substantial. However, for abdominal surgeons, these are probably two of the better reference sources available.

BJORN THORBJARNARSON, MD
Cornell University Medical Center-
New York Hospital
New York City 10021

THE PRACTICE OF EMERGENCY CARE

Second edition. By James H. Cosgriff, Jr, MD and Diann Laden Anderson, RN, MN. 652 pp, illustrated. Philadelphia, JB Lippincott Co, 1984. $34.50 (hardcover)

Dozens of textbooks describing medical care in emergency situations have been published. Most of these texts focus on the emergency aspects of traditional specialty areas. A few of the newer texts, however, have been written for the purpose of emergency medicine as a specialty and are directed to the emergency physician.

Cosgriff and Anderson have edited and largely written a new textbook for the medical team practicing emergency care that will become a standard in its own right. Although their book is actually the second edition of a 1975 text, *The Practice of Emergency Nursing*, it is essentially a new book. The new edition, with a change in title, reflects the growth, maturity, and clearer definition of the practice of emergency medicine. Although primarily written for nurses and paramedics involved in emergency care, physicians will find the book to be a comprehensive resource. It is a large, handsome book with excellent x-ray reproductions, clear illustrations, appropriate charts, and valuable management guides. These management guides present information in crisp outline form, serving both as a guideline for treatment and as an adjunct for prompt recall and review.

This second edition has 35 chapters divided into three units. Unit I focuses on general concepts, such as the Emergency Medical Service (EMS) system, legal aspects of emergency care, death, and donor organs. The psychosocial aspects of emergency care are covered in Unit II, including psychiatric treatment, death and dealing with grief, and treating sexual assault and domestic violence, which are so much a part of an emergency practice. Unit III forms the largest portion of the book and presents a variety of emergency procedures and emergency management topics. It is written in the same clear language and covers a surprising amount of normal physiology and pathophysiology. Nurses, technicians, and paramedics will find that a study of these chapters, as well as Units I and II, will provide them with a basis to guide their clinical practice. Physicians who study this text will acquire a multidisciplinary perspective of emergency medicine and will learn much to improve the quality of their practice.

This text complements many of the standard medical texts and provides information on the practice of emergency care not available elsewhere. In addition, it serves well in binding the emergency care team into a cohesive force by fostering mutual respect founded on a common knowledge base. This book should be in the libraries of all health professionals in the practice of emergency care and should be available as a reference source in emergency departments of all hospitals.

ANTHONY C. MUSTALISH, MD, MPH
Lenox Hill Hospital
New York City 10021

INFLAMMATORY BOWEL DISEASE

Edited by Tsunco Shiratori, MD and Hiroshige Nakano, MD. 457 pp, illustrated. New York, Columbia University Press, 1984. $69.50 (hardcover)

This book summarizes the presentations made at an international symposium on inflammatory bowel disease held in Japan in the fall of 1982. Most of the participants were Japanese gastroenterologists and surgeons, but there was also a small number of invited experts from the United States and United Kingdom. The symposium represented an attempt by Japanese physicians to present their research and clinical experiences with inflammatory bowel disease and to have a fruitful interchange with overseas visitors. The incidence of both Crohn's disease and ulcerative colitis in Japan is at most 10% of the incidence in other developed countries of the western world. Therefore, the experience that is conveyed is more limited than the experience of diligent investigators and clinicians in the United States. However, there are aspects of this book that are of interest.

The book is divided into sections relating to microflora, immunology, pathology, epidemiology, and treatment. One study investigated an aspect of inflammatory bowel disease that is not commonly considered, namely the coagulation status of these patients. It appears that a significant percentage of patients with this disorder have a hypercoagulable state, which may be implicated in resulting thromboembolic complications. The section of bacteriology is quite extensive, but this field of study is still an area of dubious significance in these diseases. The surgical data are somewhat repetitious. What is of interest is the tendency in Japan to try to preserve the rectum in most patients with ulcerative colitis who need surgery. At the time of this conference, Japanese surgeons had experienced a higher mortality rate than we are accustomed to, and this caused considerable comment. Experience with elemental diet and hyperalimentation are similar to that in the West, in that the benefits are usually of a transient nature. Medical treatment generally follows the western pattern, though there is one unorthodox approach, the use of intra-arterial steroids via mesenteric angiography. The results for this and for a number of other treatments are somewhat difficult to evaluate. In part this is because the concept of double-blind controlled therapeutic trials is not evident in this symposium. The extensive question and answer discussions at the end of each chapter are useful; there is ample opportunity for the experts involved to ask questions and give answers on an authoritative level.

Because of the somewhat limited scope of this symposium, it is difficult to recommend it for gastroenterologists or internists at large. For serious students of inflammatory bowel disease, there is information that is not readily available in western publications—in terms of data and some new ideas. Considerable surgical experience with rectum-saving procedures will be of interest to surgeons who are interested in inflammatory bowel disease.

CHARLES D. GERSON, MD
Mt. Sinai Hospital and Medical Center
New York City 10029

DOPPLER ULTRASOUND IN CARDIOLOGY. PHYSICAL PRINCIPLES AND CLINICAL APPLICATIONS

Second edition. By Liz Hatle, MD and Bjorn Angelsen, DrTECHN. 331 pp, illustrated. Philadelphia, Lea & Febiger, 1985. $30.00 (hardcover)

Recent years have witnessed an explosive acceleration in the use of Doppler ultrasound in non-invasive cardiac diagnosis as a complement to imaging ultrasound. Until now, however, there has not been a comprehensive text to aid the practicing cardiologist in the performance and interpretation of Doppler studies. Hatle and Angelsen have successfully filled this void with this text.

In the preface to this second edition the authors state that they intended it to be an introductory text to the use of Doppler techniques for cardiac diagnosis. Unlike the first edition, which dealt with Doppler ultrasound as an isolated technique, the second edition is directed at the combination of Doppler and two dimensional imaging techniques. This is clearly a major improvement over the first edition, as the combined technique is the dominant mode used in the United States.

The book begins with a succinct, simple, and well written introduction to Doppler principles and instrumentation. The following two chapters, dealing with the physics of blood flow and blood velocity measurement, in contrast to the introduction, are highly technical, complex, and exceedingly difficult to get through. The authors warn the reader about these chapters in the preface and suggest that the novice may omit the bulk of this material without detriment to an understanding of the rest of the book. In the next two chapters, the authors resume a less technical, more clinical approach and present a comprehensive review of pulsed and continuous wave Doppler recordings and interpretations in the normal state as well as in a variety of congenital and acquired pathologic conditions. The material is presented in an organized fashion with specific guidelines and recommendations for the assessment of each cardiac lesion. Much of the information presented by the authors is derived from their experience gained as pioneers in this growing field. The final chapters of the book are devoted to the specific vagaries of the measurement of high velocity flow with a pulsed Doppler technique and to the non-invasive measurements of cardiac output using Doppler analysis of ascending aortic flow. Each of these chapters is once again clinically oriented.

It is noteworthy that every chapter is followed by a substantial list of references. The references are current and allow the reader to research a specific interest in greater detail.

Doppler Ultrasound in Cardiology is an encyclopedic review of a rapidly developing branch of cardiovascular diagnostics. However, it is by no means a basic introductory text. The novice would have great difficulty with the material presented. In general, the presentation is readable, although at times the sentence structure seems awkward. A plethora of illustrations, diagrams, and charts are invaluable. Unfortunately, the layout is cumbersome with charts and diagrams several pages removed from the textual descriptions.

On balance, the authors have done a reputable job in amassing and digesting a vast amount of clinical and basic scientific information and presenting it in a form that provides the practicing cardiologist with guidelines for the utilization of Doppler technology. It is not intended for those with a casual interest in the subject. Rather, it is an excellent reference text and mandatory reading for the serious minded echocardiographer.

STEVEN A. FEIN, MD
Albany Medical Center Hospital
Albany, NY 12208

COMPUTED TOMOGRAPHY OF CONGENITAL BRAIN MALFORMATIONS

By Mohammad Sarwar, MD. 169 pp, illustrated. St. Louis, Warren H. Green, Inc, 1985. $42.50 (hardcover)

The author's intent is to present in an easily digestible form the method of using computed tomography to interpret congenital brain malformation, with the hope that this will allay the intimidation that is often present when one approaches this subject. This goal is in large part achieved by the book's logical organization of content, by a simplified, well depicted overview of pertinent central nervous system embryogenesis, and by well chosen, ample illustrations of both pathologic specimens and computed tomograms. The author's purpose was not to present a complete treatise on the subject, and although one may take exception to areas not covered (such as congenital brain tumors, heterotopic gray matter, and vascular malformations), one cannot fault the author's excellent distillation and understandable presentation of the material covered in this text.

The book includes a brief statistics-oriented introduction and an overview followed by a concise, well annotated description of the embryology of the central nervous system and then a chapter by chapter presentation of the major congenital brain malformations. Each of these malformations is described by a definition, and a discussion of its embryology and pathogenesis. Brief historical backgrounds are provided as well. The tables, drawings, and figures are extremely helpful in simplifying the findings, and the examples of computed tomograms are well chosen and not cluttered by an excess of annotations. The neurocutaneous syndromes are dealt with succinctly, and again the pertinent computed tomographic findings are well presented. The last chapter, titled "Miscellaneous," includes the largest number of descriptions of brain malformations and, with the exception of fetal alcohol syndrome, arachnoidal cysts, and porencephaly, these malformations are not frequently encountered. From the author's point of view, they require little comment. The index is short, representing a slightly expanded alphabetical listing of the table of contents. On the other hand, the references at the end of each chapter are pertinent and properly noted in the text.

This book is especially useful for the radiologist or clinician as a point of departure in the investigation of congenital brain abnormalities found on computed tomography and will have similar usefulness with nuclear magnetic resonance (NMR) imaging.

MICHAEL S. TENNER, MD
New York Medical College
Valhalla, NY 10595

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Reuben Peter Alderman, MD, Syracuse. Died March 9, 1985; age 89. He was a 1923 graduate of the State University of New York Upstate Medical Center. Dr Alderman was a member of the Academy of Medicine, the Onondaga County Medical Society and the Medical Society of the State of New York.

Lloyd Fred Allen, MD, Pittsford. Died February 25, 1985; age 95. He was a 1916 graduate of the State University of New York Upstate Medical Center. Dr Allen was a member of the Academy of Medicine, the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Robert H. Alterman, MD, Hollywood, Florida. Died March 3, 1985; age 77. Dr Alterman was a 1933 graduate of the Medical College of Virginia. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Robin Mowat Bannerman, MD, Buffalo. Died March 8, 1985; age 57. Dr Bannerman was a 1952 graduate of the University of Oxford School of Medicine. He was a Fellow of the American College of Physicians, and a member of the Academy of Medicine, the Society for Experimental Biology and Medicine, the Medical Society of the County of Erie and the Medical Society of the State of New York.

Jose Luis Barbosa-Saldivar, MD, New York. Died February 11, 1985; age 39. He was a 1970 alumnus of the National University of Paraguay Faculty of Medical Sciences, Asuncion. Dr Barbosa-Saldivar was a Fellow of the American College of Preventive Medicine and the American College of Physicians, and a member of the Academy of Medicine, the New York County Medical Society and the Medical Society of the State of New York.

Ivan Barnat, MD, Old Westbury. Died March 14, 1985; age 66. Dr Barnat was a 1942 graduate of the University of the Witwatersrand Medical School, Johannesburg. He was a Fellow of the American College of Surgeons, and a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Robert Joseph Booher, MD, New York. Died March 8, 1985; age 72. He was a 1938 graduate of Creighton University School of Medicine. Dr Booher was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the Academy of Medicine, the New York State Surgical Society, the Society of Surgical Oncology, the New York County Medical Society and the Medical Society of the State of New York.

Bertram Brinsley, MD, New York. Died October 31, 1984; age 59. He was a 1952 graduate of New York University College of Medicine. Dr Brinsley was a Diplomate of the American Board of Radiology, and a member of the Radiological Society of North America, Inc, the New York County Medical Society and the Medical Society of the State of New York.

Shin Ju Chang, MD, Smithtown. Died December 23, 1984; age 50. Dr Chang was a 1960 graduate of Seoul National University College of Medicine, Seoul. He was a member of the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Suffolk County Medical Society and the Medical Society of the State of New York.

Gus Chiappisi, MD, Miami, Florida. Died March 4, 1985; age 61. He was a 1956 graduate of Università degli Studi di Bologna, Facoltà di Medicina e Chirurgia, Bologna. Dr Chiappisi was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Arthur Joseph Cracovaner, MD, New York. Died March 13, 1985; age 84. He was a 1925 alumnus of Columbia University College of Physicians and Surgeons. Dr Cracovaner was a member of the New York County Medical Society and the Medical Society of the State of New York.

Mario Della Badia, MD, Long Island City. Died March 18, 1985; age 64. Dr Della Badia was a 1949 graduate of Georgetown University School of Medicine. He was a member of the American Society of Abdominal Surgeons, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Aaron Feder, MD, New York. Died March 19, 1985; age 70. He was a 1938 graduate of the University of Maryland School of Medicine. Dr Feder was a Fellow of the American College of Cardiology and the American College of Physicians, and a Diplomate of the American Board of Internal Medicine. His memberships included the Academy of Medicine, the New York Cardiological Society, the American Society of Tropical Medicine and Hygiene, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Jacob Robert Freid, MD, New York. Died March 13, 1985; age 87. He was a 1922 graduate of the State University of New York Upstate Medical Center. Dr Freid was a Fellow of the American College of Radiology and a Diplomate of the American Board of Radiology. He was a member of the New York Roentgen Society, the American Roentgen Ray Society, the Radiological Society of North America, Inc, the American Radium Society, the New York County Medical Society and the Medical Society of the State of New York.

Subramaniam Gunaratnam, MD, Cambridge. Died January 11, 1985; age 51. He was a 1961 alumnus of Ceylon Medical College, Colombo. Dr Gunaratnam was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. He was a member of the Medical Society of the County of Washington and the Medical Society of the State of New York.

Lawrence Jurkowitz, MD, College Point. Died June 21, 1984; age 47. Dr Jurkowitz was a 1958 graduate of Columbia University College of Physicians and Surgeons. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Samuel H. Kauffman, MD, Philadelphia, Pennsylvania. Died May 26, 1984; age 86. Dr Kauffman was a 1924 graduate of The General Medical College, Chicago. He was a Fellow of the American College of Anesthesiologists, and a member of the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Onondaga County Medical Society and the Medical Society of the State of New York.

Gerhard Kersten, MD, Walden. Died March 20, 1985; age 77. He was a 1935 graduate of Universität Leipzig Medizinische Fakultät, Saxony. Dr Kersten was a Fellow of the American College of Gastroenterology, and a member of the Medical Society of the County of Orange and the Medical Society of the State of New York.

John Joseph Locke, MD, Palm Bay, Florida. Died November 2, 1984; age 74. Dr Locke was a 1935 alumnus of New York Medical College, Flower and Fifth Avenue Hospitals. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Charles Terrel Lunsford, MD, Rochester. Died February 22, 1985; age 94. He was a 1920 alumnus of Howard University College of Medicine. Dr Lunsford was a member of the Academy of Medicine, the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Ernest Marcus, MD, New York. Died December 12, 1984; age 75. Dr Marcus was a 1933 graduate of Wayne State University College of Medicine. He was a member of the American Psychoanalytic Association, the American Psychiatric Association, the New York County Medical Society and the Medical Society of the State of New York.

Preston Gamaliel McLean, MD, New York. Died March 9, 1985; age 61. Dr McLean was a 1947 alumnus of the Louisiana State University School of Medicine. He was a Diplomate of the American Board of Psychiatry and Neurology, and a member of the American Psychiatric Association, the Academy of Psychoanalysis, the New York County Medical Society and the Medical Society of the State of New York.

Robert Francis McMahon, MD, Syracuse. Died June 22, 1984; age 66. Dr McMahon was a 1950 graduate of the State University of New York Upstate Medical Center, and a member of the Academy of Medicine, the Onondaga County Medical Society and the Medical Society of the State of New York.

Joseph Meiers, MD, New York. Died March 3, 1985; age 91. Dr Meiers was a 1925 graduate of Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin. He was a member of the Academy of Medicine, the American Psychiatric Association, the New York County Medical Society and the Medical Society of the State of New York.

Anthony S. Merlino, MD, Buffalo. Died January 29, 1985; age 63. Dr Merlino was a 1947 alumnus of the University of Buffalo School of Medicine. He was a Diplomate of the American Board of Internal Medicine, and a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Cavit Ozlu, MD, Rome. Died March 2, 1985; age 62. He was a 1949 alumnus of Istanbul Üniversitesi tip Fakültesi, Istanbul. Dr Ozlu was a Diplomate of the American Board of Pathology, and a member of the American Society of Clinical Pathologists, the Medical Society of the County of Oneida and the Medical Society of the State of New York.

Richard R. Panebianco, MD, Southampton. Died March 4, 1985; age 77. He was a 1932 alumnus of the University of Maryland School of Medicine. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Albert B. Peterfy, MD, New York. Died November, 1983; age 95. He was a 1913 graduate of Eotvos Lorand Tudomanyegyetem Orvostudomanyi Fakultas, Budapest. Dr Peterfy was a member of the American Psychiatric Association, the New York County Medical Society and the Medical Society of the State of New York.

Arthur A. Petronio, MD, Woodgate. Died March 1, 1985; age 67. He was a 1950 alumnus of the State University of New York Upstate Medical Center. Dr Petronio was a Diplomate of the American Board of Internal Medicine, and a member of the Academy of Medicine, the New York State Society of Internal Medicine, the Medical Society of the County of Oneida and the Medical Society of the State of New York.

Heinz J. Poetsch, MD, New York. Died March 3, 1985; age 40. He was a 1972 alumnus of Medizinische Fakultät Ludwig Maximilians-Universität München, Bayern. He was a Diplomate of the American Board of Otolaryngology, and a member of the American Academy of Otolaryngology, the New York County Medical Society and the Medical Society of the State of New York.

Philip Reichert, MD, New York. Died March 19, 1985; age 88. He was a 1923 alumnus of Cornell University Medical College. Dr Reichert was a Fellow of the American College of Physicians and the American College of Cardiology. He was a member of the Academy of Medicine, the New York County Medical Society and the Medical Society of the State of New York.

Milton Rosenberg, MD, Lake Success. Died March 1, 1985; age 62. He was a 1948 alumnus of the University of Wisconsin Medical School. He was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, a Diplomate of the American Board of Obstetrics and Gynecology, member of the Suffolk County Medical Society, and Executive Vice President of the Medical Society of the State of New York.

Morton Morris Safran, MD, Jamaica. Died January 13, 1985; age 69. He was a 1942 alumnus of Middlesex University School of Medicine. Dr Safran was a Fellow of the American Academy of Family Physicians and a Diplomate of the American Board of Family Practice. He was a member of the American Geriatrics Society, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Samuel Leib Safirstein, MD, New York. Died March 18, 1985; age 72. He was a 1939 alumnus of the Université de Paris Faculté de Médecine, Paris. Dr Safirstein was a Diplomate of the American Board of Psychiatry and Neurology, and a member of the American Psychiatric Association, the Academy of Psychoanalysis, the New York County Medical Society and the Medical Society of the State of New York.

Martin Sanders, MD, New York. Died March 19, 1985; age 58. He was a 1951 graduate of the University of Pennsylvania School of Medicine. Dr Sanders was a Diplomate of the American Board of Internal Medicine. His memberships included the International Society of Hematology, the American Rheumatism Association, the American Society of Hematology, the New York County Medical Society and the Medical Society of the State of New York.

Max Schenk, MD, New York. Died March 10, 1985; age 79. Dr Schenk was a 1930 alumnus of Schlesische-Friedrich-Wilhelms-Universität Medizinische Fakultät, Breslau. He was a Diplomate of the American Board of Radiology, and a member of the New York County Medical Society and the Medical Society of the State of New York.

Sigmund B. Silverberg, MD, Buffalo. Died February 20, 1985; age 83. Dr Silverberg was a 1926 graduate of the University of Buffalo School of Medicine. He was a member of the Academy of Medicine, the American Academy of Family Physicians, the Medical Society of the County of Erie and the Medical Society of the State of New York.

Norman Simon, MD, Scarsdale. Died March 23, 1985; age 71. Dr Simon was a 1939 alumnus of Harvard Medical School. He was a Fellow of the American College of Radiology and a Diplomate of the American Board of Radiology. His memberships included the Academy of Medicine, the New York Roentgen Society, the American Roentgen Ray Society, the Radiological Society of North America, Inc, the American Radium Society, the Society of Nuclear Medicine, the American Public Health Association, the New York County Medical Society and the Medical Society of the State of New York.

Leonard Stein, MD, Smithtown. Died March 10, 1985; age 56. He was a 1957 graduate of Rijks-Universiteit te Leiden Faculteit der Geneeskunde, Leiden. Dr Stein was a Fellow of the American College of Surgeons, the American College of Chest Physicians and the American College of Cardiology. He was a Diplomate of the American Board of Surgery and the American Board of Thoracic Surgery, and a member of the Pan American Medical Association, the Suffolk County Medical Society and the Medical Society of the State of New York.

Erich F. A. Steinthal, MD, Newburgh. Died March 3, 1985; age 93. Dr Steinthal was a 1920 graduate of Universität Heidelberg Medizinische Fakultät, Baden. He was a member of the Medical Society of the County of Orange and the Medical Society of the State of New York.

Robert James Tabacco, MD, New York. Died March 21, 1985; age 37. Dr Tabacco was a 1984 alumnus of the Mount Sinai School of Medicine. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Howard C. Taylor, MD, Southport, Connecticut. Died March 22, 1985; age 85. Dr Taylor was a 1924 alumnus of Columbia University College of Physicians and Surgeons. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Samuel Alcott Thompson, MD, Buck Hill Falls, Pennsylvania. Died March 8, 1985; age 87. He was a 1920 graduate of Jefferson Medical College of Philadelphia. Dr Thompson was a Fellow of the American College of Cardiology, the American College of Chest Physicians and the American College of Surgeons. He was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Thoracic Surgery. His memberships included the New York Society for Cardiovascular Surgery, the New York State Surgical Society, the New York Society for Thoracic Surgery, the American Association for Thoracic Surgery, the New York County Medical Society and the Medical Society of the State of New York.

Roman R. Violyn, MD, Amsterdam. Died March 5, 1985; age 80. He was a 1931 graduate of Albany Medical College of Union University. Dr Violyn was a Qualified Fellow of the International College of Surgeons, and a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Fritz Weile, MD, New York. Died February 26, 1985; age 88. Dr Weile was a 1923 alumnus of Ludwig-Maximilians-Universität Medizinische Fakultät, Bavaria. He was a member of the American Academy of Family Physicians, the American Geriatrics Society, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Dennison Young, MD, White Plains. Died March 5, 1985; age 73. He was a 1938 graduate of Tufts University School of Medicine. Dr Young was a Fellow of the American College of Physicians, and a Diplomate of the American Board of Internal Medicine. He was a member of the Academy of Medicine, the American Academy of Pediatrics, the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Burton L. Zohman, MD, Brooklyn. Died Feburary 23, 1985; age 86. He was a 1922 graduate of the State University of New York Downstate Medical Center. Dr Zohman was a Fellow of the American College of Cardiology, the American College of Physicians and the American College of Chest Physicians. He was a Diplomate of the American Board of Internal Medicine. His memberships included the New York Cardiological Society, the New York State Society of Internal Medicine, the International Society of Internal Medicine, the Pan American Medical Association, the Academy of Psychosomatic Medicine, the American Geriatrics Society, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089–1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), an abbreviated title of no greater than 40 spaces for use as a running foot, and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear on the text. When there are six or fewer authors, all should be listed; where there are seven or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

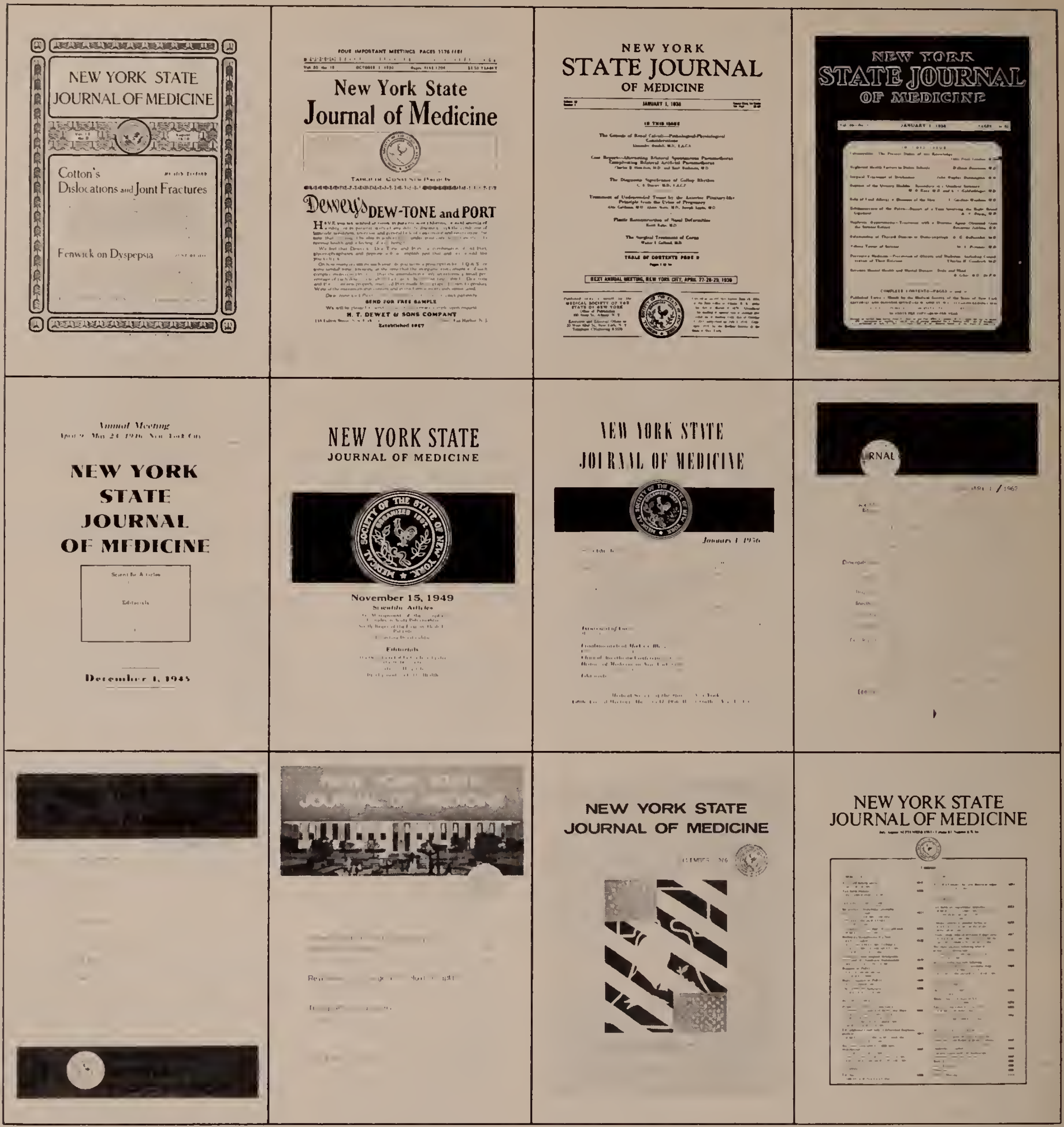
1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. McGuigan JE. Peptic ulcer. In: Petersdorf RG, Adams RD, Braunwald E, Isselbacher KJ, Martin JB, Wilson JD, eds. *Harrison's Principles of Internal Medicine*. New York, McGraw-Hill, 1983, pp 1697–1712.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Alan Blum, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

Cover to cover . . . . always good reading.


NEW YORK STATE JOURNAL OF MEDICINE
Cotton's Dislocations and Joint Fractures
Fenwick on Dyspepsia
New York State Journal of Medicine
Dewey's DEW-TONE and PORT
NEW YORK STATE JOURNAL OF MEDICINE
JANUARY 1, 1936
NEXT ANNUAL MEETING, NEW YORK CITY, APRIL 27-28-29, 1936
NEW YORK STATE JOURNAL OF MEDICINE
NEW YORK STATE JOURNAL OF MEDICINE
December 1, 1945
NEW YORK STATE JOURNAL OF MEDICINE
November 15, 1949
NEW YORK STATE JOURNAL OF MEDICINE
NEW YORK STATE JOURNAL OF MEDICINE
NEW YORK STATE JOURNAL OF MEDICINE

In 1985, the *New York State Journal of Medicine* promises to provide timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news. The journal will make a welcome gift to a colleague or student in the United States or abroad.

A free copy of the July 1985 issue, a special issue on smoking, will be included with each subscription while the supply lasts.

SUBSCRIPTION ORDER FORM Clip and mail

Circulation Department
New York State Journal of Medicine
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042

Name____________________

Address____________________

____

	1 Year	2 Years	3 Years
U.S. & Canada	☐ $25	☐ $45	☐ $60
Foreign	☐ $30	☐ $55	☐ $75

Payment required with order.

Classified Advertising

New York State Journal of Medicine

GENERAL INFORMATION

All classified advertisements are payable in advance, except for government agencies and advertising agencies; a 15% commission to recognized advertising agencies.

Frequency: Monthly
Mailing date: 15th of the month
Closing date: 30 days before mailing date

Circulation: 27,000 (mainly physicians & medical libraries)

DISPLAY ADVERTISING

	1 time	*6 time*	*12 time*	*Width*		*Depth*
1/4-page	$450	$400	$350	3-1/2″	×	4-7/8″
1/6-page	210	190	170	2-1/8″	×	4-7/8″
1/8-page	190	170	150	3-1/2″	×	2-3/8″
1/16-page	95	85	75	3-1/2″	×	1-1/8″
1/18-page	70	60	50	2-1/8″	×	1-1/2″

PARAGRAPH ADVERTISING

The first several words are set in bold face caps.

Rates: $25.00 per insertion for 50 words or less; additional words are 30 cents each. On request, Dept. numbers will be assigned by the Journal for replies to be forwarded to the advertiser. Dept. numbers are 50 cents per insertion. There is no frequency discount for paragraph ads.

Counting Words: Two initials, each abbreviation, figures consisting of 5 numerals or less, are counted as separate words. For replies, your name and address, or telephone number should appear at the end of the ad and be counted in the total number of words.

For further information, please call the Advertising Department at 516-488-6100.

CLASSIFIED ADVERTISING ORDER FORM

New York State Journal of Medicine
Attn: Classified Advertising
420 Lakeville Rd., P.O. Box 5404
Lake Success, N.Y. 11042

Date____________________

Name____

Address____

City/State/Zip____

Payment enclosed: $____________

Number of Insertions: ____________

Assign Dept. Number: ____________

Please make check payable to New York State Journal of Medicine.

1986 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
New York Hilton, New York City

Maintaining the Quality of Health Care into the 21st Century

FRIDAY, MARCH 7

10:00 a.m. - 1:00 p.m.	Cardiovascular Diseases Gastroenterology/Alcoholism Obstetrics/Gynecology Neurosurgery Physical Medicine & Rehabilitation
2:00 p.m. - 5:00 p.m.	Neurology Pediatrics/Alcoholism Chest Diseases Medical Legal & Workers Comp./Occupational Medicine Medical Liability Mutual Insurance Co.

SATURDAY, MARCH 8

10:00 a.m. - 1:00 p.m.	Ophthalmology (All Day) Internal Medicine (All Day) Hand Surgery/Anesthesiology Urology/Dermatology Psychiatry
2:00 p.m. - 5:00 p.m.	Ophthalmology Internal Medicine Otolaryngology Hand Surgery/Anesthesiology Psychiatry

SUNDAY, MARCH 9

10:00 a.m. - 1:00 p.m.	Allergy & Immunology General Surgery Women's Medical Society of N.Y./Family Practice Pathology

* * *

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis.

* * *

Technical Exhibits

Angina comes in many forms...

So does SORBITRATE®

(ISOSORBIDE DINITRATE)

Unsurpassed flexibility in nitrate therapy.

© 1985 ICI AMERICAS INC.

See following page for brief summary of prescribing information.

SORBITRATE®
(ISOSORBIDE DINITRATE)

Please consult full prescribing Information before use. A summary follows:

INDICATIONS AND USAGE: SORBITRATE (isosorbide dinitrate) is indicated for the treatment and prevention of angina pectoris. All dosage forms of isosorbide dinitrate may be used prophylactically to decrease frequency and severity of anginal attacks and can be expected to decrease the need for sublingual nitroglycerin.

The sublingual and chewable forms of the drug are indicated for acute prophylaxis of angina pectoris when taken a few minutes before situations likely to provoke anginal attacks Because of a slower onset of effect, the oral forms of isosorbide dinitrate are not indicated for acute prophylaxis

CONTRAINDICATIONS: SORBITRATE is contraindicated in patients who have shown purported hypersensitivity or idiosyncrasy to it or other nitrates or nitrites. Epinephrine and related compounds are ineffective in reversing the severe hypotensive events associated with overdose and are contraindicated in this situation.

WARNINGS: The benefits of SORBITRATE during the early days of an acute myocardial infarction have not been established. If one elects to use organic nitrates in early infarction, hemodynamic monitoring and frequent clinical assessment should be used because of the potential deleterious effects of hypotension.

PRECAUTIONS: General: Severe hypotensive response, particularly with upright posture, may occur with even small doses of SORBITRATE The drug should therefore be used with caution in subjects who may have blood volume depletion from diuretic therapy or in subjects who have low systolic blood pressure (eg, below 90 mmHg) Paradoxical bradycardia and increased angina pectoris may accompany nitrate-induced hypotension. Nitrate therapy may aggravate the angina caused by hypertrophic cardiomyopathy

Marked symptomatic, orthostatic hypotension has been reported when calcium channel blockers and organic nitrates were used in combination. Dose adjustment of either class of agents may be necessary.

Tolerance to this drug and cross-tolerance to other nitrates and nitrites may occur Tolerance to the vascular and antianginal effects of isosorbide dinitrate or nitroglycerin has been demonstrated in clinical trials, experience through occupational exposure, and in isolated tissue experiments in the laboratory The importance of tolerance to the appropriate use of isosorbide dinitrate in the management of patients with angina pectoris has not been determined However, one clinical trial using treadmill exercise tolerance (as an end point) found an 8-hour duration of action of oral isosorbide dinitrate following the first dose (after a 2-week placebo washout) and only a 2-hour duration of effect of the same dose after 1 week of repetitive dosing at conventional dosing intervals. On the other hand, several trials have been able to differentiate isosorbide dinitrate from placebo after 4 weeks of therapy and, in open trials, an effect seems detectable for as long as several months.

Tolerance clearly occurs in industrial workers continuously exposed to nitroglycerin. Moreover, physical dependence also occurs since chest pain, acute myocardial infarction, and even sudden death have occurred during temporary withdrawal of nitroglycerin from the workers In clinical trials in angina patients, there are reports of anginal attacks being more easily provoked and of rebound in the hemodynamic effects soon after nitrate withdrawal. The relative importance of these observations to the routine, clinical use of isosorbide dinitrate is not known. However, it seems prudent to gradually withdraw patients from isosorbide dinitrate when the therapy is being terminated, rather than stopping the drug abruptly.

Information for Patients: Headache may occur during initial therapy with SORBITRATE. Headache is usually relieved by the use of standard headache remedies or by lowering the dose and tends to disappear after the first week or two of use

Drug Interactions: Alcohol may enhance any marked sensitivity to the hypotensive effect of nitrates

Isosorbide dinitrate acts directly on vascular smooth muscle; therefore, any other agent that depends on vascular smooth muscle as the final common path can be expected to have decreased or increased effect depending on the agent

Carcinogenesis, Mutagenesis, Impairment of Fertility: No long-term studies in animals have been performed to evaluate the carcinogenic potential of this drug. A modified two-litter reproduction study in rats fed isosorbide dinitrate at 25 or 100 mg/kg/day did not reveal any effects on fertility or gestation or any remarkable gross pathology in any parent or offspring fed isosorbide dinitrate as compared with rats fed a basal-controlled diet.

Pregnancy Category C: Isosorbide dinitrate has been shown to cause a dose-related increase in embryotoxicity (increase in mummified pups) in rabbits at oral doses 35 and 150 times the maximum recommended human daily dose There are no adequate and well-controlled studies in pregnant women. SORBITRATE should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when SORBITRATE is administered to a nursing woman.

Pediatric Use: The safety and effectiveness of SORBITRATE in children has not been established

ADVERSE REACTIONS: Adverse reactions, particularly headache and hypotension, are dose-related In clinical trials at various doses, the following have been observed.

Headache is the most common (reported incidence varies widely, apparently being dose-related, with an average occurrence of about 25%) adverse reaction and may be severe and persistent Cutaneous vasodilation with flushing may occur Transient episodes of dizziness and weakness, as well as other signs of cerebral ischemia associated with postural hypotension, may occasionally develop (the incidence of reported symptomatic hypotension ranges from 2% to 36%) An occasional individual will exhibit marked sensitivity to the hypotensive effects of nitrates and severe responses (nausea, vomiting, weakness, restlessness, pallor, perspiration, and collapse) may occur even with the usual therapeutic dose. Drug rash and/or exfoliative dermatitis may occasionally occur Nausea and vomiting appear to be uncommon Case reports of clinically significant methemoglobinemia are rare at conventional doses of organic nitrates The formation of methemoglobin is dose-related and, in the case of genetic abnormalities of hemoglobin that favor methemoglobin formation, even conventional doses of organic nitrate could produce harmful concentrations of methemoglobin.

DOSAGE AND ADMINISTRATION: For the treatment of angina pectoris, the usual starting dose for sublingual SORBITRATE is 2.5 to 5 mg, for chewable tablets, 5 mg; for oral (swallowed) tablets, 5 to 20 mg, and for controlled-release forms, 40 mg

SORBITRATE should be titrated upward until angina is relieved or side effects limit the dose In ambulatory patients, the magnitude of the incremental dose increase should be guided by measurements of standing blood pressure.

The initial dosage of sublingual or chewable SORBITRATE for prophylactic therapy in angina pectoris patients is generally 5 or 10 mg every 2 to 3 hours. Adequate controlled clinical studies demonstrating the effectiveness of chronic maintenance therapy with these dosage forms have not been reported

SORBITRATE in oral doses of 10 to 40 mg given every 6 hours or in oral controlled-release doses of 40 to 80 mg given every 8 to 12 hours is generally recommended The extent to which development of tolerance should modify the dosage program has not been defined *The oral controlled-release forms of isosorbide dinitrate should not be chewed*

DOSAGE FORMS AVAILABLE: Sublingual Tablets (2.5, 5, 10 mg), Chewable Tablets (5, 10 mg), Oral Tablets (5, 10, 20, 30, 40 mg); Sustained Action Tablets (40 mg)

STUART PHARMACEUTICALS
Division of ICI Americas Inc.
Wilmington, DE 19897

STR-2282

Don't let your lungs go to pot.

Smoking marijuana is a lot more dangerous than you think. And a lot less cool.

AMERICAN
LUNG
ASSOCIATION®
The Christmas Seal People®

Space contributed by the publisher as a public service

continued from p 8A

Cat 1 credits. Boston. Info: Department of Continuing Education, Harvard Medical School, Boston, MA 02115. Tel: (617) 732-1525.

MICHIGAN

Sept 26-28. **Implant Surgery for the Hand and Upper Extremity.** Grand Rapids. 18 cat 1 credits. Info: American Society for Surgery of the Hand, 3025 South Parker Road, Suite 65, Aurora, CO 80014. Tel: (303) 755-4588.

NEW MEXICO

Sept 12-14. **2nd Santa Fe Colloquium on Cardiovascular Therapy—Contemporary Treatment of Angina Pectoris: Medical Therapy, Percutaneous Angioplasty and Coronary Bypass Grafting.** Santa Fe. Info: American College of Cardiology, Extramural Programs Department, 9111 Old Georgetown Rd, Bethesda, MD 20814. Tel: (301) 897-5400, ext 230.

TEXAS

Sept 5-7. **Second National Conference On Diet, Nutrition and Cancer.** Houston. Info: American Cancer Society, 90 Park Ave, New York, NY 10016. Tel: (212) 599-8200.

Sept 6-8. **Survival of Quality Medical Care in a Competitive Arena.** 12 Cat 1 credits. **Protecting Your Medical Practice: Planning for Payment Changes.** 12 Cat 1 credits. **Malpractice Prophylaxis.** 13 Cat 1 credits. San Antonio. Info: Jeanette Stone, Southern Medical Association, PO Box 190088, Birmingham, AL 35219-0088. Tel: (800) 423-4992.

BACK COPIES NEW YORK STATE JOURNAL OF MEDICINE

For a limited time, back copies may be purchased at the following rates plus postage.

1971-1980 . . . $2.00
1981-1983 . . . 3.00

Exception: for the special issue of December 1983 which costs $5.00.

Send your order to:
Circulation Dept., New York State Journal of Medicine, 420 Lakeville Road, P.O. Box 5404, Lake Success, NY 11042.

Inquiries: (516) 488-6100, Circulation Dept.

MISCELLANEOUS

PROFESSIONAL MISCONDUCT ATTORNEYS. WILLIAM L. WOOD, JR., formerly Executive Director of the New York State Office of Professional Discipline and ANTHONY Z. SCHER, formerly Director of Prosecutions, announce the opening of their law office, WOOD & SCHER. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct proceedings, malpractice, licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. WOOD & SCHER, Attorneys at Law, One Chase Road, Scarsdale, New York 10583, Telephone 914-723-3500.

DOCTOR, YOU CAN'T BEAT the quality or the price! Holter Monitor Scanning Service. Physician owned, trained and supervised. No contracts to sign. We can arrange for lease/purchase of Holter equipment. New dual channel recorders with test cables $1,550. Now using UP Service for faster turn around time. DCG Interpretation (313) 879-8860.

ARCHITECTURE & PLANNING, Comprehensive services from analysis of alternative office locations through design and construction. Experienced in both new construction and renovation. Lewis & Gould (212) 807-6588.

MISCELLANEOUS—CONT'D

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuariat certification and employee statements of participation—partnership agreements and professional incorporation—No insurance required—references upon request—Jack Wachstock, PC, 3000 Marcus Ave., Lake Success, N.Y. 11530 (516) 437-4848.

MEDICAL EDITOR/WRITER: Seeks freelance assignments. Experienced in clinical/socio-economic subjects, MD manuscripts preparation, article research, presentations, graphics, copyediting, proofreading. Call (212) 861-7280.

ARCHITECTURAL DESIGN AND CONSTRUCTION: Designers, builders of custom professional offices. Consulting services available. Whether you're opening a new office, relocating, remodeling or expanding an existing office in New York City area and adjacent New Jersey counties. Donald Young, Inc. Tel: (718) 948-5191.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate, with no points, fees or charges of any kind. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

ACCOUNTING AND TAX SERVICES—Professional accounting services including preparation of individual income tax and business tax returns, consultation on all tax and accounting matters. Call anytime. Marvin J. Siegel, CPA, (718) 275-0858. If no answer leave message. References available upon request.

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

Don't let your lungs go to pot.

Smoking marijuana is a lot more dangerous than you think And a lot less cool

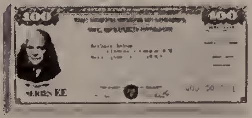
AMERICAN LUNG ASSOCIATION
The Christmas Seal People®

Space contributed by the publisher as a public service

Used with permission from the Charles Martin Conlon Collection owned by The Sporting News.

SOME OF THE GREATEST THINGS IN AMERICA NEVER CHANGE.

SOME DO.



Now Paying 9.49%.

In the 1800's, baseball was a child's sandlot game. Then came the curve ball, the slide, the crowds and the legends—like Babe Ruth. Baseball had become our national pastime.

Some things never change.

But one great American tradition *has* changed—U.S. Savings Bonds. Now Savings Bonds pay higher variable interest rates like money market accounts. Currently, Bonds are paying 9.49%.

Just hold Savings Bonds for five years and you get the new variable interest rates. Plus, you get a guaranteed return. That means you can earn a lot more, but never less than 7.5%.

You can purchase Bonds at almost any financial institution, or easier yet, through the Payroll Savings Plan where you work.

Buy U.S. Savings Bonds. Like baseball, they're another part of our proud heritage. For more information, call toll-free 1-800-US-BONDS.

U.S. SAVINGS BONDS
Paying Better Than Ever

Variable rates apply to Bonds purchased on and after 11/1/82 and held at least five years. Bonds purchased before 11/1/82 earn variable rates when held beyond 10/31/87. Bonds held less than five years earn lower interest.

A public service of this publication.

Angina Protection with Benefits for a Lifetime

ONCE-DAILY CONTROL WITH HEART-SAVING BENEFITS

By reducing heart rate and cardiac contractility, INDERAL LA helps protect the heart from the potentially serious and debilitating consequences of ischemia. A highly effective antianginal agent for around-the-clock control of symptoms, INDERAL LA also provides cardiovascular protection for a sense of security in the years ahead.

PROTECTION AND EXPERIENCE NO CALCIUM BLOCKER CAN MATCH

Unlike calcium blockers, INDERAL LA—either alone or with a nitrate—is recommended for early treatment of angina in the majority of patients. Equally important, INDERAL LA delivers the proven performance and safety profile of INDERAL tablets—confirmed by millions of patients during 16 years of clinical use.

START WITH 80 MG ONCE DAILY

Dosage may be increased to 160 mg once daily, as needed, to achieve optimal control. INDERAL LA should not be used in congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma. Please see next page for further details and brief summary of prescribing information.

ONCE-DAILY **INDERAL LA**

(PROPRANOLOL HCl) *LONG ACTING CAPSULES*

Ayerst.

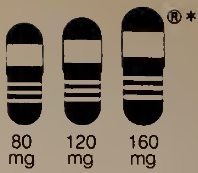
The appearance of INDERAL LA capsules is a registered trademark of Ayerst Laboratories

JUST ONCE EACH DAY FOR SIMPLIFIED CORE THERAPY IN ANGINA

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES



Just once each day...

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*
INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules.

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established. Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain. Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use Effects on plasma volume appear to be minor and somewhat variable INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction. Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential The significance of the membrane action in the treatment of arrhythmias is uncertain

The mechanism of the antimigraine effect of propranolol has not been established Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved. In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction. Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

Propranolol is not significantly dialyzable.

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock; 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)— PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g., dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General:* Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL is not indicated for the treatment of hypertensive emergencies.

Beta adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure

Clinical Laboratory Tests: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose There are no adequate and well-controlled studies in pregnant women INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman

Pediatric Use: Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular: bradycardia, congestive heart failure, intensification of AV block, hypotension, paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type.

Central Nervous System: lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances; hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics.

Gastrointestinal. nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic: pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory: bronchospasm.

Hematologic: agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily. If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

*The appearance of INDERAL LA capsules is a registered trademark of Ayerst Laboratories

8833 384

Ayerst.
AYERST LABORATORIES
New York, N.Y. 10017

FOR PHYSICIANS AND SURGEONS WITH A PART-TIME EXPOSURE ONLY

Now offering Medical Malpractice Insurance on an ***occurrence basis*** at drastically reduced rates thru Frontier Insurance Co., Monticello, NY.

With the tail on claims made policies, you might lose your shirt.

Call or write for further information and application.

MEDICAL MQ QUADRANGLE

Medical Quadrangle, Inc.
P. O. Box 456
318 Bedford Road
Bedford Hills, New York 10507
914 666-0170
212 829-3654
"Specializing in Medical Liability Insurance"

Need A Temporary Physician?
During: vacations, CME's, illness, recruiting.
Want Free Time While You Practice Medicine?
Join CompHealth's Locum Tenens Physician Group.
For further information, call 412-741-3310.

CompHealth A Physician Group
WILSON ROSS, Regional Administrator
114 Centennial Ave., Sewickley, PA 15143

NATIONAL EMERGENCY SERVICES, INC. (N.E.S.)
Directorships, full- and part-time emergency department opportunities are available throughout New York State and the country for primary care physicians with ER experience. Enjoy a variety of practice settings, competitive remuneration, paid malpractice insurance, and flexible scheduling. For information and application materials contact the recruitment department at (516) 365-4010, (Outside N.Y. State 1-800-645-4848) or send C.V. to N.E.S., One Hollow Lane, Suite 304, Lake Success, N.Y. 11042.

Index to Advertisers

Ayerst Laboratories 16A,17A,18A
Certified Medical Representatives Institute .... 3rd Cover
Classified Advertising 5A,15A,19A
Knoll Pharmaceuticals 2A,3A,4A
Eli Lilly and Company .... 2nd Cover
Marion Laboratories 9A,10A
McGraw Hill Publications 6A
Medical Quadrangle 19A
Stuart Pharmaceuticals 13A,14A
Upjohn Company .... 4th Cover
U.S. Army Reserve 20A
Winthrop Breon 7A,8A

SURGEONS

Have you considered the U.S. Army Reserve?

1775

☐ SERVE IN THE ARMY RESERVE MEDICAL DEPARTMENT AS A COMMISSIONED OFFICER

☐ CME PROGRAMS FUNDED BY THE U.S. ARMY

☐ FLEXIBLE HOURS AND LOCATIONS AS NOT TO INTERFERE WITH YOUR PRACTICE

☐ NON-CONTRIBUTIVE RETIREMENT ANNUITY

☐ A CHANCE TO BROADEN YOUR MEDICAL EXPERTISE

CALL COLLECT
(718) 352-5850

USAR AMEDD Procurement
P.O. Box 4649
Bay Terrace, NY 11360-4649

NYMS-8

Name: ______________________________

Specialty: ______________________________

Address: ______________________________

Business Phone: ______________ Home Phone: ______________

Best Time to Call: ______________________________

Medical Alert

®

Do You Know This Vital Sign?

To professionals in medicine and pharmacy, this is the mark of a distinguished member of the healthcare community—the Certified Medical Representative.

This achievement signifies the successful completion of hundreds of hours of advanced study in pharmacology, physiology, microbiology, biochemistry and other medically-related courses.

CMR candidates learn through home study. Twice yearly, rigorous examinations are given at 50 major universities throughout the country. The completion of this comprehensive educational program is an independent, unbiased way of attaining certification. The program is accredited by the American Council on Education, and is recognized industry-wide.

The results—Certified Medical Representatives—have the vital signs of the medical professionals they serve: dedication and determination, knowledge and understanding, competence and consideration.

Write or call for a free catalog and more information today.

The Certified Medical
Representatives Institute, Inc.
4316 Brambleton Avenue, S.W., Dept. SNY 8-5
Roanoke, Virginia 24018
703/989-4596

Know Your CMR

New

Motrin® 800 TABLETS mg

ibuprofen

Extra strength

Convenience

Economy

Upjohn

The Upjohn Company
Kalamazoo, Michigan 49001 USA

© 1985 The Upjohn Company

J-5491 June 1985

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

SEPTEMBER 1985 • Volume 85, Number 9

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
OCT 11 1985

Contents

COMMENTARIES

Immunosuppression for renal allograft recipients: the role of cyclosporine 523a
DAVID E.R. SUTHERLAND, MD

Multiple pregnancies: spontaneous and induced 525
ROBERT LANDESMAN, MD,
BRIJ B. SAXENA, PhD, Dsc

Rounded atelectasis 527
ALAN D. HILGENBERG, MD

The application of non-invasive diagnostic techniques to the pediatric patient 528
ARVIN E. ROBINSON, MD

Reflecting on the lectures of Professor Budd 529
GORDON W. DOUGLAS, MD

RESEARCH PAPERS

Cyclosporine: preliminary experience in 79 patients with renal transplants 531
V. A. TELLIS, MD, A. J. MATAS, MD,
D. GLICHLICK, MD, T. QUINN, PA,
R. SOBERMAN, MD, R. WEISS, MD,
F. J. VEITH, MD

Increased survival time of renal allograft recipients 534
BARBARA J. BYER, RN, WENDY E. HOY, MD, ALLYN G. MAY, MD,
DAVID W. KNUTSON, MD

THE CPC

A 45-year-old man with weight loss, fever, chills, and diarrhea 539

HISTORY

Medical education in New York City in 1866–1867—a student's notebook of Professor Charles Budd's lectures on obstetrics at New York University—Part II 548
BERT HANSEN, PhD

CASE REPORTS

Perinatal aspects of high multiple gestation 560
JALLEH VAFAI, MD,
DONALD L. SHAPIRO, MD

Rounded atelectasis 563
GIL HAUER SANTOS, MD,
HELEN MOREHOUSE, MD

Acute cholecystitis due to *Aeromonas hydrophila* 564
WILLIAM J. PARSONS, MD

Neonatal hydrops of the gallbladder: evaluation by cholescintigraphy and ultrasonography 565
JOHN B. AMODIO, MD, EILEEN FONTANETTA, MD,
MORRIS COHEN, MD, AMIEL Z. RUDAVSKY, MD,
MURRAY J. ROSENZWEIG, MD

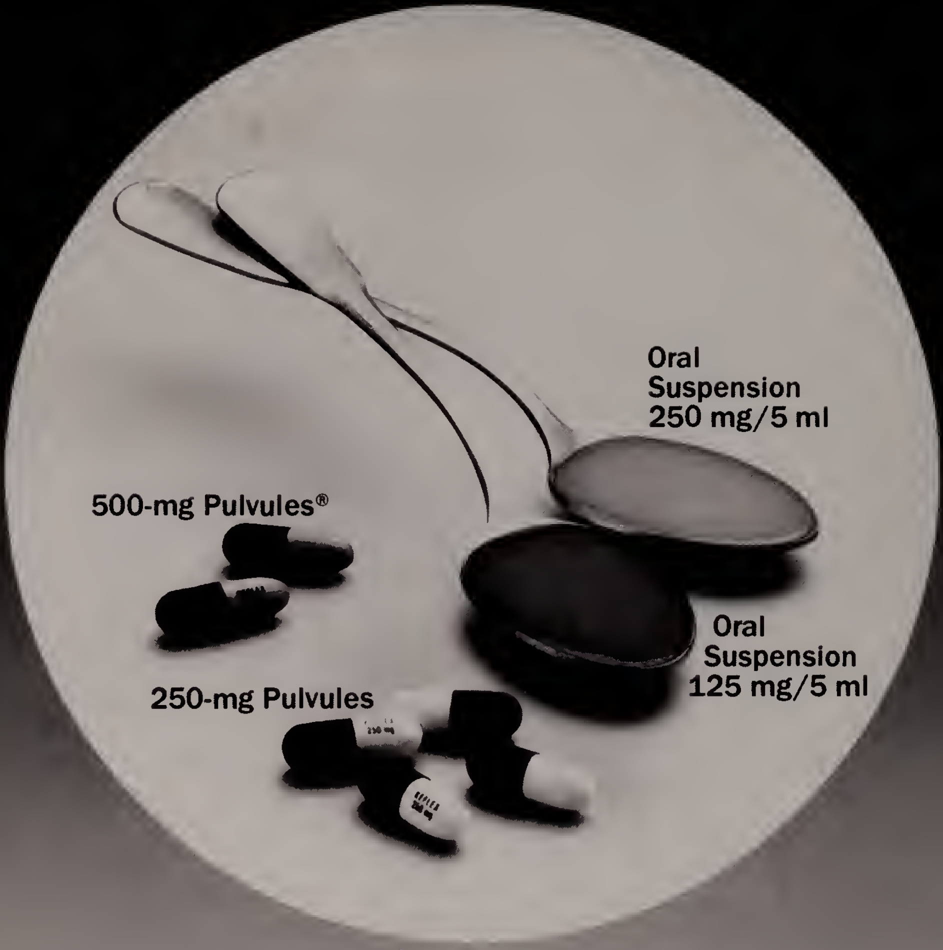
MSSNY MALPRACTICE FUND HONOR ROLL 1b

OBITUARIES 568
GUIDELINES FOR AUTHORS 571
MEDICAL MEETINGS AND LECTURES 14a

M. D. S.

Library of the College Physician
of Philadelphia Dec. 85
19 S. 22nd St.
Phila, PA 19103

Easy To Take



Keflex®
cephalexin

Additional information available to the profession on request.

DISTA
420113

Dista Products Company
Division of Eli Lilly and Company
Indianapolis, Indiana 46285
Mfd. by Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
ELIZA H. CALDWELL, MD
URSULA A. MATULONIS (Medical Student)
FRANK C. MAXON, JR, MD
RICHARD B. NOLAN, MD
JOHN T. PRIOR, MD

Editor	ALAN BLUM, MD
Deputy Editor and Book Review Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Production Manager	KATHLEEN WALSH TULLEY
Librarian and Administrative Assistant	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS
Editorial Assistant	MILDRED ARFMANN
Secretary	JOSEPHINE KREMPASKY
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1985

RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MARK C. HOMONOFF, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. Synergistic Media Sales, 50 East 42nd St., Suite 504, New York, N.Y. 10017. Telephone: (212) 682-2200.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

DANIEL F. O'KEEFFE, MD,	*President*
ALLISON B. LANDOLT, MD,	*Past-President*
JOHN A. FINKBEINER, MD,	*President-Elect*
SAMUEL M. GELFAND, MD,	*Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
CHARLES D. SHERMAN, JR, , MD,	*Treasurer*
MORTON KURTZ, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1986	ROBERT A. MAYERS, MD, Westchester THOMAS D. PEMRICK, MD, Rensselaer RALPH E. SCHLOSSMAN, MD, Queens STANLEY A. STECKLER, MD, Suffolk ROBERT J. NEWBORN, Albany *(Councilor representing the medical student membership)*
Term Expires 1987	DAVID M. BENFORD, MD, New York DUANE M. CADY, MD, Onondaga JAMES M. FLANAGAN, MD, Wayne M. THEODORE TANENHAUS, MD, Brooklyn
Term Expires 1988	JAMES H. COSGRIFF, JR, MD, Erie RICHARD A. HUGHES, MD, Warren ANTONIO F. LaSORTE, MD, Broome SIDNEY MISHKIN, MD, Nassau

Trustees

G. REHMI DENTON, MD, Albany, *Chairman*
RALPH M. SCHWARTZ, MD, New York
EDGAR P. BERRY, MD, New York
RICHARD D. EBERLE, MD, Onondaga
JOSEPH F. SHANAPHY, MD, Richmond
VICTOR J. TOFANY, MD, Monroe
BERNARD J. PISANI, MD, New York

Executive Vice-President	GEORGE J. LAWRENCE, JR, MD
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Associate	IRMA A. ERICKSON
Director, Division of Scientific Publications	ALAN BLUM, MD
Director, Division of Management Services	GEORGE FORREST, JR
Assistant Director, Division of Governmental Affairs	JERRY S. HOFFMAN, ESQ
Director, Division of Communications	EDWARD HYNES
Director, Division of Insurance and Membership Benefits	BERNARD M. JACKSON
Director, Division of Membership	EUNICE SKELLY
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Scientific Activities	PETER B. FARNSWORTH, MD
Director, Division of Advertising-Sales and Meeting Services	ROBERT MILLER
General Counsel	DONALD R. MOY, JD
Director, Division of Computer Services	IVAN H. NAJMAN
Acting Director, Division of Medical Services	MARIO MENGHINI
Director, Division of Long-Range Planning	DEBORAH R. SIEGEL
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. Editorial material published in the *Journal* is covered by copyright. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

THE PHYSICIAN'S ATVANTAGE

FUTURE transforms the IBM PC AT into a nine-user system to manage your practice.

Now FUTURE Information Systems offers the ATvantage that lets you practice medicine instead of accounting: Computone's powerful combination of Medical Office Management System software and AT expansion hardware. The total system that provides everything you need to automate your office from one of the country's fastest-growing computer dealers.

You can build a single IBM PC AT into a nine-user multi-tasking system with the AT ATvantage board, using any IBM PC, PCjr or inexpensive stand-alone terminals you may already own.

You can reduce paperwork with easy-to-use Medical Office Management System software, designed to let your staff enter or access information and simultaneously perform up to five related functions. Many accounting and management control operations are automatically updated with patient entry and posting.

And you can do it all with FUTURE beside you every step of the way, providing in-house or on-site training, complete hardware and software maintenance and continuous support.

Get the Physician's ATvantage working for you. Contact FUTURE Information Systems today.

Features of the Physician's ATvantage:

☐ Instant access to patient balance, insurance submission and insurance payment information
☐ Open-item patient accounting
☐ Patient statements and "Super-bill"
☐ Third-party insurance processing
☐ Patient/guarantor relationships
☐ Insurance rebilling or write-off
☐ Cash flow control
☐ Patient history
☐ Problem accounts
☐ Accounts receivable reductions of 20-50%
☐ Patient referrals
☐ Appointment scheduling
☐ Automatic audit trails
☐ Single doctor office, or several
☐ Multi-office practices

FUTURE℠
INFORMATION SYSTEMS
A Computone company

MOVING YOU UP IN THE WORLD℠

Business Market Sales Centers:
Downtown Manhattan: 95 Trinity Place • (212) 732-3905
Albany: 1492 Central Ave • (518) 456-8162

Midtown Manhattan: 52 East 53rd St. • (212) 751-7888
Schenectady: Woodlawn Plaza, 1594 State St. • (518) 382-1182
Glens Falls: 28 Ridge St • (518) 798-1446
Kingston: King's Mall, Rt. 9-W • (914) 336-4155

IBM PC, IBM PCjr and IBM PC AT are trademarks of International Business Machines Corp. ATvantage is a trademark of Computone Systems Inc

SURGEONS

Have you considered the U.S. Army Reserve?

1775

☐ SERVE IN THE ARMY RESERVE MEDICAL DEPARTMENT AS A COMMISSIONED OFFICER
☐ CME PROGRAMS FUNDED BY THE U.S. ARMY
☐ FLEXIBLE HOURS AND LOCATIONS AS NOT TO INTERFERE WITH YOUR PRACTICE
☐ NON-CONTRIBUTIVE RETIREMENT ANNUITY
☐ A CHANCE TO BROADEN YOUR MEDICAL EXPERTISE

CALL COLLECT
(718) 352-5850

USAR AMEDD Procurement
P.O. Box 4649
Bay Terrace, NY 11360-4649

NYMS-9

Name: ______________________________
Specialty: ______________________________
Address: ______________________________
Business Phone: ______________ Home Phone: ______________
Best Time to Call: ______________________________

Right from the start in hypertension...

Once-daily INDERAL LA (propranolol HCl) for smooth blood pressure control without the potassium problems of diuretics

Once-daily INDERAL LA (propranolol HCl) avoids the risk of diuretic-induced ECG abnormalities due to hypokalemia.[1,2] In addition, INDERAL LA preserves potassium balance without additive agents or supplements while providing simple, well-tolerated therapy with broad cardiovascular benefits.

Once-daily INDERAL LA for the cardiovascular benefits of the world's leading beta blocker

Simply start with 80 mg once daily. Dosage may be increased to 120 mg to 160 mg once daily as needed to achieve additional control.

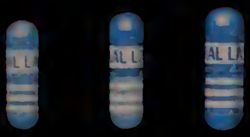
Like conventional INDERAL tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.

For beta-1/beta-2 blockade

Once-daily

INDERAL LA

(PROPRANOLOL HCl) [illegible]



80 mg 120 mg 160 mg

The appearance of these capsules is a registered trademark of Ayerst Laboratories.

Please see brief summary of prescribing information on the next page for further details

Once-daily
For beta-1/beta-2 blockade INDERAL® LA
(PROPRANOLOL HCl) LONG ACTING CAPSULES

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain. Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use Effects on plasma volume appear to be minor and somewhat variable INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction. Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential. The significance of the membrane action in the treatment of arrhythmias is uncertain

The mechanism of the antimigraine effect of propranolol has not been established Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital. For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm

Propranolol is not significantly dialyzable

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

The appearance of these capsules
is a registered trademark
of Ayerst Laboratories.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g , dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin.

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General.* Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure

Clinical Laboratory Tests Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS. Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers. INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman

Pediatric Use Safety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: bradycardia, congestive heart failure, intensification of AV block, hypotension; paresthesia of hands; thrombocytopenic purpura; arterial insufficiency, usually of the Raynaud type

Central Nervous System: lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances, hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics

Gastrointestinal. nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic: pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory. bronchospasm.

Hematologic: agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune. In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous. alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval.

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic The dosage may be increased to 120 mg once daily or higher until adequate blood pressure control is achieved. The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained. Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis. If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

REFERENCES

1. Holland OB, Nixon JV, Kuhnert L: Diuretic-induced ventricular ectopic activity. *Am J Med* 1981;70:762-768. **2.** Holme I, Helgeland A, Hjermann I, et al: Treatment of mild hypertension with diuretics. The importance of ECG abnormalities in the Oslo study and in MRFIT. *JAMA* 1984;251:1298-1299

Ayerst® AYERST LABORATORIES
New York, N Y. 10017

9411/1184

Copyright © 1984 AYERST LABORATORIES
Division of AMERICAN HOME PRODUCTS CORPORATION

Classified Advertising

PHYSICIANS WANTED

EXCELLENT OPPORTUNITY FOR READY MADE PRACTICE for General Practice Specialist or Internist willing to do a little general practice, available immediately, in an attractive Medical Building with in-house lab and x-ray facilities. Permanent relationship very desirable. Beautiful countryside-Cortland, NY. Phone collect (607) 756-9941 or submit c.v. with references to: Donald R. Gibbs, M.D., 110 North Main Street, Cortland, NY 13045.

NEW YORK, NEW PALTZ—Seeking full-time experience or residency trained physicians; ACLS certified; for state of the art FEC in beautifull Mid-Hudson Valley, 80 miles from NYC. Start date is July 15, 1985. Package includes Health, Life, Disability and Malpractice Insurance. Excellent salary, attractive scheduling and potential profit sharing. Send CV/Inquiries to Gary W. Greer, M.D., RD 3 Box 44A, Cogan Station, PA., 17728: phone 1-717-494-0420.

ARIZONA BASED PHYSICIAN RECRUITMENT firm has opportunites Coast to Coast. "Professionals working with Professionals". Over 13 years experience. Call (602) 795-7474; or send CV to: Mitchell & Associates, Inc., 2761 N. Country Club Rd., Suite 202, Tucson, AZ 85716.

PHYSICIANS NEEDED for private practice opportunities nationwide—all specialties. Hospital-based positions available in Ohio—Ob/Gyn, Internal Medicine, E.R.—must have Ohio license. Escape rising New York malpractice rates. Call (216) 292-7445 or send curriculum vitae to Physician Staffing, Inc., 29525 Chagrin Blvd. #313, Cleveland, Ohio 44122.

A FINE COMMUNITY OF 15,000 on Lake Erie is seeking two general or family practice physicians interested in private practice to replace two physicians who have retired. A progressive 86 bed JCAH accreditied acute care hospital serves the community. Assistance is available. Please contact George Whitney, Administrator; Brown Memorial Hospital, 158 West Main Road; Conneaut, Ohio 44030; (216) 593-1131.

ORTHOPEDIC SURGEON—Single specialty group desires third BE/BC ortho to join their team in upstate NY. Training and/or experience in arthroscopy desired. Guarantee plus productivity allowance. Partnership opportunity available. Serving area 60,000; office convenient to fully-equipped 158 bed hospital. Family-oriented lifestyle; outstanding schools and recreational area. Reply Dept. 395 c/o NYSJM.

PHYSICIANS WANTED—CONT'D

WANTED: SPINE SURGEON, experienced in all modern spinal surgical techniques and willing to keep up-to-date with new ones. No fulltime spinal surgeon within 200 mile radius. 650 bed hospital. Contact John W. McFadden, M.D., 320 S. Gloster Street, Tupelo, MS 38801.

DESPERATELY SEEKING ASSOCIATE to share some expenses and gradually (3–5 yrs.) take over active internal medicine practice in prime location, White Plains, NY. Prefer creative, industrious, entrepreneurial individual with some capital. Send cv to Dept. 393 c/o NYSJM.

INTERNAL MEDICINE, large practice outside Buffalo, New York for sale. For more information send C.V. to: Health Care Personnel Consulting, Inc., 400 GSB Building, One Belmont Avenue, Bala Cynwyd, PA 19004 (215) 667-8630.

SURGERY: GENERAL PRACTICE AVAILABLE—this GP/GS combination practice in upstate New York is a unique opportunity for the physician interested in a broad practice. Send C.V. to: Health Care Personnel Consulting, Inc., 400 GSB Building, One Belmont Avenue, Bala Cynwyd, PA 19004 (215) 667-8630.

PRACTICES AVAILABLE

SELLING PRACTICES IS OUR ONLY BUSINESS—Want to maximize the return on your investment? Before you buy or sell, call for a consultation to assure the best financial and transfer terms. We guide you through the entire sales process from initial meeting to closing. Serving 100 mile radius of New York City. Countrywide Business Brokerage, Inc., 319 East 24th Street, Suite 23-G, New York, NY 10010 (212) 686-7902.

MEDICAL PRACTICE SALES and Appraisals. We specialize in the valuation and selling of medical practices. If interested in buying or selling a medical practice, contact our Brokerage Division at: The Health Care Group, 400 GSB Building, Bala Cynwyd, PA 19004. (215) 667-8630.

FLORIDA PEDIATRICS SOLO PRACTICE: well established, fully equipped, good clientele, quality practice, charts, equipment, goodwill transferable. Office building for purchase or rent. Terms flexible. Reply Dept. 394 c/o NYSJM.

REAL ESTATE FOR SALE OR RENT

Manheim Realty, Inc.

Commercial, Industrial & Business Brokerage

MANHEIM. . .the professional name in private practice sales. Let the professional division of MANHEIM REALTY INC. help you in buying or locating professional space. We can insure that you will receive personal service and that your business will be held in the strictest confidence. Please contact:

Ruth Karp, R.N., EAB Plaza, Uniondale, NY 11555
(516) 794-6600

REAL ESTATE FOR SALE OR RENT—CONT'D

BAY SHORE, Leasing new luxurious medical building. Located near Southside Hospital. Minimum suite 740 sq. ft. Large parking area, handicapped designed. Individual gas heat, a/c-electric. (516) 665-4064, 10 A.M. to 5 P.M., Monday to Friday.

WANTED: MEDICAL OFFICE BUILDINGS. As principals we are seeking "existing buildings"—fully tennanted by physicians, dentists and health care services. Contact: Paul Gellert, Gelco Associates, 155 West 68th St., N.Y., N.Y. 10023; (212) 724-7900.

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

RHINEBECK, NY. Fine residential area. Professional apartment for rent, 1300 sq. ft. 1¾ hours from Grand Central Amtrak, Two minutes to Northern Dutchess Hospital. Near hotels, restaurants, shopping. Large waiting room, office, three examination rooms, lab, ample storage, closet space. Air conditioned. Early occupancy. (914) 876-6303.

LIDO BEACH, L.I.—Doctor's house for sale. Corner, brick/shingle, custom 10 room high ranch, 4 bedrooms, 3½ baths, maids' quarters, 2 room, ½ bath professional office with separate entrance. Central a/c, underground sprinklers, fire/burglar alarms, fireplace, intercom, beautifully landscaped, 1 block from ocean/beach and bay. Near golf course and tennis. $400,000.00. Principals only, (516) 432-8036.

BEAUTIFUL EXTENDED 9 ROOM HIGH RANCH. 1/3rd acre, private park like grounds, huge sun-deck plus patio, 2 car garage, custom mirrors, imported tiles, 2 new bathrooms, tiled and mirrored new kitchen, air conditioned, huge den with fireplace, new above ground pool with deck, wall to wall carpeting. Owner relocating, sacrificing, many extras, all appliances. $145.000. Owner (516) 231-1216.

THE INTELLIGENT CHOICE. Diagnosis: Spiraling Rent Syndrome, Inflamed Hospital Costs. Prognosis: Continually Worsening Condition. Prescription: Enclave Commercial Condominium, 224 East 52nd Street, 7,000 square feet, new construction, divisible, 3 floors, 3 units. Build equity, stabilize long-term costs, increase profit potential, reap tax advantages of depreciation, interest and expense deductions. Offices customized for purchasers: partitions, HVAC, carpeting, hung ceilings, kitchenette, private baths, amenities. Suitable: Imaging Center, Fertility Clinic, Sports Medicine, Cosmetic Surgery, Allergy Center, Stress Management, Preventive Medicine, Eye Microsurgery, Cosmetic Dermatology, Diagnostic Center. Central to Manhattan's major medical centers, convenient to transportation, in finest commercial and residential neighborhood. JOSEPH P. DAY REALTY CORP., Douglas L. Zinn, Ruth A. Zales, Executive Agents. (212) 889-7460. This is not an offering which can be made only by prospectus.

Added Control for America's Coughs

Robitussin A-C®

Robitussin and codeine

Each 5 ml (1 teaspoonful) contains:

Guaifenesin, USP	100 mg
Codeine Phosphate, USP	10 mg

(Warning May be habit forming)

Alcohol 3.5 percent

In a palatable, aromatic syrup

The expectorant action of guaifenesin plus the **A**dvantage of **C**odeine!

A·H·ROBINS
Pharmaceutical Division, Richmond, Virginia 23261-6609

© A.H. Robins Company 1985

BALANCED CALCIUM CHANNEL BLOCKADE!

CARDIZEM
(diltiazem HCl)
balances potent coronary vasodilation with a low incidence of side effects

Low incidence of side effects
CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

Reduces angina attack frequency*
42% to 46% decrease reported in multicenter study.[1]

Increases exercise tolerance*
In Bruce exercise test,[2] control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes (*P*<.005).

CARDIZEM®
(diltiazem HCl)

THE BALANCED CALCIUM CHANNEL BLOCKER

*Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

References:
1. Strauss WE, McIntyre KM, Parisi AF, et al: Safety and efficacy of diltiazem hydrochloride for the treatment of stable angina pectoris: Report of a cooperative clinical trial. Am J Cardiol 49:560-566, 1982.
2. Pool PE, Seagren SC, Bonanno JA, et al: The treatment of exercise-inducible chronic stable angina with diltiazem: Effect on treadmill exercise. Chest 78 (July suppl):234-238, 1980.

Please see full prescribing information on following page.

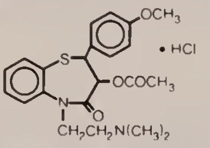
2/84

PROFESSIONAL USE INFORMATION

cardizem®
(diltiazem HCl)
30 mg and 60 mg tablets

DESCRIPTION

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one,3-(acetyloxy)-5-[2-(dimethylamino)ethyl]-2,3-dihydro-2-(4-methoxyphenyl)-, monohydrochloride,(+)-cis-. The chemical structure is.



Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform. It has a molecular weight of 450.98. Each tablet of CARDIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration.

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth muscle.

Mechanisms of Action. Although precise mechanisms of its antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways

1. Angina Due to Coronary Artery Spasm: CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial and subendocardial. Spontaneous and ergonovine-induced coronary artery spasm are inhibited by CARDIZEM.
2. Exertional Angina: CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal and maximal exercise work loads.

In animal models, diltiazem interferes with the slow inward (depolarizing) current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Diltiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance.

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricular conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load. Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect; cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of diltiazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by diltiazem.

Intravenous diltiazem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Diltiazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, diltiazem significantly prolongs sinus cycle length (up to 50% in some cases).

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Diltiazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40%. CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 60 mg are given; a 120-mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem.

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm.** CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina).** CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS).

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia.
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing.
Cardiac Failure	Administer inotropic agents (isoproterenol, dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

The oral/LD_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort acute anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy**—CARDIZEM may be safely coadministered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

Cardizem 30-mg tablets are supplied in bottles of 100 (NDC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1771-49). Each green tablet is engraved with MARION on one side and 1771 engraved on the other. CARDIZEM 60-mg scored tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1772-49). Each yellow tablet is engraved with MARION on one side and 1772 on the other.

Issued 4/1/84

Another patient benefit product from
PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MISSOURI 64137

We've been studying medicine almost as long as you have.

Over 17 years and untold manhours. That's what CyCare has invested in the study of health care. We've long since earned our diploma. While many aspiring competitors failed to make the grade.

We chose one specialty. The delivery of health care is a specialized business. Your data processing company should understand it thoroughly. That's tough to do if they're also marketing to banks, factories and the like.

From the beginning, CyCare decided to commit only to the medical industry. Our staff of over 600 has been living and breathing it ever since.

Experience only CyCare can claim. CyCare has studied with thousands of physicians, administrators, and nurses. We've worked with nearly 850 clients of all specialties and size. In 17 years, we've treated data processing challenges of every kind.

You know experience is the best teacher. So choose a company that's been around long enough to learn.

Our knowledge benefits you. Our experience taught us that each client is different. We designed systems that easily accomodate any type of case. We learned you didn't want useless "bells and whistles", so we developed practical software that enhances the delivery of patient care. We discovered the fear of system obsolescence. So we created modular systems that can be expanded at any time.

Learning never stops. Like you, we never stop learning. We invest more in research each year than most of our competitors gross in sales. We listen to your ideas, look for new ways to improve your practice, and stay abreast of industry needs. It's the only way to take the lead.

Compare our credentials. Examine CyCare thoroughly. Demand as much from us as you demand from yourself. Look at our experience, our financial stability. Examine our products and talk to our clients. Find out why CyCare is the leading supplier to medical group practices, HMOs and ambulatory care facilities nationwide.

Put us to the test. We're prepared.

Ask about CyCare's C100 APPOINTMENT SCHEDULING. The physician time management system for small and medium size practices.

☐ Rush free details to me about CyCare.

☐ Have a representative contact me.

My business card/letterhead is attached.

No. of Phys. ____

Mail to: CyCare, 520 Dubuque Building Dubuque, Iowa 52001
319/556-3131
NYSJM 9/85

Sales and Service Offices:
Atlanta, GA; Cherry Hill, NJ; Chicago, IL; Dallas, TX; Denver, CO; Miami, FL. Minneapolis, MN; New York, NY; Portland, OR; San Diego, CA; Spokane, WA; Canada: Toronto, Ont.

Authorized WANG National ISO

cycare
a step ahead...

Put the world of medicine in your pocket every two weeks.

Right now, thousands of physicians like you are reading journals for in-depth information. But how can *anyone* keep up with the complex, rapidly changing world of medicine as a whole? By reading *Medical World News.*

For more than 115,000 busy physicians, *Medical World News* is the secret to staying in touch with the latest developments in therapy, research, practice, pharmacology, economics, legislative action, highlights of other periodicals and new medical trends.

We're doing it for them, and we can do it for you.

Every two weeks, we can bring you up-to-the-minute news from the nation's medical pressure points: Atlanta, Chicago, Dallas, Houston, Los Angeles, New York, San Francisco and Washington D.C. A network of contributing editors in major U.S., Canadian and European cities adds an international perspective to the mix. **Our policy of unbiased reporting gives you information you can trust.** And our easy-to-read style makes it enjoyable to stay informed.

medical
world
news The credible source.

© 1985 HEI Publishing

EXCLUSIVE

NEWS FOR NEW YORK

TALWIN® Nx

Ⓒ IV Each tablet contains pentazocine HCl, USP, equivalent to 50 mg base and naloxone HCl, USP, equivalent to 0.5 mg base.

NOW APPROVED FOR MEDICAID

T-540 NDC 0024-1951-04
100 tablets
Ⓒ IV
Talwin® Nx
Each tablet contains pentazocine hydrochloride, USP, equivalent to 50 mg base and naloxone hydrochloride, USP, 0.5 mg.
Caution: Federal law prohibits dispensing without prescription.
Winthrop

Talwin® Nx

Ⓒ IV Each tablet contains pentazocine HCl, USP, equivalent to 50 mg base and naloxone HCl, USP, equivalent to 0.5 mg base.

Major Analgesic Gains Reimbursement Status

Albany—TALWIN Nx has recently been included on the New York State Medicaid list of reimbursable drugs.

Physicians can now make available to their Medicaid patients the many advantages of TALWIN Nx for moderate to severe pain conditions such as fractures, acute lumbosacral pain, severe sprains, joint pain, and acute trauma.

The newly listed TALWIN Nx is characterized by dependable efficacy and a proven record of safety.[1] One tablet provides pain relief equivalent to one grain of codeine, yet allows patients to function.* TALWIN Nx offers a 15 to 30 minute onset of action, and is logical therapy for persistent pain because tolerance buildup is rare. The product is especially appropriate for many elderly patients since it has a low risk of constipation.†

---

*Since sedation, dizziness, and occasional euphoria have been noted, ambulatory patients should be warned not to operate machinery, drive cars, or unnecessarily expose themselves to hazards.

†**See important product information concerning contraindications, warnings, adverse reactions, patient selection, and prescribing and precautionary recommendations on following page.**

[1] Data from the Boston Collaborative Drug Surveillance Program, July 1984.

TALWIN Nx now approved for the Medicaid Drug List by New York State.

Winthrop-Breon

© 1985 Winthrop-Breon Laboratories

Talwin® Nx

Each tablet contains pentazocine HCl, USP, equivalent to 50 mg base and naloxone HCl, USP, equivalent to 0.5 mg base.

Analgesic for Oral Use Only

Contraindications: Hypersensitivity to either pentazocine or naloxone.

> TALWIN® Nx is intended for oral use only. Severe, potentially lethal, reactions may result from misuse of TALWIN® Nx by injection either alone or in combination with other substances. (See **Drug Abuse and Dependence** section.)

Warnings: *Drug Dependence.* Can cause physical and psychological dependence. (See **Drug Abuse and Dependence.**) *Head Injury and Increased Intracranial Pressure.* As with other potent analgesics, respiratory depressant effects of the drug may elevate cerebrospinal fluid pressure due to CO_2 retention; these effects may be markedly exaggerated in the presence of head injury, other intracranial lesions, or a preexisting increase in intracranial pressure. Can obscure the clinical course of patients with head injuries; in such patients, use with extreme caution and only if deemed essential. *Usage with Alcohol.* Due to potential for increased CNS depressant effects, alcohol should be used with caution. *Patients Receiving Narcotics.* Pentazocine is a mild narcotic antagonist. Withdrawal symptoms have occurred in patients previously given narcotics, including methadone. *Certain Respiratory Conditions.* Should be administered with caution in respiratory depression from any cause, severely limited respiratory reserve, severe bronchial asthma and other obstructive respiratory conditions, or cyanosis. *Acute CNS Manifestations.* Therapeutic doses have resulted in hallucinations (usually visual), disorientation, and confusion which have cleared spontaneously within hours. Such patients should be closely observed and vital signs checked. Reinstitution of the drug should be done cautiously since acute CNS manifestations may recur.

Precautions: *CNS Effect.* Use cautiously in patients prone to seizures; seizures have occurred though no cause and effect relationship has been established. *Impaired Renal or Hepatic Function.* Decreased metabolism of pentazocine in extensive liver disease may predispose to accentuation of side effects; it should be administered with caution in renal or hepatic impairment. In long-term use, precautions should be taken to avoid increases in dose by the patient. *Biliary Surgery.* Some evidence suggests that unlike other narcotics pentazocine causes little or no elevation in biliary tract pressures; the clinical significance of these findings is not yet known. *Information for Patients.* Since sedation, dizziness, and occasional euphoria have been noted, ambulatory patients should be warned not to operate machinery, drive cars, or unnecessarily expose themselves to hazards. May cause physical and psychological dependence taken alone and may have additive CNS depressant properties in combination with alcohol or other CNS depressants. *Myocardial Infarction.* Use with caution in patients with myocardial infarction who have nausea or vomiting. *Drug Interactions. Usage with Alcohol:* See **Warnings.** *Carcinogenesis, Mutagenesis, Impairment of Fertility.* No long-term studies in animals to test for carcinogenesis have been performed. *Pregnancy Category C.* Should be given to pregnant women only if clearly needed. *Labor and Delivery.* Use with caution in women delivering premature infants. Effect on mother and fetus, duration of labor or delivery, need for forceps delivery or other intervention or resuscitation of newborn, or later growth, development, and functional maturation of the child is unknown. *Nursing Mothers.* Caution should be exercised when administered to a nursing woman. *Pediatric Use.* Safety and effectiveness in children below the age of 12 years have not been established.

Adverse Reactions: *Cardiovascular:* Hypotension, tachycardia, syncope. *Respiratory:* Rarely, respiratory depression. *Acute CNS Manifestations:* In rare instances, hallucinations (usually visual), disorientation, and confusion which have cleared spontaneously within a period of hours. Such patients should be closely observed and vital signs checked. Acute CNS manifestations may recur if the drug is reinstituted. *Other CNS Effects:* Dizziness, lightheadedness, hallucinations, sedation, euphoria, headache, confusion, disorientation; infrequently weakness, disturbed dreams, insomnia, syncope, visual blurring and focusing difficulty, depression; rarely tremor, irritability, excitement, tinnitus. *Autonomic:* Sweating; infrequently flushing; and rarely chills. *Gastrointestinal:* Nausea, vomiting, constipation, diarrhea, anorexia, rarely abdominal distress. *Allergic:* Edema of the face; dermatitis, including pruritus; flushed skin, including plethora; infrequently rash, rarely urticaria. *Ophthalmic:* Visual blurring and focusing difficulty. *Hematologic:* Depression of white blood cells (especially granulocytes), which is usually reversible, moderate transient eosinophilia. *Other:* Headache, chills, insomnia, weakness, urinary retention, paresthesia.

Drug Abuse and Dependence: *Controlled Substance.* TALWIN Nx is a Schedule IV controlled substance.

Dependence and withdrawal symptoms have been reported with orally administered pentazocine. Patients with a history of drug dependence should be under close supervision. Possible abstinence syndromes in newborns after prolonged use of pentazocine during pregnancy have been reported. In prescribing for chronic use, the physician should take precautions to avoid increases in dose by the patient. Tolerance to the analgesic effect is rarely reported; there is no long-term experience with oral use of TALWIN Nx.

The amount of naloxone present (0.5 mg per tablet) has no action when taken orally and will not interfere with the pharmacologic action of pentazocine; however, this amount of naloxone given by injection has profound antagonistic action to narcotic analgesics. TALWIN Nx has a lower potential for parenteral misuse than the previous oral pentazocine formulation, but is still subject to patient misuse and abuse by the oral route.

Severe, even lethal, consequences may result from misuse of tablets by injection either alone or in combination with other substances, such as pulmonary emboli, vascular occlusion, ulceration and abscesses, and withdrawal symptoms in narcotic dependent individuals.

Overdosage: *Treatment:* Oxygen, intravenous fluids, vasopressors, and other supportive measures should be employed as indicated. Assisted or controlled ventilation should also be considered. For respiratory depression, parenteral naloxone is a specific and effective antagonist.

Please consult full product information before prescribing.

WIN 5-51296

Winthrop-Breon

Winthrop-Breon Laboratories
Division of Sterling Drug Inc.
New York, NY 10016

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meetings and lectures unless the information is submitted at least two months prior to the event.*

MANHATTAN

Oct 2. **Advances in Pediatric Hematology–Oncology.** 3½ Cat 1 credits. *Oct 21–25.* **Current Concepts in Medical Oncology '85.** 27 Cat 1 credits. Info: CME Planning Office, Memorial Sloan-Kettering Cancer Center, 1275 York Ave, New York, NY 10021. Tel: (212) 794-6754.

Oct 3–4. **8th Annual Current Concerns in Adolescent Medicine—A Practitioner's Guide to Teenage Health Care.** 13 Cat 1 credits. The Warwick Hotel. *Oct 11.* **Early Breast Cancer: The Psychological Perspective.** 7 Cat 1 credits. The Grand Hyatt. Info: Ann J. Boehme, CME, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

Oct 5–6. **Fine Needle Aspiration of the Breast and Thyroid Gland.** 12½ Cat 1 credits. *Oct 18–19.* **Endometriosis Symposium.** 10½ Cat 1 credits. *Oct 21–23.* **Sonography with CT, MR Correlations.** 16 Cat 1 credits. *Oct 24–25.* **The Fourth Annual Symposium on Interventional Radiology.** 12½ Cat 1 credits. *Oct 25–26.* **The First New York City Marathon Sports Emergency Symposium.** 11 Cat 1 credits. Info: NYU Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Oct 11. **Cosmetology Symposium.** *Oct 23.* **Therapy, 1985.** Info: Beverley R. Baptiste, Medical Education, Beth Israel Medical Center, Nathan D. Perlman Pl, New York, NY 10003. Tel: (212) 420-2849.

Oct 19. **The Cataract: Visual Pitfalls.** Info: Martha Klapp, Manhattan Eye, Ear and Throat Hospital, 210 East 64th St, New York, NY 10021. Tel: (212) 605-3762.

Oct 25–26. **Third Annual Postgraduate Symposium on Gynecological Endocrinology and Infertility.** 16 Cat 1 credits. Info: Department of Obstetrics and Gynecology, Lenox Hill Hospital, 100 E 77th St, New York, NY 10021. Tel: (212) 794-4096.

(*Continued on p 20A*)

NEW YORK STATE JOURNAL OF MEDICINE

September 1985 • Volume 85, Number 9

COMMENTARIES

Immunosuppression for renal allograft recipients: The role of cyclosporine

Cyclosporine is a cyclic unadecapeptide product of fungal fermentation that was found by Borel and Lafferty[1] in 1976 to have immunosuppressive properties. Its effects on the immune system are selective, with T helper cell and lymphokine production being inhibited to a much greater degree than other functions.[2]

Calne et al[3] introduced cyclosporine into clinical practice in 1978 at the University of Cambridge in England. This group experimented with cyclosporine under a variety of protocols, including using it in combination with relatively high doses of other immunosuppressive drugs. Their initial findings included a high incidence of infections and other complications when cyclosporine was used with other agents, so they settled on a protocol in which cyclosporine alone, given at high doses, was used to treat recipients of renal allografts. Prednisone was added only if there were rejection episodes. They thought that cyclosporine by itself could prevent rejection in a large proportion of the recipients. However, the incidence of nephrotoxicity was high, and more than half of the patients eventually required corticosteroid therapy.

Starzl et al,[4] then at the University of Colorado, were the first to report on a clinical trial of cyclosporine in the United States. This group used cyclosporine in lower doses than at the University of Cambridge and also used a lower dose of prednisone for prophylactic purposes. This basic protocol was then adopted by other American centers involved in the initial evaluation of this drug. Cyclosporine was looked upon as a substitute for azathioprine and as a drug that could reduce the need for steroid therapy.

The initial results reported with the use of cyclosporine showed relatively high graft survival rates compared to historical control patients. However, as Tellis et al[8] point out in an article in this issue of the *Journal*, the results in kidney transplantation with conventional immunosuppression using azathioprine, prednisone, and antilymphocyte globulin (ALG) were already improving, particularly in recipients given blood transfusions.

Only three randomized prospective studies of cyclosporine versus conventional immunosuppression in a large number of patients have been conducted. Of these, two were multi-institutional trials, in Europe[9] and Canada,[10] and one was at a single institution, the University of Minnesota.[11] The Canadian and European trials were performed in recipients of cadaveric grafts alone, and in both trials graft survival rates at one year were significantly higher among patients given cyclosporine versus those treated with azathioprine: 72% versus 52% ($P = .001$) in the European study and 84% versus 67% ($P = .003$) in the Canadian trial. In the Minnesota study, however, a significant difference between the two regimens could not be shown, primarily because the graft survival rates in the group treated with conventional immunosuppression were much higher than in the other two trials (Table).

The conventional immunosuppressive regimen at the University of Minnesota was much like that applied at Montefiore Hospital in New York as described by Tellis et al.[8] The results at the two institutions with this regimen are similar. In the Minnesota randomized trial, 75% of cadaveric grafts in recipients treated with azathioprine, prednisone, and ALG were functioning at one year,[12] as were 69% of those transplanted at Montefiore.[8] At both institutions the cadaveric graft survival rates with conventional immunosuppression were higher than in the randomized trials conducted in Europe and Canada. ALG was not used in the European trial,[9] and it was used in only a few centers in the Canadian trial.[10] However, at the institutions that used ALG in the Canadian trial, the graft survival rates were equivalent between recipients treated with cyclosporine or azathioprine.[13]

In the Minnesota trial, certain advantages and disadvantages were associated with the use of cyclosporine.[11] Cyclosporine is nephrotoxic, and recipients treated with cyclosporine had significantly higher serum creatinine levels than those treated with azathioprine. Patients in whom the grafts did not function immediately were more difficult to manage. Hypertension and hyperkalemia were also more common in the group given cyclosporine, as were neurologic symptoms. The major advantages of cyclosporine therapy in the Minnesota trial related to a lower incidence of rejection episodes, a lower infection rate, and decreased hospital stays, primarily because the need to give ALG was obviated. Lymphoma occurred in only one patient treated with cyclosporine. Indeed, it now appears that lymphoma does not occur more frequently in association with cyclosporine therapy than with conventional immunosuppression treatment.[14]

In the Minnesota trial, a detrimental effect of acute tubular necrosis (ATN) was seen in both recipients given cyclosporine and control patients. At one year, graft survival rates were significantly lower in both groups of patients whose grafts underwent ATN, as opposed to those whose grafts did not undergo ATN (Table).

Patient and Renal Allograft Functional Survival Rates at One Year in Minnesota Randomized Trial of Cyclosporine (CSA), Prednisone Versus Azathioprine (AZA) Prednisone-Antilymphocyte Globulin for Immunosuppression*

	CSA	AZA
Patient Survival (131 versus 115)	92%	92%
Graft survival**	86%	79%
LRD (48 versus 40)	94%	87%
CAD (83 versus 75)	82%	75%
ATN† (26 versus 19)	73%	74%
No ATN (53 versus 51)	92%	82%

* From Sutherland et al.[12] Reprinted courtesy of *Am J Kidney Dis*.

** LRD = HLA mismatched living related donors; CAD = cadaver donor transplants.

† ATN = Delayed or lack of immediate graft function.

© 1985, National Kidney Foundation, Inc.

During the randomized trial at the University of Minnesota only 60 of 131 patients selected at random to receive cyclosporine treatment (46%) were maintained on this protocol for an indefinite period. Early in the trial, cyclosporine toxicity was managed by switching 25 patients to azathioprine. In 46 patients, the cyclosporine dose was simply lowered to a level at which toxicity disappeared, but that by itself would be inadequate to achieve immunosuppression, and azathioprine was added to the regimen. Thus, more than half of the patients treated with cyclosporine-prednisone eventually received azathioprine as well. Azathioprine was either added to the regimen or substituted for cyclosporine for the following reasons: cyclosporine nephrotoxicity in approximately 50% of patients; delayed graft function in 25%; and ongoing rejection in 25%. The modified regimens were effective, and graft survival rates at one year were equivalent to those among recipients who remained on the original cyclosporine protocol.[15] The serum creatinine level in patients who were switched to azathioprine declined from 2.8 mg/dL to 2.1 mg/dL; in those patients who received azathioprine in addition to the initial regimen, the level also decreased from 2.8 mg/dL to 2.1 mg/dL.

The observations at the University of Minnesota challenge the original recommendation of Calne et al[3] that cyclosporine should not be used in conjunction with other immunosuppressive agents. Cyclosporine in low doses can be used safely with azathioprine. Animal experiments had also shown a greater synergism for the therapeutic effects as opposed to the toxic effects of each drug. Cyclosporine may be more useful as a substitute for prednisone than for azathioprine. Azathioprine is a relatively nontoxic drug; the main side effect is leukopenia, and this can be easily managed by serial monitoring of the white blood count and adjustment of azathioprine dose.

For these reasons, a new immunosuppressive protocol was initiated at the University of Minnesota after conclusion of the randomized trial. The new protocol consists of azathioprine, low dose cyclosporine, and low dose prednisone (triple therapy). This protocol has been in effect for one year, and in a recent analysis of 75 recipients of cadaveric grafts and of 38 recipients of related grafts, six-month graft survival rates were 88% and 95%, respectively. Among the recipients of cadaveric grafts treated with triple therapy, the mean serum creatinine level at six months was 1.8 mg/dL versus 2.3 mg/dL in the patients treated with cyclosporine in the randomized trial. In living related recipients the mean serum creatinine level was 1.4 mg/dL versus 2.0 mg/dL in the patients given cyclosporine in the randomized trial.

This protocol was initiated in an attempt to use the profound immunosuppressive yet infectious-sparing properties of cyclosporine while avoiding nephrotoxicity. Preliminary observations suggest that this objective is being achieved.[16] Because of the lowered graft survival rates associated with delayed graft function, for recipients of cadaveric grafts, cyclosporine is not given until graft function is established; during the interval, ALG can be administered. Further fine tuning of these protocols is possible. The current protocol used in Minnesota for recipients of cadaveric grafts is similar to that now employed by Tellis et al,[8] in which cyclosporine is not used until serum creatinine levels reach normal limits. The major difference between the protocols at the two institutions is the continued use of azathioprine in combination with low dose cyclosporine in the Minnesota trial. However, I am in agreement with the statement by Tellis et al[8] that further studies are necessary and greater experience is required to better define the role of cyclosporine in renal transplantation. Nevertheless, it is clear that results of kidney transplantation for the treatment of end-stage renal failure are now excellent, and that most patients with kidney failure should be offered transplants rather than being relegated to chronic hemodialysis.

DAVID E. R. SUTHERLAND, MD
Professor
Department of Surgery
University of Minnesota
P.O. Box 280
420 Delaware St, SE
Minneapolis, MN 55455

1. Borel JF, Lafferty KJ: Cyclosporine: speculation about its mechanism of action. *Transplant Proc* 1983; 15:1881–1885.
2. Wiesinger D, Borel JF: Studies on the mechanism of action of cyclosporin A. *Immunobiology* 1980; 156:454–463.
3. Calne RY, White DJG, Thiru S, et al: Cyclosporin A in patients receiving renal allografts from cadaver donors. *Lancet* 1978; 2:1323–1327.
4. Starzl TE, Weil R 3rd, Iwatsuki S, et al: The use of cyclosporin A and prednisone in cadaver kidney transplantation. *Surg Gynecol Obstet* 1980; 151:17–26.
5. Kahan BD, Van Buren CT, Flechner SM, et al: Cyclosporine immunosuppression migrates immunological risk factors in renal allotransplantation. *Transplant Proc* 1983; 15:2469–2478.
6. Ferguson RM, Rynasiewicz JJ, Sutherland DE, Simmons RL, Najarian JS: Cyclosporin A in renal transplantation: a prospective randomized trial. *Surgery* 1982; 92:175–182.
7. Tilney NL, Milford EL, Araujo JL, Strom TB, Carpenter CB, Kirkman RL: Experience with cyclosporine and steroids in clinical renal transplantation. *Ann Surg* 1984; 200:605–613.
8. Tellis VA, Matas AJ, Glichlick D, et al: Cyclosporine—Preliminary experience in seventy-nine renal transplants. *NY State J Med* 1985; 85:531–533.
9. European Multicenter Trial Group: Cyclosporine and cadaver renal transplantation: one year followup of a multicenter trial. *Lancet* 1983; 2: 986–989.
10. The Canadian Multicenter Transplant Study Group: A randomized clinical trial of cyclosporine and cadaver donor renal transplantation. *N Engl J Med* 1983; 309:809–815.
11. Najarian JS, Fryd DS, Strand MH, et al: A single institution, randomized, prospective trial of cyclosporine versus azathioprine-antilymphocyte globulin for immunosuppression in renal allograft recipients. *Ann Surg* 1985; 201:142–157.
12. Sutherland DE, Fryd DS, Strand MH, et al: Results of the Minnesota Randomized Prospective Trial of cyclosporine versus azathioprine-antilymphocyte globulin for immunosuppression in renal allograft recipients. *Am J Kidney Dis* 1985; 5:318–327.
13. Halloran P, Ludwin D, Apile M, et al: Randomized comparison between CsA and conventional therapy plus Minnesota antilymphocyte globulin in cadaver renal transplantation. *Transplant Proc* 1983; 15:2513–2516.
14. Starzl TE, Nalesnik MA, Porter KA, et al: Reversibility of lymphomas and lymphoproliferative lesions developing under cyclosporine-steroid therapy. *Lancet* 1984; 1:583–587.
15. Simmons RL, Canafax DM, Strand MH, et al: Management and prevention of cyclosporine nephrotoxicity after renal transplantation: use of low doses of cyclosporine, azathioprine, and prednisone. *Transplant Proc* (in press).
16. Canafax DM, Sutherland DE, Simmons RL, et al: Combination immunosuppression: three drugs (azathioprine, cyclosporine, and prednisone) for mismatched related and four drugs (antilymphocyte globulin, azathioprine, cyclosporine, and prednisone) for cadaver renal allograft recipients. *Transplant Proc* (in press).

Multiple pregnancies

Spontaneous and induced

The early detection of twins by sonography at six to eight weeks of gestation occurs in one out of 50 pregnancies. Usually at the time of delivery half of this group delivers only a single fetus.[1] Without ovarian stimulation, spontaneous multiple births are a rarity. The incidence of twins is 1 in 90 births; of triplets, 1 in 8,000; of quadruplets, 1 in 700,000; and of quintuplets, 1 in 6.4 million. In many medical centers the frequency of multifetal pregnancies are higher due to the use of superovulatory agents. The physical properties of the human uterus provide ideal security only for a single term pregnancy. The twin frequently dies through loss of blood supply and may sometimes be found at term by diligent search, as a fetus papyraceus along the membranes of the live born infant.

In this issue of the *Journal*, Vafai and Shapiro[2] describe the successful delivery of quintuplets ranging in size from 1,380 to 1,540 grams following ovarian stimulation with HMG-hCG (human menopausal gonadotropin-human chorionic gonadotropin). The quintuplets were first detected at 10 weeks. Labor was recognized at 20 weeks by periodic uterine contraction and minimal cervical dilation. Through expert obstetric care using complete bed rest and intravenous and oral ritodrine, the staff of the University of Rochester delayed labor until 32 weeks. Betamethasone was administered to speed up lung maturation. At this time a cesarean section was performed as the safest and least traumatic method of delivery. The average hospital stay for the five infants was 50 days. The cost of special duty nurses around the clock in the high risk perinatal units becomes prohibitive for the average family and requires city and state funding or private philanthropy. In special perinatal units, infants who weigh from 1,200 to 1,500 grams will require four weeks of intensive perinatal care costing $20,000 and three weeks of special care costing $10,000. This expense of $30,000 does not include medical consultation fees which may total an additional $5,000. If complications occur during the clinical course, including severe respiratory distress syndrome, patent ductus arteriosus, bilateral pneumothorax, intraventricular hemorrhage, and severe visual disturbances, the increased hospital stay and the special care, including surgery, may double the cost to $100,000 per infant.

At the present time there are two approaches to treating anovulation with or without amenorrhea: therapy with clomiphene or human menopausal gonadotropin (HMG) with or without human chorionic gonadotropin (hCG). Clomiphene is the first choice for initiating normal ovulation. Clomiphene is administered in doses of 50 mg, 100 mg, or 150 mg for five days beginning at the third to fifth day of the menstrual cycle for anovulation produced by the contraceptive pill, by polycystic ovaries, or by unknown causes. Multifetal pregnancies are more frequent (10% to 15%) with clomiphene, and this drug may double the usual frequency of twins and triplets.

Human menopausal gonadotropin (HMG) is the principal medication for inducing superovulation. This hormone mixture may be administered when clomiphene therapy is unsuccessful, as with disorders of the hypothalamic pituitary ovarian axis. Formerly, HMG consisted of equal amounts of follicle stimulating hormone, 75 IU, and luteinizing stimulating hormone (LH), 75 IU. Recently, chemical purification has improved the product by virtually eliminating the LH. This has provided for better simulation of the normal ovarian follicle when little LH is secreted before the LH surge, 34 hours before ovulation.

The use of HMG in patients has the disadvantage of multiple pregnancies. Ovulation induction with pulsatile gonadotropic releasing hormone (GnRH) has the advantage of simulating the normal cycle and resulting in a single pregnancy.[3] Subcutaneous pulsatile GnRH is a safer and more effective method of producing single follicle ovulation.

Endocrinologists use various methods for producing superovulation. Ultrasonography of the ovary is the ideal way to follow the development and maturation of the oocytes.[4] Observations are started as early as day eight of the menstrual cycle when the stimulated follicles are 8 mm to 10 mm in size. The growth rate is usually 1 mm to 2 mm each day. When multiple follicles develop in close proximity following HMG therapy, compression can occur and measurement of size becomes increasingly difficult due to distortion. When multiple follicles are maturing, the LH surge usually does not occur; hCG is administered to simulate a normal LH surge. If hCG is given too early, before maturation in the cycle, atresia of the developing follicles may occur. Poor follicular development sometimes results in spite of routine stimulation with HMG. Unruptured luteinized follicles are rarely recognized and are seen only when a follicle persists many days after other follicles rupture. Hyperstimulation is a serious complication, and if the estradiol level is greater than 4,000 pg/mm, hCG should not be given. This may occur if eight to 10 follicles are maturing which can be visualized by ultrasonography. Admission to the hospital is mandatory to determine the cause of hyperstimulation: ascites, pleural effusions, hemoconcentration, oliguria, and hypotension make up the syndrome of overstimulation. Any electrolyte imbalance must be treated. Usually, as the patient's condition stabilizes, the entire clinical picture will reverse over several weeks.

Some medical units prescribe clomiphene, 100 mg on day 3 of the cycle along with two ampules of HMG on the fourth and fifth days of the cycle. The continuation of HMG therapy requires follow-up at least every 48 hours by serum estradiol levels and monitoring the growth of the graafian follicles through direct sonography. Each follicle of 17 mm to 20 mm in size at maturity will provide 400 pg/mL of estradiol; at that time 5,000 IU of hCG is administered. Ovulation usually occurs 34 hours later. If more than three follicles are maturing, the cycle can be aborted if the couple uses barrier contraception. If there are several mature follicles—ranging from three to eight—some may not result in pregnancy. If a pelvic sonogram is performed at seven weeks the pregnancy sacs should be easily visualized. If there are more than three pregnancies visualized in the uterine fundus, prematurity may complicate the delivery. Some endocrinologists believe that with more than three pregnancies the complication rates and costs are so high due to premature delivery, and the final results are likely to be disastrous for the infants, mother, and physician. Recently, in California, a cesarean section was performed for septuplets resulting from treatment with HMG. All of the infants weighed less than 1,000 grams. Four of the seven have already died, and it is hoped that one of the group will reach a normal mental and physical status.

The great advance in the use of clomiphene and HMG during the past five years and the development of early and reliable ovulation detection and pregnancy tests[5] have made possible the development of in vitro fertilization and the management of early pregnancy. Some groups use clomiphene and HMG together, and others use HMG alone. Superovulation is maximized to obtain as many mature eggs as possible by laparoscopy or direct needle puncture through both walls of the bladder.

Laparoscopy for retrieval of eggs is expensive, usually requiring a hospital stay and general anesthesia. Ultrasonography guided percutaneous aspiration of ovarian follicles under local or basal local anesthesia is a rapid non-traumatic technique. When the urinary bladder is filled with isotonic saline, an ultrasound sector scanner identifies a puncture channel through which a 25 mm-long 18 to 19 gauge needle is directed. The needle traverses both walls of the bladder. A puncture site is made with the follicle projected on the sonographic screen. A vacuum pressure of 100 mm to 170 mm is applied; the follicles are seen to collapse around the advancing needle. The aspirate is collected for each follicle. The direction of the needle may be realigned for each follicle. If the egg is not recovered, the follicular space may be irrigated several times; these flushing attempts frequently dislodge the egg. The expansion and collapse of the follicle may be visualized on the sonographic screen. The oocyte recovery rate may be greater than 50%, and usually the oocyte is recovered intact. With this method repeat procedures are possible in subsequent months without the trauma of entering the peritoneal cavity via laparoscopy.[6]

Frequently, in obese patients and following multiple abdominal operations with many adnexal adhesions, an abdominal transducer is ineffective for retrieving oocytes. In the past several years, vaginal transducers have been designed which have produced excellent images of the ovaries. This is particularly valuable with prolapsed ovaries in the cul de sac as well as for observing an early pregnancy in a deeply retroverted uterus. The Philips 2000 real time sector scanner has provided access to those organs in which abdominal ultrasound would be ineffective.[7]

In 1984, gamete intrafallopian transfer (GIFT), a new technique,[8] was developed which placed both the oocyte and the sperm into the fimbriated end of the fallopian tube. Superovulation is followed by egg retrieval and then both the oocyte and sperm are introduced into the ampulla of the tube where fertilization normally takes place. This is an excellent method for men with low sperm counts and where problems exist with follicular rupture and ovum transfer.

Fertilization and growth of the early blastocyst occurs in the laboratory. Within 24 to 48 hr, the early embryos are replaced in the uterus. Because of the serious complication of prematurity in multiple births beyond triplets, only three fertilized eggs are placed in the uterus. This process of in vitro fertilization is the technique that is used to produce pregnancy in women who have had multiple tubal operations, have lost both tubes from ectopic pregnancies, or have serious tubal disease or, in some instances, this technique is used when the man has a low sperm count. One of the great advantages of the in vitro system is that the number of replaced fertilized eggs may be limited so that early premature deliveries with quadruplets and quintuplets may be virtually eliminated. The management of multifetal pregnancies continues to be a serious problem in all the major intensive perinatal centers in New York State.[9]

ROBERT LANDESMAN, MD
BRIJ B. SAXENA, PhD, DSc

Division of Reproductive Endocrinology
Department of Obstetrics and Gynecology
The New York Hospital-
Cornell University Medical College
1300 York Ave
New York, NY 10021

1. Varma TR: Ultrasound evidence of early pregnancy failure in patients with multiple conceptions. *Br J Obstet Gynaecol* 1979; 86:290-292.
2. Vafai J, Shapiro DL: Perinatal aspects of high multiple gestation. *NY State J Med* 1985; 85:560-562.
3. Hurley DM, Brian RJ, Burger HG: Ovulation induction with subcutaneous pulsatile gonadotropin-releasing hormone: singleton pregnancies in patients with previous multiple pregnancies after gonadotropin therapy. *Fertil Steril* 1983; 40:575-579.
4. Ritchie WG: Ultrasound in the evaluation of normal and induced ovulation. *Fertil Steril* 1985; 43:167-181.
5. Saxena BB, Landesman R: Diagnosis and management of pregnancy by the radioreceptorassay of human chorionic gonadotropin. *Am J Obstet Gynecol* 1978; 131:97-107.
6. Lenz S, Lauritsen JG: Ultrasonically guided percutaneous aspiration of human follicles under local anesthesia: a new method of collecting oocytes for in vitro fertilization. *Fertil Steril* 1982; 38:673-677.
7. Meldrum D, Chetkowski RJ, Steingold KA, et al: Transvaginal ultrasound scanning of ovarian follicles. *Fertil Steril* 1984; 42:803-805.
8. Asch RH, Ellsworth LR, Balmaceda JP, Wong PC: Pregnancy after translaparoscopic gamete intra-fallopian transfer. *Lancet* 1984; 2:1034-1035.
9. Loucopoulos A, Jewelewicz R: Management of multifetal pregnancies: sixteen years' experience at the Sloane Hospital for Women. *Am J Obstet Gynecol* 1982; 143:902-905.

Rounded atelectasis

Rounded atelectasis is an unusual and interesting peripheral pulmonary lesion which was first described in 1890. The condition is a specific form of partial atelectasis of a lobe of the lung, which is located in the periphery immediately adjacent to the visceral pleura, is rounded in shape, and is always associated with disease of the overlying visceral pleura. Although Blesovsky[1] first described this entity in the English language literature under the title "The folded lung" in 1966, previous obscure references to such a condition had been made in French and German reports concerning collapse therapy of tuberculosis. In 1971 Hanke[2,3] used the term round atelectasis in describing the condition, and he proposed a mechanism for its occurrence.

Although the lung is the site of the atelectasis that is visible on chest radiographs, the primary disease process develops in the pleura. Visceral pleural thickening or a visceral pleural plaque always overlies the round area of atelectatic lung. In some cases, a pleural effusion may begin the atelectasis in the early phase of development of the lesion, but it seems more likely that the situation described as "shrinking pleuritis with atelectasis" is most often responsible for this condition.[4] It is postulated that contraction of the pleural fibrosis and plaque results in infolding of the underlying lung and subsequent rounded atelectasis. Tuberculosis and asbestosis are established causes of the pleural disease which can result in rounded atelectasis. The case described by Santos and Morehouse[5] in this issue of the *Journal* may be the first report to document chest trauma with hemothorax as the cause of the pleural disease responsible for a case of rounded atelectasis.

The clinical presentation of patients with rounded atelectasis is non-specific and, in fact, most patients with this condition are asymptomatic. The abnormality is typically detected on a routine chest roentgenogram. The radiographic findings include a peripheral pleural-based parenchymal mass with rounded contour making an acute angle with the pleural surface. Many of these lesions also have a curvilinear density extending from the mass towards the hilum of the lung, described as a comet-tail, and consisting of the relatively compressed bronchovascular structures that supply the atelectatic zone. Rounded atelectasis is most often seen in the lower lobes, where there are accompanying radiographic signs of loss of lung volume, but it has occasionally been described in the lingula and in the middle lobe. In addition, patients with this disorder show evidence of pleural disease, such as plaques and pleural thickening.

The radiologic differential diagnosis of such a density includes many conditions, but a peripheral carcinoma of the lung and malignant mesothelioma are the most serious alternate diagnoses. Once the plain chest films and tomograms—often in the lateral plane—have been obtained, the characteristics of this lesion will usually suggest the diagnosis of rounded atelectasis. An additional piece of helpful information in excluding a malignant process is a percutaneous needle aspiration biopsy of the lung lesion. In a case of rounded atelectasis, this will show only nondiagnostic pulmonary cells.

Since this condition is becoming more widely recognized, the management of most asymptomatic patients in whom the diagnosis of rounded atelectasis has been made on radiographic findings should include careful follow-up, with periodic examinations and repeat chest films. In symptomatic patients with chest pain, or in those in whom the diagnosis is unclear based on radiographs, thoracotomy is indicated. When rounded atelectasis is encountered at surgery, decortication with unfolding of the atelectatic lung is the preferred therapy. Excision of the involved area is done only if the pathological diagnosis is unclear at the time of exploration. It is hoped that an appreciation of the existence of this entity combined with knowledge of the causes of the pleural disease responsible for producing it will result in appropriate management.

ALAN D. HILGENBERG, MD
Assistant Professor
Department of Surgery
Massachusetts General Hospital
Harvard Medical School
Boston, MA 02114

1. Blesvosky A: The folded lung. *Br J Dis Chest* 1966; 60:19-22.
2. Hanke R: Rund atelektasen (kugel-und walzenatelektasen): ein beitrag zur differentialdiagnose intrapulmonaler rundherde. *Fortschr Rontgenst* 1971; 114:164-183.
3. Hanke R, Kretzschmar R: Round atelectasis. *Semin Roentgenol* 1980; 15:174-182.
4. Dernevik L., Gatzinsky P, Hultman E, Selin K, William-Olsson GW, Zettergren L: Shrinking pleuritis with atelectasis. *Thorax* 1982; 37:252-258.
5. Santos GH, Morehouse H: Rounded atelectasis. *NY State J Med* 1985: 85:563-564.
6. Hilgenberg AD, Mark EJ: Case records of the Massachusetts General Hospital. *N Engl J Med* 1983; 308:1466-1472.

The application of non-invasive diagnostic techniques to the pediatric patient

In the early days of fluoroscopy, physicians were often left literally "in the dark" when trying to evaluate a young child. The development of image intensifiers substituted one disadvantage with another. The bulky new machines would envelope the patient to such an extent that small infants would be nearly lost. Although radiation exposure to the patient was lessened, critically ill infants often suffered from a less sterile and cold environment. Computerized image manipulation has been of little added value in treating an infant because of matrix size resolution factors, prolonged imaging time, and radiation exposure. However, such has not been the case for diagnostic ultrasound.

Evaluation using portable devices has added a new dimension to the study of newborn infants. Numerous anatomical regions within the newborn infant can be delineated without risk of ionizing radiation to the patient or surrounding personnel. The ability to follow any abnormal findings allows consideration of more conservative management. This is well illustrated by Amodio et al[1] in this issue of the *Journal.*

The authors describe a palpable gallbladder in a vomiting infant who did not have evidence of intestinal obstruction. A large gallbladder was demonstrated by ultrasound and cystic duct obstruction was found at DISIDA-cholescintigraphy. With close observation by repeated ultrasound examinations, these clinicians were able to provide non-interventional support until resolution of the abnormal findings.

Gallbladder disease is usually not considered when a child presents with an abdominal mass or intestinal symptoms. This is especially true in the newborn infant who is ill or premature. There is often hesitation about subjecting such a delicate infant to manipulative procedures, particularly when the performance of these procedures requires variations in the infant's controlled environment.

It must be remembered that ultrasound is used predominantly for structural detail in identifying size and contour of solid or fluid-filled masses. The determination of functional aspects of anatomy usually requires other methods such as radionuclide studies. Even with ultrasound, the range of normal variation is always of concern when evaluating the pediatric patient. Neonatologists have noted that the gallbladder is occasionally palpable in newborn infants. True hydrops appears as massive enlargement and cystic duct obstruction in various conditions such as septicemia and lymph node disease.[2-8] Enlarged gallbladders can also be detected in infants undergoing prolonged parenteral feedings, but without significant infection or obstruction.[9,10] Leichty et al[10] question the lack of hormonal contractile stimuli in combination with continued bile production as a precipitating mechanism to the appearance of over-distention. A three-year-old child described by Rumley and Rogers[4] had no anatomic point of obstruction, but massive distention of the gallbladder was preceded by dehydration. The 19-day-old infant described by Appleby et al[11] was suspected of having septicemia, but no organism could be identified.

As with other examinations in children, ultrasonographic studies must be evaluated on the basis of a large variation in gallbladder size. Slovis et al[7] presented a table of gallbladder dimensions in normal children which indicated that the longitudinal axis of a two-year-old's gallbladder could be as great as 5 cm. Unfortunately, no indication was given for gallbladder size in infants, although the gallbladder can be disproportionately large in the abdomen of the newborn. A palpable gallbladder that appears prominent at ultrasound examination, but does not demonstrate evidence of obstruction by functional studies such as DISIDA scintigraphy, may represent only a variation of normal. Normal variants should be suspected when spontaneous resolution is noted without specific therapy.

The case presented in this issue of the *Journal* has added another etiologic consideration for gallbladder disease.[1] There was a history of maternal diabetes in the child presented. It must be remembered that patients with diabetes suffer from intestinal motility problems such as gastric retention. Maternal diabetes has been implicated in colon inertia syndrome or the small left colon syndrome where difficulty in passing stool is attributed to neuronal immaturity. Perhaps even delayed resorption of pulmonary fluid, a transient phenomenon seen in newborns of mothers with diabetes, could also be included within this category. Lack of gallbladder contraction on the basis of similar neurogenic dysfunction or immaturity may lead to over-distention.

When a prominent gallbladder is documented by diagnostic ultrasonography, a normal variation may be present. In such instances, a functional study, such as DISIDA scintigraphy, is necessary to confirm associated cystic obstruction. In other instances, gallbladder distention may be a secondary manifestation of a more generalized disease process. It is also important to note that noninvasive procedures can be applied to the newborn infant without requiring the child to be removed from his protective environment. Reliable and repetitive studies can replace more invasive techniques and therapeutic maneuvers.

ARVIN E. ROBINSON, MD
Professor and Chairman
Department of Radiology
College of Medicine
University of South Alabama
2451 Fillingim St
Mobile, AL 36617

1. Amodio JB, Fontanetta E, Cohen M, Rudavsky AZ, Rosenzweig MJ: Neonatal hydrops of the gallbladder. Evaluation by cholescintigraphy and ultrasonography. *NY State J Med* 1985; 85:565-566.

2. Robinson AE, Erwin JH, Wiseman HJ, Kodroff MB: Cholecystitis and hydrops of the gallbladder in the newborn. *Radiology* 1977; 122:749-751.

3. Kumari S, Lee WJ, Baron MG: Hydrops of the gallbladder in a child: diagnosis by ultrasonography. *Pediatrics* 1979; 63:295-297.

4. Rumley TO, Rodgers BM: Hydrop of the gallbladder in children. *J Pediatr Surg* 1983; 18:138-140.

5. Magilavy DB, Speert DP, Silver TM, Sullivan DB: Mucocutaneous lymph node syndrome: report of two cases complicated by gallbladder hydrops and diagnosed by ultrasound. *Pediatrics* 1978; 61:699-702.

6. Bradford BF, Reid BS, Weinstein BJ, Oh KS, Girdany BR: Ultrasonographic evaluation of the gallbladder in mucocutaneous lymph node syndrome. *Radiology* 1982; 142:381-384.

7. Slovis TL, Hight DW, Philippart AI, Dubois RS: Sonography in the diagnosis and management of hydrops of the gallbladder in children with mucocutaneous lymph node syndrome. *Pediatrics* 1980; 65:789-794.

8. Neu J, Arvin A, Ariagno RL: Hydrops of the gallbladder. *Am J Dis Child* 1980; 134:891-893.

9. Barth RA, Brasch RC, Filly RA: Abdominal pseudotumor in childhood: distended gallbladder with parenteral hyperalimentation. *AJR* 1981; 136:341-343.

10. Liechty EA, Cohen MD, Lemons JA, Jansen RD, Schreiner RL: Normal gallbladder appearing as abdominal mass in neonates. *Am J Dis Child* 1982; 136:468-469.

11. Appleby GA, Forestier E, Starck CJ: Hydrops of the gallbladder in the neonatal period. *Acta Paediatr Scand* 1981; 70:117-120.

Reflecting on the lectures of Professor Budd

Teachers of clinical medicine are well aware that their responsibilities to students go far beyond the presentation of complex technical data, or the dry recitation of clinical experiences. Learning is most successful when it also emphasizes the attitudes and behavior necessary to deal with that intricate object, the patient. In medicine there are few rules, other than compassion, and the teacher must expose his own values. It is, essentially, a performing art.

How refreshing it is to read Professor Budd's lectures on obstetrics delivered more than a century ago. Charles Amos Budd was 35 at the time of these lectures, and his students were comparable in age to college undergraduates today. There is no doubt that he was an imposing figure, both at the lecturn and in medical circles in New York. Three years earlier he was one of 14 obstetricians who gathered in the home of T. Gaillard Thomas to found the New York Obstetrical Society. Others in that group included Fordyce Barker, who introduced the hypodermic syringe to America in 1856, as well as Thomas Addis Emmet, Emil Noeggerath, and Edmund Peaslee, all early pioneers in gynecological surgery. Budd himself was noted as a distinguished teacher and lucid speaker, but did little writing.

It is not difficult to take one's place, in imagination, among Professor Budd's eager students and to hear his words 12 decades later. They serve well today. He urged prompt response to the call of a laboring patient, without regard to the reports of others on progress of the case. He advocated catheterization to relieve the obstructed bladder, which was controversial at the time. Particularly delightful are his warnings on blindly cutting the cord beneath a sheet for privacy: "if not careful, you are apt to cut off a finger or a penis." (Doubtless this provoked a reaction from the class.)

Forceps deliveries then, as now, were not without controversy, and Budd strikes a surprisingly modern note: "I strenuously dissuade you from making familiar companions of your instruments because they are not wanted..." He goes on to describe the accoucheur who always has the lever in his pocket as officious and "meddlesome." The term meddlesome obstetrics has survived and is still widely used to describe unwarranted intervention in the normal process of parturition.

Budd's advice on behavior in the presence of the patient constitutes an obstetrical homily. Speaking to these young men with no clinical experience, he advised, "Conduct yourself gently and with dignity." And further, "never enter the chamber with a long face, nor roll up your sleeves before you have notified her of an examination. Sit down alongside of her and attempt cheerfully to engage her in some general conversation and you will easily gain her confidence." Good bedside manners were never better described.

Many readers will wince at Budd's description of "old nurses with side curls and carpet slippers" and "an antipathy to young practitioners," but perhaps this was exaggeration to emphasize the poignance of a personal experience. By contrast, he was tolerant in his consideration of his patient's wishes to allow her husband to remain in the labor room, or if undesired, that he be "delicately put out."

Today, lectures such as these no longer exist, and presentations are heavily supported by audio-visual aids, predistributed notes, and much data. The lecturer would be fortunate indeed if his words received the attention given by Charles Inches to Professor Budd's stimulating remarks.

GORDON W. DOUGLAS, MD
Professor and Chairman
Department of Obstetrics and Gynecology
New York University Medical Center
550 First Ave,
New York, NY 10016

FROM THE LIBRARY

REPORT OF A CASE OF TWO PAIR OF TWINS WITHIN ONE YEAR AND FIVE DAYS

In the course of our profession, there are always cases occurring which are peculiar, if not instructive. A singular case of births occurred in our vicinity in the years of 1855 and 1856. Mrs. L., aged 22, about one year after marriage, was confined on the 20th day of March, 1855, after a labor of six hours, with a pair of twins, a boy and girl.

It appeared that only seven months of the time of gestation had passed: nothing uncommon in the case occurred at that time. She recovered in the course of a few days; had plenty of nurse, still the children were not nourished by it. In four weeks, they both sickened and died within a few days of each other.

On the 25th day of March, 1856, being one year and five days from the first confinement, I was called to visit her; she was in labor about four hours, and gave birth to a pair of girls, full grown, weighed eight pounds and some ounces each. She soon recovered from this confinement, and was about the house in less than two weeks. Her milk was abundant and nourishing, the children grew fleshy and healthy through the season; she nursed them about thirteen months. Soon after her first menstruation in the month of May, 1857, she was violently attacked with peritoneal inflammation, accompanied with high fever, and lived only nine days. Here, in this case, Mrs. L. was the mother of four living children within one year and five days; a most singular succession of births, not occurring perhaps once in a thousand cases; certainly it is the only one that I have seen in my practice.

The mystery of this case, if any, was the violence of the attack so soon after the first menstruation. Was it caused by the circulation being more rapid and freely in those organs preparatory to the third conception?

CHARLES BARROWS, MD
Clinton, Oneida County, NY
Transactions of MSSNY, 1861, p 183

Cyclosporine: preliminary experience in 79 patients with renal transplants

V. A. Tellis, MD, A. J. Matas, MD, D. Glichlick, MD, T. Quinn, PA, R. Soberman, MD, R. Weiss, MD, F. J. Veith, MD

ABSTRACT. Between November 1983 and December 1984, 79 cadaver kidney transplant recipients were treated at Montefiore Medical Center with the immunosuppressive agents cyclosporine and prednisone. One-year patient survival was 98% and kidney survival was 73%. These results, though excellent, were not statistically different from results in this institution immediately preceding the use of cyclosporine. Cyclosporine is a potent immunosuppressive agent, often effective when other agents have failed. However, it is toxic and expensive. Further study and experience is necessary to define its role in cadaver kidney transplantation.

(NY State J Med 1985; 85:531–534)

INTRODUCTION

Cyclosporine, a new immunosuppressive agent, was shown in preliminary trials to increase significantly one-year survival following kidney transplant.[1-3] Subsequently, a similar effect was seen with extra-renal transplants,[4,5] and the drug is now used in transplantation of the kidney, heart, liver, pancreas, lung, and bone marrow. With increased use of cyclosporine, a number of side effects have become apparent. Of major concern is that the drug is both nephrotoxic and hepatotoxic, making the management of both liver and kidney transplants more difficult. In addition, cyclosporine is expensive, with an average initial cost of $6,000 for the first year after a transplant and $3,000 for each successive year. It is important to determine whether the benefits of cyclosporine justify both the risks and the costs. We reviewed preliminary experience with the clinical use of this drug and compared it to historical experience with conventional immunosuppression. Patient and graft survival rates in transplant recipients receiving cyclosporine have been compared with recent results in patients receiving prednisone and azathioprine.

PATIENTS AND METHODS

Since the inception of the transplant program at Montefiore Medical Center, the basic drugs used for immunosuppression following transplantation have been prednisone and azathioprine. The protocol for the use of these drugs has been relatively unchanged since 1977. Prednisone was started at 100 mg daily, and this was tapered to 30 mg per day at two weeks and to 20 mg per day by one month after the transplant. The dose of azathioprine was maintained at 2 mg/kg/day. Patients who experienced rejection episodes were treated with intravenous methylprednisolone (1 g daily for three days). Beginning in January, 1982 the medical center's treatment policy changed in that all patients who suffered rejection episodes were treated according to a strict protocol. Rejection episodes were diagnosed by clinical and laboratory criteria and were confirmed by percutaneous allograft biopsy. For the first and, if necessary, second rejection episode patients were selected at random to receive either antilymphocyte globulin (ALG) (Minnesota) (20 mg/kg/day for 14 days) plus methylprednisolone (1 g daily for three days) or methylprednisolone alone, with ALG added if there was no response to the steroid treatment. Graft and patient survival for these two groups were not significantly different,[6] and for the purpose of this report, all patients who received a transplant during this time period and were treated with conventional immunosuppression will be grouped together.

Prior to the release of cyclosporine by the Food and Drug Administration (FDA) in November 1983, 15 patients received the drug as part of a familiarization protocol. All transplant recipients given cyclosporine underwent pretransplant transfusions. The first three patients received a starting dose of 14 mg/kg/day orally. The subsequent 12 patients received 17 mg/kg/day orally, or an equivalent dose intravenously (5 mg/kg) if there was a concern about the absorption of cyclosporine in the immediate period after surgery. In all of these patients cyclosporine levels were determined but were not used in clinical decision making.

The subsequent 64 patients received cyclosporine according to a consistent protocol. In the immediate period after surgery cyclosporine (5 mg/kg/day) was administered intravenously over six hours. When gastrointestinal absorption of the drug was established, the intravenous medication was discontinued and oral cyclosporine (14 mg/kg/day) was started. In these patients, cyclosporine levels were measured and the dose was adjusted to maintain serum trough levels at 100 ng% to 200 ng% by radioimmunoassay. In addition, pharmacokinetic studies were done on all patients, and when indicated the drug was given in two divided daily doses. Rejection episodes were diagnosed by clinical and laboratory criteria and were frequently confirmed by biopsy. Because cyclosporine nephrotoxicity can be difficult to differentiate from rejection, the dose of cyclosporine was reduced in all patients in whom the diagnosis was in doubt. If serum creatinine levels continued to rise, antirejection treatment was instituted. All rejection episodes were treated with methylprednisolone, 1 g intravenously, daily for three days. If there was no response to antirejection therapy, a 14-day course of antilymphocyte globulin (20 mg/kg/day) was instituted. Cyclosporine was withheld while ALG was being given, but was restarted at the end of the ALG course.

Patient and graft survival were calculated by the cumulative life table method.[7] The statistical significance of the difference in cumulative survival was determined by the logrank test.[8]

From the Department of Surgery, Montefiore Medical Center, Bronx, New York.

Address correspondence to Dr Tellis, Associate Professor, Department of Surgery, Co-director, Transplant Program, Montefiore Medical Center, 111 E 210th St, Bronx, NY 10467.

TABLE I. Characteristics of Patients Who Received Transplants in 1982–1983 and were Given Prednisone/Azathioprine vs Those Who Received Transplants in 1983–1984 and were Given Prednisone/Cyclosporine

	Prednisone/ Azathioprine	Cyclosporine
Number of patients	88	79
Mean age (± standard error)	37 ± 1.0	37 ± 1.3
Percent male	64	75
Race		
% Black	47	37
% Caucasian	42	43
% Hispanic	10	19
Second or third transplants	9	15
High risk factors		
diabetes	11	8
hypertension	18	19
oxalosis	1	—

RESULTS

Demographic characteristics of patients receiving conventional immunosuppression during 1982 and 1983 and cyclosporine during 1983 and 1984 were similar (Table I). For patients receiving prednisone and azathioprine who underwent a transplant between 1977–1979, 1980–1981, and 1982–1983, one-year patient survival was 90%, 92%, and 95% respectively. In comparison, one-year patient survival among recipients of cyclosporine (1983–1984) was 98%. Patient survival was improving before the introduction of cyclosporine and there was no significant difference in patient survival between patients who received transplants in 1982–1983 and who were given prednisone and azathioprine, and those subsequently receiving cyclosporine.

One-year survival for patients who received cadaver donor grafts and were given prednisone and azathioprine during three successive time periods is shown in Figure 1. As with patient survival, graft survival was steadily improving prior to the introduction of cyclosporine. For patients receiving cadaver transplants between 1977 and 1979, one-year graft survival was 36%. Subsequent to the routine use of pretransplant blood transfusions in late 1979, one-year graft survival increased in 1980 and 1981 to 51%. During 1982 and 1983, when patients were randomized for antirejection therapy, one-year graft survival increased to 69%.

In comparison, for those patients who received transplants in 1983 and 1984 together with cyclosporine therapy, one-year graft survival was 73% (Fig 2). One-year graft survival for patients treated with cyclosporine is compared to one-year survival in patients receiving prednisone and azathioprine during 1982 and 1983 (Fig 2). There is no significant difference in graft survival. The reasons for graft loss in all 79 patients receiving cyclosporine are listed in Table II. Rejection remains the major cause of allograft loss.

TABLE II. Reason for Graft Loss in 79 Patients Treated with Cyclosporine

Cause of Graft Loss	Number
Chronic rejection	10
Acute irreversible rejection	4
Primary non-function	2
Cortical necrosis	1
Died with function	1

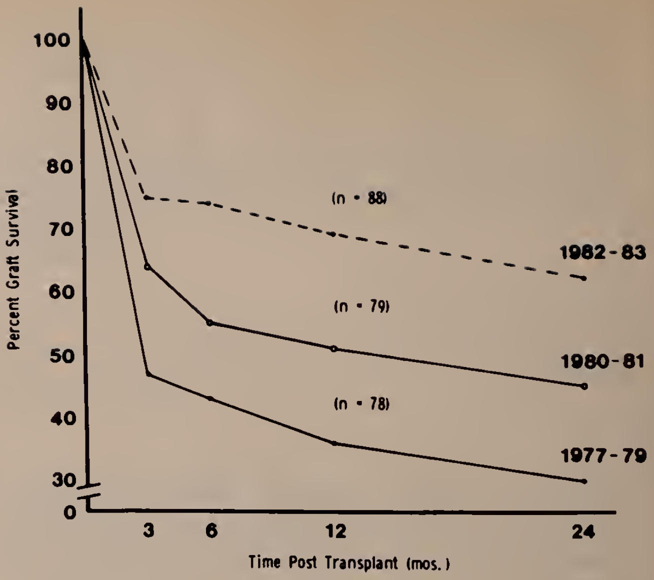


FIGURE 1. Comparison of cadaver kidney graft survival in patients maintained on prednisone and azathioprine during three successive time periods.

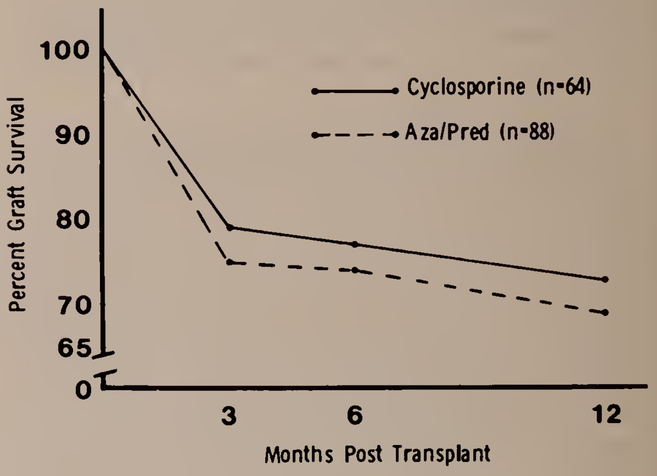


FIGURE 2. Comparison of cadaver kidney graft survival in patients maintained with prednisone and azathioprine in 1982–1983 vs graft survival in those maintained on prednisone and cyclosporine in 1983–1984.

DISCUSSION

To understand the widespread enthusiasm with which cyclosporine was received, it is necessary to know the results of cadaver donor transplantation in the pre-cyclosporine era. For example, the standards committee of the American Society of Transplant Surgeons reported in 1981 on a multicenter trial of 881 transplants.[9] One-year cadaver graft survival was 55%. For comparison, early nonrandomized cyclosporine trials reported a one-year graft survival in excess of 75%, a 20% improvement.[1,3] In addition, in randomized trials from both Canada and Europe, one-year graft survival in patients receiving prednisone and azathioprine was 55% to 65%, whereas patients who were selected at random to receive cyclosporine had significantly better one-year graft survival (75%).[10,11] Cyclosporine was reported to have made a similar impact on heart, heart-lung, lung, pancreas, liver, and bone marrow transplantation.

At the time that cyclosporine was approved by the Food and Drug Administration, we were just completing a randomized trial evaluating antirejection therapy in patients receiving prednisone and azathioprine for maintenance immunosuppression. Data from that study showed a one-year graft survival of 69%.[6] This differed from previous one-year cadaver graft survival at this center and was also significantly better than the one-year graft survival of patients receiving prednisone and azathioprine in the previously mentioned studies. The improvement in graft survival at this institution can be attributed to three phenomena: the institution of deliberate, third-party, random blood transfusions prior to transplantation; the randomized study in which ALG was used to treat rejection episodes; and a significant increase in the number of transplants done annually at this institution, thus increasing the experience of all personnel involved. This raises a question as to how much of a role cyclosporine has played in the overall improvement of kidney transplant survival. Recent reports from many other centers now also show that results with the use of cyclosporine, although excellent, are not statistically different from the centers' previous best results.[12,13] In addition, in a prospective randomized trial, Najarian et al[14] have shown no difference in graft survival between cyclosporine and azathioprine/ALG treated patients.

There are a number of drawbacks to routine cyclosporine use. First, the drug is expensive. Second, it is associated with a number of complications (Table III), the most important for kidney transplantation being that the drug is nephrotoxic. Under the best of circumstances, serum creatinine levels are higher in patients receiving cyclosporine than in those receiving other immunosuppressive agents. Although in the short term this is not associated with permanent renal damage (serum creatinine levels fall if cyclosporine is stopped), it is unknown whether there will be a long-term detrimental effect. In addition, when used in patients with early posttransplant oliguria, cyclosporine will prolong the time that dialysis is required and sometimes result in irreversible renal damage. Finally, although the drug doses used have been higher than those used in renal transplantation, there have been reports of heart and bone marrow transplant recipients maintained on cyclosporine who have developed renal failure.[15,16]

TABLE III. Adverse Reactions Reported to Occur in More Than 3% of Cyclosporine Recipients

System	Reaction
Autonomic nervous	Paresthesia; flushing
Central nervous	Tremor; convulsions; headache
Cardiovascular	Hypertension
Gastrointestinal	Gum hyperplasia; diarrhea; nausea/vomiting; hepatic dysfunction; abdominal discomfort; anorexia
Genitourinary	Renal dysfunction
Skin	Hirsutism; acne

These disadvantages, however, are considered by many centers to be offset by the ability to use considerably lower doses of corticosteroids with cyclosporine than with azathioprine, thus lowering morbidity and mortality rates following transplantation and permitting earlier discharge from the hospital. While this is a common perception, it is important to note that many centers had instituted low-dose steroid regimens prior to the introduction of cyclosporine, with a reported lowering of morbidity and mortality and without lowering graft survival.

A great deal has been learned about cyclosporine since it was approved by the FDA. First, it is not a miracle drug. Current data would suggest that it is the drug of choice for heart, liver, and lung transplantation. In addition, it is probably the drug of choice for patients undergoing a second or third renal transplant.[17] Further studies are necessary to determine whether cyclosporine should be used in all first renal transplants. If the previously used regimens can yield similar results with equal morbidity and mortality but at a much lower cost, it may be prudent to use these regimens initially and switch to cyclosporine if they fail. However, some patients do require cyclosporine. There are numerous anecdotal incidents in which cyclosporine can clearly be credited with the salvage of a kidney that appeared destined for failure using standard immunosuppressive protocols. However, to date there are no means for predicting which patients will require cyclosporine and which will have long-term graft survival with prednisone and azathioprine.

Second, if cyclosporine is given during episodes of posttransplant oliguria, the duration of this disorder appears to be prolonged and diagnosing concurrent rejection or cyclosporine toxicity, or both, is especially difficult. Currently, all patients at Montefiore Medical Center are started on prednisone and azathioprine following a transplant, and when serum creatinine begins to fall the azathioprine is discontinued and cyclosporine is started.

Third, there are some patients who do poorly on cyclosporine, and whose creatinine drops significantly once the drug is stopped. Cyclosporine nephrotoxicity can occur with low or nonmeasurable serum cyclosporine levels. If toxicity is diagnosed and lowering the dose of cyclosporine does not improve renal function, the patient should be switched to azathioprine. If a rejection episode ensues, cyclosporine may be restarted. Finally, there are a few patients in whom the side effects of cyclosporine therapy, such as parasthesias, are particularly disabling. The quality of life for this subgroup improves dramatically when cyclosporine is discontinued.

In summary, there is little question that cyclosporine is a potent and effective immunosuppressive agent and is an important addition to the resources for treating graft rejection. However, further studies are necessary and greater experience is required to better define the role of this drug in renal transplantation.

REFERENCES

1. Calne RY, White DJ, Pentlow BD, et al: Cyclosporine A: preliminary observations in dogs with pancreatic duodenal allografts and patients with cadaveric renal transplants. *Transplant Proc* 1979; 11:860-864.

2. Merion RM, White DJ, Thiru S, Evans DB, Calne RY: Cyclosporine; five years' experience in cadaveric renal transplantation. *N Engl J Med* 1984; 310:148-154.

3. Starzl TE, Weil R 3d, Iwatsuki S, et al: The use of cyclosporine A and prednisone in cadaver transplantation. *Surg Gynecol Obstet* 1980; 151:17-26.

4. Reitz BA, Bieber CP, Raney AA, et al: Orthotopic heart and combined heart and lung transplantation with cyclosporine-A immune suppression. *Transplant Proc* 1981; 13:393-396.

5. Iwatsuki S, Startzl TE, Shaw BW, et al: Long-term use of cyclosporine in liver recipients. Reduction of dosages in the first year to avoid nephrotoxicity. *Transplantation* 1983; 36:641-643.

6. Matas AJ, Tellis VA, Quinn T, et al: Treatment of renal transplant rejection episodes in patients receiving prednisone and azathioprine—a cost effective approach. *Transplantation* 1985; 40:35-39.

7. Peto R, Pike MC, Armitage P, et al: Design and analysis of randomized clinical trials requiring prolonged observation of each patient. II. Analysis and examples. *Br J Cancer* 1977; 35:1-39.

8. Colton T: *Statistics in Medicine*. Boston, Little, Brown & Co, 1976, p 50.

9. Standards Committee of the American Society of Transplant Surgeons: Current results and expectations of renal transplantation. *JAMA* 1981; 246:1330-1331.

10. Canadian Multicentre Transplant Study Group: A randomized clinical trial of cyclosporine in cadaveric renal transplantation. *N Engl J Med* 1983; 309:809-815.

11. European Multicenter Trial: Cyclosporine A as sole immunosuppressive agent in recipients of kidney grafts from cadaver donors: preliminary results of a European multicenter trial. *Lancet* 1982; 2:57-60.

12. Squifflet JP, Pirson Y, Jamart J, et al: Cyclosporine in cadaveric renal transplantation at a center with good results using conventional treatment. *Transplant Proc* 1985; 17:1212-1217.

13. Halloran P, Ludwin D, Aprile M, the Canadian Multicentre Transplant Study Group: Comparison of antilymphocyte globulin-imuran, cyclosporine, and antilymphocyte globulin-cyclosporine therapy for cadaver renal transplantation. *Transplant Proc* 1985; 17:1201-1203.

14. Najarian JS, Fryd DS, Strand M, et al: A single institution, randomized, prospective trial of cyclosporine versus azathioprine-antilymphocyte globulin for immunosuppression in renal allograft recipients. *Ann Surg* 1985; 201:142-157.

15. Shulman H, Striker G, Deeg HJ, Kennedy M, Storb R, Thomas ED: Nephrotoxicity of cyclosporin A after allogenic marrow transplantation. *N Engl J Med* 1981; 305:1392-1395.

16. Myers BD, Ross J, Newton L, Luetscher J, Perlroth M: Cyclosporine-associated chronic nephropathy. *N Engl J Med* 1984; 311:699-705.

17. Rosenthal JT, Hakala TR, Starzl TE, Iwatsuki S, Shaw BW: Second cadaver kidney transplants: improved graft survival in secondary kidney transplants using cyclosporin A. *J Urol* 1984; 131:17-18.

Increased survival time of renal allograft recipients

BARBARA J. BYER, RN, WENDY E. HOY, MD, ALLYN G. MAY, MD, DAVID W. KNUTSON, MD

ABSTRACT. Improved patient and renal allograft survival was observed for 139 transplants performed at the University of Rochester Medical Center from 1977 through 1982, as compared with earlier experience. Improved results may be due to better crossmatching techniques, preoperative blood transfusions, prospective use of mixed lymphocyte cultures for transplants from living related donors, donor-specific transfusions in patients with a stimulatory mixed lymphocyte culture, and the use of anti-thymocyte globulin to treat rejections. These data compare favorably with the published results of larger centers, although we cannot know with certainty whether or not the case mix differed substantially from that of the larger centers.

(NY State J Med 1985; 85:534-538)

The use of cyclosporine A for immunosuppression appears to have resulted in marked improvements in the success rates for renal transplantation.$^{1-3}$ Recent evidence also suggests that rates of patient survival and successful engraftment have improved at many kidney transplant centers *before* cyclosporine became available to these centers.$^{4-6}$ We have analyzed the results with renal transplantation performed with conventional immunosuppression therapy from 1977 through 1982 to ascertain whether similar trends could be observed at the University of Rochester Medical Center and to gain insight into how newer techniques may have affected the results.

PATIENTS AND METHODS

Patients with end-stage renal disease referred to the University of Rochester Medical Center's program for transplantation were each evaluated by a nephrologist, a transplant nurse practitioner, a urologist, a vascular surgeon, and a psychiatrist to insure optimal management of medical and psychiatric problems that might affect the outcome of kidney transplantation. Willing family members, including adult offspring, were tested for histocompatibility with the patient by successively determining ABO blood type and HLA type and performing lymphocytotoxic crossmatches and, finally, a mixed lymphocyte culture (MLC). Complete HLA mismatches were excluded at this stage. When no related donor could be identified, patients were placed on a list of potential recipients awaiting a kidney from a cadaver donor (CD). Recipients for available cadaveric organs were selected on the basis of ABO compatability, a negative lymphocytotoxic crossmatch using most recent serum and up to five earlier serum samples, and the best HLA match. Priority was given to patients with a negative crossmatch who where known to be high responders in crossmatches with a panel of random donor lymphocytes.

Pretransplant nephrectomy was performed in 12 patients who had evidence of chronic kidney infections, in one patient with enlarged polycystic kidneys, and in two patients with uncontrolled hypertension. Pretransplant splenectomy was performed in five patients with hypersplenism, and for other reasons in one patient. Four additional patients had undergone both splenectomy and bilateral nephrectomies prior to 1973, when this had been the medical center's routine practice.

Prior to 1978, blood transfusions were avoided. In December 1978, a policy was initiated to transfuse potential recipients of cadaveric kidneys before surgery with 5 units of blood, and since November 1981, living related donor (LRD) recipients have also received transfusions of at least 2 units from random donors. Beginning in December 1980, three donor specific transfusions were given to LRD recipients who had a stimulatory one-way MLC response (stimulation index > 5).

Immunosuppression and treatment of rejection. Patients received a loading dose of azathioprine (300 mg intravenously or

---

From the Departments of Medicine (Drs Hoy and Knutson), Surgery (Dr May), and Nursing (Ms Byer), University of Rochester School of Medicine and Dentistry, Rochester, NY.

Address correspondence to Dr Knutson, Director, Nephrology Laboratory Research, University of Rochester School of Medicine and Dentistry, 601 Elmwood Ave, Rochester, NY 14642.

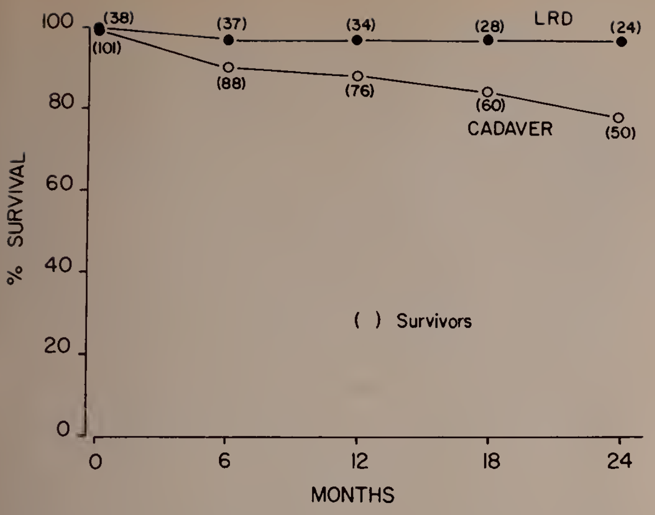


FIGURE 1. Actuarial patient survival following transplantation with kidneys from living related donors (●) versus cadaveric donors (○) (*P* = .06). The numbers in parentheses refer to the number of survivors at each time interval.

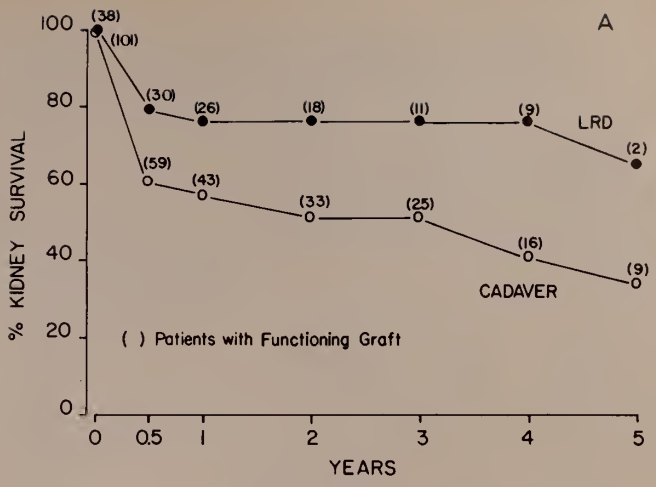


FIGURE 2. Actuarial graft survival following transplantation with kidneys from living related donors (●) versus cadaveric donors (○) (*P* < .02). The numbers in parentheses refer to the number of functioning kidneys at each time interval. Data for all transplants done in 1977 through 1982 are shown.

orally) on the day of surgery and 2 to 3 mg/kg/day as tolerated thereafter, but never more than 200 mg/day. Prednisone was administered at initial doses of 1 to 1½ mg/kg/day, and this was gradually tapered to 10 to 15 mg/day, or an equivalent alternate day dose. Local allograft irradiation (150 rads every other day for four days) was administered the first week following transplantation in CD recipients only. Patients who suffered acute rejection episodes in 1977 through 1981 were treated with methylprednisolone (1 g/day intravenously for three days). Since December 1981, the dose of steroids used to treat rejection episodes was reduced to 3 mg/kg/day for three days of prednisone or methylprednisolone. Beginning in December 1981, anti-thymocyte globulin was used to treat rejection episodes in selected patients. The drug was administered intravenously at 10 to 15 mg/kg/day for 14 days; one patient received 17 doses and another 21 doses.

Statistical analyses. Statistical comparisons of survival data were done by product limit analysis with attention given to both the Wilcoxon (Breslow) statistic, which emphasizes the importance of differences early in the survival curves, and the Savage (Mantel-Cox) statistic, which is more sensitive to late differences.[7] These two methods resulted in very similar *P* values, so only the latter figure is given, since the latter differences were of primary interest. However, because these two statistics were similar, greater confidence can be placed in the interpretation regarding the differences observed. Statistical comparisons for Table III were done by the Chi square test, in which all combinations of data from large centers for the years 1977–1978 and 1982–1983 were tested.

RESULTS

Between 1977 and 1982, 139 kidney transplants were performed in 131 patients (38 LRD and 101 CD). Fifteen percent of the recipients were black and 14% had diabetes mellitus. The mean age of patients was 36.9 years, with a range of 6 to 72 years. Twelve patients were less than 18 years of age and six were younger than age 13. There were 81 male and 58 female patients. Sixteen patients received a second kidney transplant and three had third transplants. Transplants were performed on all candidates from the nine-county area of referral, with the exception of one patient who was referred by another center; this patient, who had leukopenia and in whom the first transplant had failed, was referred in 1982 so that he could receive cyclosporine on an experimental basis.

Patient and graft survival. Patient survival curves are shown in Figure 1. One and two-year survival in the LRD group was 97%; the single death in this group was due to suicide. Two-year patient survival for CD recipients was 78%. The difference in survival between CD and LRD recipients was associated with a chance occurrence of 6% (*P* = .06). Causes of early (<6 months) and late (>6 months) deaths are shown in Table I. Of the 20 deaths, four occurred in patients who had resumed dialysis for 5 to 16 months following graft loss; the causes of death included two cardiovascular events, one stroke, and one suicide. Deaths occurred in nine patients over the age of 50 and in six patients with diabetes mellitus. Pulmonary infection was the major cause of early deaths. The three deaths listed as "medical suicide" (Table I) occurred when patients succumbed after deliberately omitting all medications including insulin, or refused to resume hemodialysis following allograft failure, or both. Allograft survival rates (Fig 2) for all LRD patients was greater than for all CD patients (*P* < .02). LRD graft survival was 76% at one year and 65% at five years. CD graft survival was 57% at one year and 34% at five years.

TABLE I. Causes of Death in Transplant Recipients.[*]

Cause	Early < 6 mo	Late 6 mo to 2 yr
Infection		
Pulmonary	5	1
Sepsis	0	1
Cardiac arrest	2	1
Stroke	0	2
Postoperative hemorrhage	1	0
Suicide	1	0
Medical suicide[†]	1	2
Hepatic failure	0	1
Homicide	0	1
Sudden death	1	0
Total	11	9

[*] Data include four patients who died 5 to 16 months following graft loss and return of the patients to dialysis.

[†] Medical suicide defined as voluntary withdrawal of all medications including insulin or refusal to return to dialysis following graft failure.

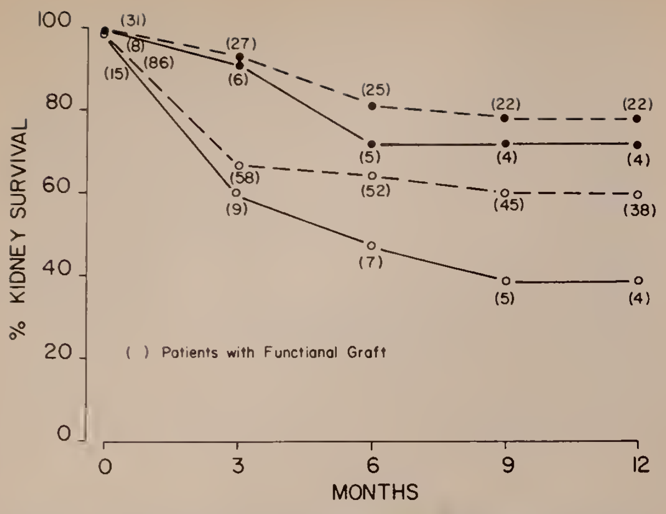


FIGURE 3. Actuarial graft survival following transplantation with kidneys from living related donors (●) versus cadaveric donors (○) in diabetic (solid lines) versus non-diabetic (broken lines) patients. Differences between diabetics and non-diabetics did not reach statistical significance ($.1 < P < .2$).

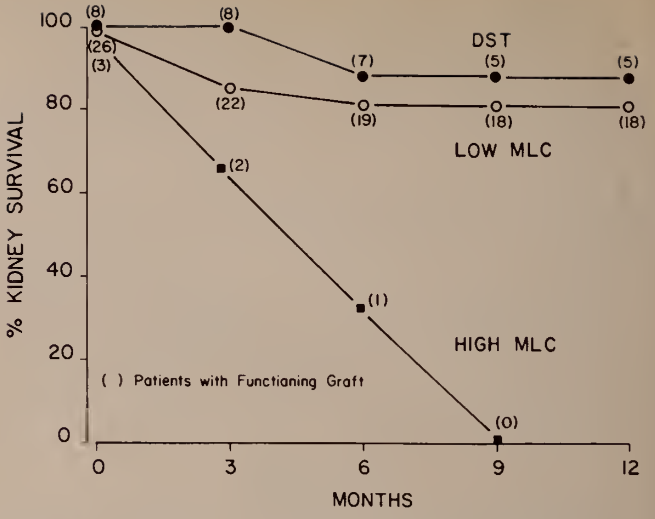


FIGURE 4. Allograft survival for patients with a high stimulation index in the MLC who received donor specific transfusions (●) or did not receive donor specific transfusions (■) and patients with low MLC indices (○). Numbers in parentheses refer to the number of functioning grafts at each time interval.

Figure 3 compares one-year allograft survival in recipients with and without diabetes mellitus. Twenty-two transplants were performed in 19 patients with diabetes (15 CD and 7 LRD recipients). One-year graft survival for LRD transplants in patients with diabetes was 72%, compared with 76% in non-diabetics. Forty percent graft survival at one year was seen in diabetics receiving CD kidneys, compared to 60% graft survival in their non-diabetic counterparts. While these results are in general agreement with the experience in larger series, none of the differences reached statistical significance ($0.1 < P < 0.2$). Causes of allograft loss in diabetics within the first six months of transplantation included two cases of renal artery occlusion within 24 hours of uneventful surgery, one suicide, and seven losses to rejection—including two in which immunosuppressive therapy was withdrawn because of life-threatening infection. Diabetics who receive transplanted kidneys may be at increased risk of early allograft loss due to vascular complications and infection.

Mixed lymphocyte cultures and donor specific transfusions. The predictive value of one-way MLCs for graft survival was analyzed. From 1978 to 1980, the results of MLCs were not used for decision making in LRD evaluations. During this time the graft was lost to rejection within nine months in all three instances in which LRD transplants were done in the presence of an MLC with a stimulation index greater than 5 (Fig 4). Donor specific transfusions[8-10] were begun for prospective LRD transplant patients with a high or stimulatory MLC in early 1981; these results are shown in Figure 4. There were too few data points in Figure 4 to allow statistical analysis. Of 12 potential recipients of one haplotype match kidneys who received donor specific transfusions, four (33%) were sensitized to their donor by transfusion and could not undergo the transplant. Two of the patients had positive T-cell and B-cell crossmatches at 4°C and 37°C, one patient showed a positive T-cell crossmatch only, and one patient's sample was positive in the warm B-cell crossmatch. One recipient was transfused across an Rh barrier after extensive removal of donor erythrocytes by sedimentation, as described by Schweitzer.[11] Allograft survival in patients given donor specific transfusions was 100% at three months and 88% at one year. One graft failed at five months due to rejection. All other recipients, followed 4 to 6 months at the time of this review, were stable with serum creatinine levels of 0.9 mg% to 1.4 mg%.

TABLE II. Treatment of rejection with anti-thymocyte globulin.*

Indication	Number	Success†		
		3 mo‡	6 mo	12 mo
Accelerated rejection in first week	2	1	1	1
Steroid resistant rejection	2	0	0	0
"Camel back" rejection	2	2	1	0
Third rejection within 2 to 5 mo	7	7	4	2
Total	13	9/12	6/7	3/3

* Fifteen courses of anti-thymocyte globulin were given to 13 patients.

† Success = current serum creatinine levels stable at 1.1 to 2.9 mg% (mean, 1.8 mg%).

‡ Time intervals refer to months after completing a course of anti-thymocyte globulin therapy.

Anti-thymocyte globulin. Anti-thymocyte globulin was used to treat 13 patients with acute allograft rejection. Two patients received two courses of the medication (Table II). Two patients who had previously experienced early allograft loss also experienced early accelerated rejection of their second kidney and were treated with the drug. Rejection was successfully reversed in one of these two patients. Two patients who had experienced "camel back" rejections had apparent improvement in renal function. Subsequently, one of these patients resumed hemodialysis four months following therapy with anti-thymocyte globulin and the other has excellent renal function after 17 months. Seven patients were treated for a third rejection two to five months following transplantation. One of these grafts in the only LRD recipient treated with anti-thymocyte globulin failed after five months. In the remaining six patients followed for a minimum of three months after treatment, serum creatinine levels ranged from 1.2 mg% to 2.9 mg%, with a mean of 1.8 mg%. One-year allograft survival in all patients treated with this medication was 64%.

DISCUSSION

We have reviewed the experiences of the University of Rochester Medical Center in kidney transplantation for six years beginning in 1977 to determine whether reported improvements in both patient and graft survival were evident at this center. Table III compares the results for 1977 and 1978 with those published by the Standards Committee of the American Society of Transplant Surgeons,[12] as well as with more recent experiences from 1982 and 1983 at this medical center. The Standards Committee surveyed seven large and experienced centers for data on renal transplants for 1977 and 1978. The success rates at this center for LRD and CD transplants in 1977 and 1978 compared favorably with those of the larger centers. Results from 1982–1983 for CD allograft survival also compare favorably with those recently reported by the Canadian Multicentre Transplant Study Group for patients receiving prednisone and azathioprine.[4] Advantages of convenience and personalized service make it preferable for patients to have this procedure performed at a center located near the patient's home. The data presented in this report should reassure referring physicians that intermediate size centers such as this one can achieve transplant success rates comparable to larger centers. Since this center is the sole transplant program for the nine-county area that surrounds Rochester, it is not likely that the case mix differs greatly from that of the larger centers mentioned. However, transplants performed in relatively healthier patients could contribute to the success rates at this center or to those of the reference centers.

Recipient mortality rates have also improved since the early 1970s.[6] This may be due to improved techniques, better recognition and treatment of infections, and less aggressive treatment of severe or recurring episodes of rejection. However, patient survival rates may now be reaching a plateau; this may reflect underlying medical problems and the increasing mean age of patients with end-stage renal disease.

Graft survival rates have also improved since the early 1970s, when one-year graft survival for CD transplants was 45% to 50%.[13] Graft survival rates appear to have continued to improve in more recent years.[5] The more recent results from this medical center in 1982 and 1983 may also support this contention (Table III) in that one year survival for CD donors in 1982–1983 was significantly better than the results for large centers in 1977–1978 ($P < .05$). However, the 1982–1983 results were not statistically different from the 1977–1978 results at this medical center ($P = .09$). Improved graft survival at this center is attributed to the use of preoperative blood transfusions,[3] the prospective use of the MLC for LRD transplants, donor specific transfusions in selected patients with stimulatory indices in the MLC, the use of anti-thymocyte globulin to treat rejection, and improved methods for tissue typing and performing crossmatches.

The data in this report underscore the poorer prognosis for cadaveric graft survival in patients with end-stage renal disease due to diabetes mellitus. In contrast, graft survival in diabetics that received an LRD kidney was similar to graft survival in non-diabetics. The use of LRD kidneys over CD kidneys may confer a greater *relative* advantage for diabetic patients, and this advantage may outweigh the theoretical risk of later developing diabetes in donors with similar HLA types as the recipients.[14] The hospital committee on transplantation of the University of Rochester Medical Center will accept relatives of diabetics as kidney donors if the donor is over 25 years of age and at least 10 years older than the age at which the prospective recipient developed diabetes.

Among recipients of renal transplants in this study, diabetics had an increased incidence of allograft failure due to vascular thrombosis and infection. Three of four deaths from suicide or "medical suicide" occurred in diabetics. Diabetic patients often present with multisystemic problems. Satisfactory therapy may not yet be available for many of these patients when they develop end-stage renal disease.

TABLE III. Overall patient survival and kidney graft survival at one year for seven large centers(12) and the University of Rochester Medical Center

	Large Centers (n = 7)	University of Rochester Medical Center	
	1977–1978	1977–1978	1982–1983
Mean number of transplant procedures/year (range)	63 (40–148)	21 (20–22)	24 (21–27)
Percent patient survival at one year			
LRD	95.1 ± 3.7	100	100
CD	88.6 ± 7.7	88.4	93.2
Percent graft survival at one year			
LRD	78.6 ± 8.5	85.7	90
CD	55.0 ± 5.7	56.9	73.4

* $P < .05$ compared with CD graft survival for large centers by Chi square test; all other horizontal comparisons of the data were not significant ($P > .1$).

The outcome of LRD transplants after donor specific transfusions for patients with stimulatory MLCs is encouraging. Without such therapy, the success rates appear to be no better than for CD transplants.[10] However, recipient sensitization to the potential donor can become an obstacle to performing the transplant in about one third of published cases[9] and in the population reported here.

Anti-thymocyte globulin therapy is expensive and inconvenient for the patient, and we cannot assess the long-term benefit at this time. Serious side effects of hypotension and respiratory distress were encountered in one patient. Treatment with this medication appeared to benefit some patients with severe and recurring rejection reactions.[15-16]

Acknowledgments. Statistical analyses were performed by Paul J. Carpenter, PhD, University of Rochester Medical Center. Secretarial assistance was provided by Lynn Carpenter and Mary Mangefrida.

REFERENCES

1. Merion RM, White DJ, Thiru S, Evans DB, Calne RY: Cyclosporine: five years' experience in cadaveric renal transplantation. *N Engl J Med* 1984; 310:148–154.
2. Rosenthal JT, Hakala TR, Iwatsuki S, Shaw BW Jr, Starzl TE: Cadaveric renal transplantation under cyclosporine-steroid therapy. *Surg Gynecol Obstet* 1983; 157:309–315.
3. Terasaki P, Perdue S, Sasaki N, Mickey MR, Whitby L: Improving success rates of kidney transplantation. *JAMA* 1983; 250:1065–1068.
4. Stiller CR: Update for the Canadian Multicentre trial of cyclosporine in renal allografts. *N Engl J Med* 1984; 310:1464–1465.
5. Krakauer H, Grauman JS, McMullan MR, Creede CC: Recent US experience in the treatment of end-stage renal disease by dialysis and transplantation. *N Engl J Med* 1983; 308:1558–1563.
6. Vollmer WM, Wahl PW, Blagg CR: Survival with dialysis and transplantation in patients with end-stage renal disease. *N Engl J Med* 1983; 308:1553–1558.
7. Dixon WJ (ed): *BMDP Statistical Software.* California, University of California Press, 1981.
8. Salvatierra O Jr: Experiences and future considerations with donor-specific blood transfusions in living-related transplantation. *Am J Kidney Dis* 1981; 1:119–124.

9. Salvatierro O Jr, Amend W, Vincenti D, et al: Pretreatment with donor-specific blood transfusions in related recipients with high MLC. *Transplant Proc* 1981; 13:142–149.

10. Cochrum K, Hanes D, Potter D, et al: Improved graft survival with donor-specific transfusion pretreatment. *Transplant Proc* 1981; 13:190–193.

11. Schweizer RT, Bartus SA, Silver H, McLean RH: Kidney transplantation using donor-specific blood transfusions despite Rh incompatability. *Transplantation* 1981; 32:345–347.

12. Standards Committee of the American Society of Transplant Surgeons: Current results and expectations of renal transplantation. *JAMA* 1981; 246:1330–1331.

13. Advisory Committee to the Renal Transplant Registry: The 13th report of the human renal transplant registry. *Transplant Proc* 1977; 9:9–26.

14. Gamble DR: An epidemiological study of childhood diabetes affecting two or more siblings. *Diabetologia* 1980; 19:341–344.

15. Hardy MA, Nowygrod R, Elberg A, Appel G: Use of ATG in treatment of steroid-resistant rejection. *Transplantation* 1980; 29:162–164.

16. Shield CF, Cosimi AB, Tolkoff-Rubin N, Rubin RH, Herrin J, Russell PS: Use of antilymphocyte globulin for reversal of acute allograft rejection. *Transplantation* 1979; 28:461–464.

AROUND THE STATE

VA MEDICAL CENTER CHANGES DISPENSING POLICY

Effective October 1, 1985 the Veterans Administration Medical Center in Syracuse, New York will change its drug dispensing policy for the veteran population it serves. Formerly, the facility provided any prescription or non-prescription drug ordered by a physician for treatment of an eligible veteran. However, drug availability will now be limited to those drugs currently listed as Medicaid Reimbursable Drugs by the New York State Department of Health. This measure will enable the Syracuse Veterans Administration Medical Center to continue to provide its patients with high quality pharmaceutical services despite a substantially reduced level of funding for the pharmacy program.

The veterans who will be affected by this move reside in the following eighteen counties of central New York: Broome, Cayuga, Chemung, Chenango, Cortland, Herkimer, Jefferson, Lewis, Madison, Oneida, Onondaga, Oswego, Schuyler, Seneca, St. Lawrence, Tioga, Tompkins, and Wayne. Physicians who treat veteran patients residing in these areas are requested to consider these restrictions when writing prescriptions to minimize both confusion on the part of the patient, and the number of physician-pharmacist interactions that will occur as these restrictions are implemented.

THE CPC

A 45-year-old man with weight loss, fever, chills, and diarrhea

Case Presentation

David Hirsh, MD (Medical Resident): A 45-year-old man was admitted for the first time to Long Island Jewish-Hillside Medical Center with complaints of weight loss, nocturnal chills, and bright red blood from the rectum over the preceding eight months.

Eight months earlier, in a setting of otherwise good health, he had noted loss of appetite, and occasional night sweats and chills; in the subsequent eight-month interval, he had progressively lost 15 pounds. He continued his usual occupation as a horse trainer in Florida. He noted occasional black tarry stools for which he finally consulted a physician three months prior to the present admission. He was found to be anemic; on admission to another hospital, diagnostic evaluations, including a gastrointestinal series, barium enema, sigmoidoscopy, and bone marrow biopsy, revealed a gastric ulcer and a hypoplastic bone marrow which remained unexplained. Following several blood transfusions, he was discharged approximately two weeks prior to his admission to this hospital.

During this two-week interval, he continued to have chills, and loose yellow stools developed which tended to float but showed no mucus or blood. A cough productive of whitish sputum and retrosternal pleuritic chest pain also developed. He sought treatment 24 hours following an episode of passing bright red blood per rectum.

His medical history included a seizure disorder over a 20-year period; he was treated with diphenylhydantoin until one week prior to the present admission. He had had one episode of "yellow jaundice" at age 19 years. He claimed that it was due to an allergy to penicillin; he admitted to intravenous drug abuse, including intravenous cocaine six months prior to this admission. He had been in the habit of drinking one or two pints of alcoholic beverage daily for the preceding 27 years; he had a 50 pack-year history of cigarette smoking.

His father had died of cancer of unstated type; one brother had been admitted to a hospital for treatment of tuberculosis 20 years previously. No other family medical history was known.

A review of the patient's physical condition was unremarkable, except for urinary hesitancy and dribbling. He claimed to have had clubbing of fingers all of his life.

Physical examination on admission to this hospital revealed an extremely cachectic black man in no apparent distress. His blood pressure was 90/60 mm Hg; pulse rate, 72/min; respiratory rate, 16/min; temperature, 98°F. His skin had many scars as well as circular 1-cm, deeply pigmented macules over the left lower leg. There was bilateral temporal muscle wasting. The lungs were clear on percussion and auscultation. The heart rhythm was regular; the sounds were normal; there was a grade II/VI systolic ejection murmur over the left lower sternal border; there were no rubs or gallops. The liver span was about 6 cm. The genitalia were not remarkable. The rectal examination was negative; the prostate was within normal limits. The stool was negative on guaiac test for blood. There was marked clubbing of the finger nails. The peripheral arterial pulses were normal; the neurologic examination was not remarkable.

Urinalysis revealed a specific gravity of 1.011, a trace-positive test for protein, and zero to one white blood cell on microscopic examination. A 24-hour specimen of urine, with a volume of 3,430 mL, contained 2.3 g of protein (normal range, 150 mg per 24 hours) and 652 mg of creatinine, at the lower limit of normal for a 40-kg man (normal range, 15 to 25 mg/kg body weight).

The hemoglobin level was 8.1 g/dL; hematocrit, 23.5%; white blood cell count, 3,000/mm^3 with 52% segmented polymorphonuclear leukocytes, 11% band forms, 19% lymphocytes, 13% monocytes, 4% eosinophils, and 1% atypical lymphocytes. The blood smear showed normocytic normochromic red blood cells, with a few atypical lymphocytes and a few helmet-type red blood cells. The mean corpuscular volume was 73 cubic micra (normal range, 80 to 94 cubic micra). The platelet count was 73,000/mL^3. The prothrombin time was 11.1 seconds (control, 11.3 sec); the partial thromboplastin time was 41.1 sec (normal control, 25 to 35 sec). The serum fibrinogen level was 390 mg/dL (normal range, 150 to 350 mg/dL). The erythrocyte sedimentation rate was 84 mm/hr. The VDRL test was negative. Stool was negative for blood on guaiac test.

The serum electrolyte levels were as follows: sodium, 133 milliosmoles per liter; potassium, 3.8 mmol/L; chlo-

---

Based on the clinicopathologic conference (CPC) held at the Long Island Jewish-Hillside Medical Center, New Hyde Park, NY, on November 16, 1984.

Case Presentation: David Hirsh, MD, Medical Resident; *Clinical Discussion:* Joseph Shapiro, MD, Attending Physician; *Pathology Presentation:* Belinde Aftalion, MD, Staff Pathologist; *Conference Moderator:* Herbert Diamond, MD, Chairman, Department of Medicine; *Conference Coordinator:* Sylvia Belenko.

Editor: JOHN T. FLYNN, MD

Address correspondence to Dr Flynn, Consulting Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Rd, Lake Success, NY 11042.

ride, 104 mmol/L; and bicarbonate, 19 mmol/L. Other serum chemical values were as follows: glucose, 87 mg/dL; blood urea nitrogen, 26 mg/dL; creatinine, 1.2 mg/dL; amylase, 110 units/mL (normal range, 4 to 25 units/mL); uric acid, 5.1 mg/dL; calcium, 7.4 mg/dL; and phosphate, 1.5 mg/dL. The serum cholesterol was 98 mg/dL; serum alkaline phosphatase, 45 units/L (normal range, 13 to 39 units/L); serum glutamic oxaloacetic transaminase (SGOT), 19 units/mL (normal range, 10 to 40 units/mL); lactic dehydrogenase, 125 units/mL (normal range, 60 to 120 units/mL); and serum creatine phosphokinase, 62 microunits/mL (normal range for males, 5 to 55 microunits/mL). The serum vitamin B_{12} and folate levels were within normal limits; the serum iron level was 40 μg/dL (normal, 50 to 150 μg/dL); and the total iron binding capacity was 165 μg/dL (normal range, 235 to 450 μg/mL). The serum magnesium level was 1.3 mg/dL. The total serum protein was 6.8 g/dL (albumin, 2.9 g); serum protein electrophoresis indicated albumin, 43%; alpha 1 globulin, 5.8%; alpha 2 globulin, 10%; beta globulin, 14.15%; and gamma globulin, 27%. The serum cortisol level was 45 μg/dL (normal range, 5 to 20 μg/dL).

The arterial blood gas determinations on room air were pH, 7.22; P_{CO_2}, 22 mm Hg; and P_{O_2}, 65 mm Hg; oxyhemoglobin saturation was 87%.

A serum test for hepatitis B surface antigen was negative.

A chest film was not remarkable; the cardiac silhouette was within normal limits (Fig 1). The electrocardiogram indicated a regular sinus rhythm and no changes suggestive of an acute process.

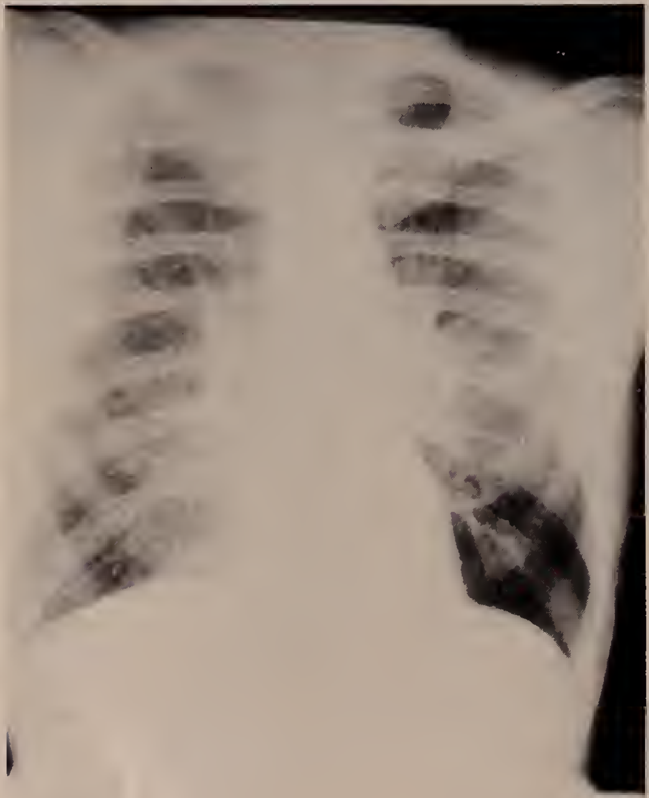


FIGURE 1. Chest film on admission showing normal cardiac silhouette and bilateral increase in bronchovascular markings.

Throughout the hospital stay, 20 sets of blood cultures were reported as negative, except for one culture that grew *Staphylococcus epidermidis*, thought to be a contaminant. The purified protein derivative skin test for tuberculosis was negative; there was also no dermal response to Candida or to mumps antigens.

The patient's early hospital course was notable for recurrent fever, spiking daily to 104°F to 105°F. A bone marrow biopsy revealed focal lymphocytosis and eosinophilia, and an increase in erythroid precursors; there were normal iron stores and no granulomas were seen. Routine cultures of the bone marrow specimen and smears for acid-fast bacilli were negative.

The patient's diarrhea continued; he was given many packed red blood cell and platelet transfusions. On the fifth hospital day, since he continued to have persistently recurrent spiking fever, antituberculosis treatment with isoniazid and rifampin was begun as a therapeutic trial. A liver-spleen scan on the sixth hospital day was reported to be normal; a gallium 67 scan showed focal mediastinal uptake. The patient developed a tendency to hypocalcemia which persisted throughout his hospital stay.

The differential diagnostic considerations at this time included tuberculosis, sarcoidosis, lymphoma, pre-leukemia, and possibly the acquired immune deficiency syndrome (AIDS). However the serum angiotensin-converting enzyme level was normal.

By the eighth hospital day, the patient's pancytopenia worsened; the hemoglobin level was 10 g/dL; hematocrit, 29%; total white blood cell count, 1,400/mm^3 with 52% polymorphonuclear leukocytes, 11% band forms, 22% lymphocytes, 5% monocytes, and 10% eosinophils. Computerized tomography (CT) of the chest showed a 2-cm left hilar mass, bilateral pleural effusions, and localized atelectasis. The following day, because of continued deterioration of his nutritional status, hyperalimentation was begun.

On the 11th hospital day, a sonogram was done in an effort to assist thoracentesis, but the effusion could not be located. He was noted to have oral candidiasis which was treated with local nystatin.

The pancytopenia continued to worsen; the hemoglobin level fell to 7 g; hematocrit, 21%; white blood cell count, 700/mm^3; and platelet count, 36,000/mm^3. With persisting temperature elevations to 105°F, treatment was continued with red blood cell transfusions, isoniazid, rifampin, and hyperalimentation.

On the 13th hospital day, the patient developed sudden onset of dyspnea and tachypnea; the lungs, however, were clear on physical examination. He became very lethargic, and a scheduled mediastinoscopy had to be cancelled. On the following day, the arterial blood gas values on room air were pH, 7.43; P_{CO_2}, 19 mm Hg; P_{O_2}, 90 mm Hg; oxyhemoglobin saturation of 90%; and a bicarbonate level of 12 mmol/L. The serum glucose level was 460 mg/dL, and later reached levels of 700+ mg/dL, at which point insulin was administered; hypocalcemia persisted. The patient was considered to have metabolic acidosis with no anion gap, most probably on the basis of the hyperalimentation, which was discontinued.

On the 15th hospital day, a tube was introduced into the trachea because of the persisting acidosis, which was believed not to be due to diabetes mellitus. Treatment now included ampicillin, oxacillin, and gentamicin; the electrocardiogram was noted to be compatible with an anteroseptal myocardial infarction, but the creatine phosphokinase and MB fractions were within normal limits. The patient's temperature fell to 95.1°F; the stools were now positive for blood on guaiac test, and the pancytopenia continued the need for repeated transfusions. There was improvement in mental status, though he remained disoriented. Hemodynamic evaluation by means of a Swan-Ganz catheter indicated a cardiac output of 4.58 L/min; right auricular pressure of 15 mm Hg (normal range, 2 to 8 mm Hg); right ventricular systolic and diastolic pressures of 30/15 mm Hg (normal range, 15–30/2–8 mm Hg); pulmonary artery pressures, 30/20 mm Hg (normal range, 15–30/4–12 mm Hg); and pulmonary capillary wedge pressure 12 mm Hg (normal mean, 2–12 mm Hg).

On the 17th hospital day, he developed diffuse abdominal pain. The total serum bilirubin level at that time was 12.7 mg/dL (direct, 11 mg); SGOT, 130 U/L; and serum glutamic pyruvic transaminase (SGPT), 47 U/L. Treatment with isoniazid and rifampin was discontinued; however, the following day the serum bilirubin level was 14 mg (direct bilirubin, 11 mg); serum alkaline phosphatase, 122 U/L; SGOT, 29 U/L; and SGPT, 1,785 U/L. The hemodynamic variables were stable, and he had a temperature of 99.2°F. However, his hematocrit had dropped abruptly from 31.5% to 24.8% over a 24-hour period; a sonogram of the right upper quadrant did not show abdominal masses, biliary obstruction, or hematoma formation; bilateral pleural effusions were noted.

Over the next two days, his continued improvement justified discontinuance of intratracheal intubation, but subsequent deterioration of his status with recurrent respiratory distress required prompt replacement of the intratracheal tube.

On the 21st hospital day, mediastinoscopy was performed; a lymph node biopsy showed only non-specific inflammatory changes, without granuloma formation or evidence of lymphoma. An acid-fast stain of material from the lymph node was positive for acid-fast bacilli.

On the 23rd hospital day, the patient developed renal tubular acidosis. Treatment with fluid restriction was not successful; urine output continued to exceed fluid intake, with continued loss of urine electrolytes and bicarbonate.

By the 25th day, the serum bilirubin levels were returning toward normal; treatment with isoniazid and rifampin was reinitiated, together with ethambutol and streptomycin. On the following day, a liver biopsy showed cholestasis and pericardal inflammation; a blood smear with acid-fast stain was negative for tubercle bacilli. Thereafter, the patient's general condition worsened; he began to deteriorate mentally, was suspected at one point of having developed an aspiration pneumonia, and experienced episodes of hypotension requiring intravenous fluids and vasopressor therapy with dopamine intravenously. Hyperalimentation was reinitiated. Despite all efforts, he lapsed into stupor, and renal tubular and respiratory acidosis worsened. He developed persistent hypotension with bradycardia, and on the 34th hospital day he was successfully resuscitated from an episode of cardiac arrest. On the following day, however, the patient expired.

CLINICAL DISCUSSION

Joseph Shapiro, MD (Attending Physician): This is a difficult and complex case. The patient's illness progressed for a period of about nine months. The illness was mainly characterized by chills and intermittent but prevailing fever. The patient's gastrointestinal symptoms can be summarized by indicating that they suggest malabsorption, with yellow fatty floating loose stools but without mucus, pus, or intermingled blood. His pulmonary symptoms included pleuritic pain and cough; whatever disease he had at that time involved the pulmonary parenchyma and the pleura. He had minor urinary symptoms, with hesitancy and dribbling and a reported normal prostate examination. He had a progressively severe course and subsequently died.

An important fact in his medical history is his intravenous drug addiction. Another item of considerable importance is the family history of tuberculosis. It is obvious from the protocol that he was considered by our clinicians to have tuberculosis, but that diagnosis does not make sense to me. A period of eight to nine months is too long for a history of miliary tuberculosis; he would have been dead many months previously. If he had a parenchymal pulmonary tuberculous infection with such a history and with recurrent fever, there should have been clear evidence of caseating cavitating pulmonary disease, with tuberculous aspiration bronchopneumonia. In fact, however, his chest films were normal. I think we can dismiss the possibility of tuberculosis as an explanation of his underlying disease. The fact that a few acid-fast bacilli were found in a lymph node biopsy from the thorax does not mean very much. I believe that one never really recovers from pulmonary tuberculosis. Rather, a few bacilli in the lung or lymph nodes become locked up, and when one becomes seriously ill with another disease, the locking-up process begins to deteriorate, and some of the bacilli will become visible. But that process would not account for the kind of progressive illness which this patient experienced.

It is fashionable nowadays to talk about the acquired immune deficency syndrome in intravenous drug abusers. I doubt that AIDS is present in this patient; the entity that kills the intravenous drug addict is *Pneumocystis pneumonia*, which this patient did not have. He had few pulmonary symptoms and his chest films showed very little. So I am not going to accept the diagnosis of AIDS.

What about the diarrhea? Patients with this much loss of resistance can be susceptible to all sorts of protozoal and bacterial infections of the gastrointestinal tract. In the protocol that I received, the statement was made that the stools were "negative"; but the stools were not described, and the protocol does not indicate what tests were performed on them. The house officer indicated that a number of stool cultures were performed and that the stools were examined for different types of ova and parasites. I do not think that this patient had inflammatory bowel disease; the illness is much too severe, and there was no pus or blood described in the stool. Some of the gastrointestinal findings, such as the more acute episodes of bleeding, are not explained. In spite of the fact that this patient was admitted because of rectal bleeding, there is no mention of a sigmoidoscopy in the protocol.

I can not think of any gastrointestinal neoplasm that would behave in this fashion. A lymphoma involving the intestine would be unusual in a 45-year-old black man; lymphoma of the bowel is a disease most often seen in near-Eastern-Arabic-Shephardic Jews. There should have been lumps in the abdomen, which were not felt; and the disorder should have been noted in the CT scan of the abdomen, which was not seen. A neoplasm of the bowel appears to have been unlikely in this patient.

The prominent disease among intravenous drug abusers is bacterial endocarditis, which is what this man seems to have had. Following is the evidence in favor of that condition. A cardiac murmur was noticed on admission to the left of the xiphoid process of the sternum, but it was not noted whether this murmur increased with inspiration or how it behaved otherwise. It is only mentioned once, but this murmur suggests tricuspid insufficiency. It would have been better if the clinicians had had the patient take a deep breath, and had heard the murmur grow louder. The electrocardiogram on admission was said to have shown no acute symptoms, but maybe it showed incomplete bundle branch block. I did not see it.

What about the subsequent electrocardiogram? Suddenly from an initial test which was reported to show no acute findings, we are told that the later ones show anteroseptal infarction, but with enzyme tests negative for myocardial infarction. About 80% to 90% of patients with prolonged bacterial endocarditis develop myocardial lesions, usually embolic; so there would be every reason why there would have been some electrocardiographic changes. He could also have had a large embolus which plugged a coronary artery, but I don't think so because there was not enough complaint of chest pain.

Also, where would the 2 grams of albumin be coming from in the 24-hour urine? That is not normal; I consider this also to be evidence for bacterial endocarditis. It is not uncommon that patients with bacterial endocarditis have lesions in the glomeruli which used to be called focal embolic glomerulonephritis, but now are thought to be due to antigen-antibody deposition and not to emboli. I think he will be found to have some hydronephrosis and some segmental arteriolitis of the glomeruli, with a low specific gravity in the urine. In the presence of a long-standing chronic infection, renal amyloidosis would have to be considered.

What caused the tremendous jump in blood sugar toward the end of his illness? Could he not have had an embolus into his pancreaticoduodenal artery? That would have been a good time to do an amylase test and tests for blood lipids, to see if there wasn't some embolic damage to the pancreas.

What characterizes the endocarditis of drug abusers? The disease is primarily due to staphylococcus, secondly to fungi, and thirdly to unusual gram-negative organisms. This last has been described in an outbreak in San Francisco some years ago.[1] Staphylococcus is the most prominent organism; the lesions are located on the right side of the heart, primarily in the tricuspid area.

What about the clubbing? He is supposed to have had it all of his life, but people most often are not aware of clubbing of their fingers. I am inclined to think that his clubbing was not old, and that it was additional evidence that this illness was bacterial endocarditis.

I do not know why he had anemia. The early finding of hypoplastic bone marrow at the previous hospital suggested severe marrow suppression with anemia, a low white blood cell count, and thrombocytopenia. At the same time normal serum levels of fibrinogen, iron, vitamin B_{12}, and folate were noted. Possibly the history of diphenylhydantoin use has some significance here; Williams' textbook of hematology mentions diphenylhydantoin as a cause of aplastic anemia.[2] I thought that was a good possibility, but by the time he reached this hospital, he had already stopped the diphenylhydantoin. The later finding of an active bone marrow alters this initial picture and leaves overwhelming infection as the best possibility to explain the anemia, the leukopenia, and the thrombocytopenia that were seen at this hospital.

With respect to the blood smear, it was reported that helmet cells were present. These do not occur in most anemias, but they do occur in diseases in which the red cells are mechanically broken down. They occur in the presence of abnormal heart valves and of disseminated intravascular coagulation, which this patient did not have. His serum fibrinogen level is not decreased, in fact it is a little bit above the high range of normal. I think that the helmet cells are evidence in favor of bacterial endocarditis; he had a speedy flow of blood on the right side of the heart with much turbulence, so that he also had hemolytic anemia. I would have liked to have seen tests for haptoglobins and for reticulocytes, and the results of Coombs tests, but I would have expected them to be normal or negative. They are not listed in the protocol.

What other findings were suggestive of endocarditis? Where are the positive blood cultures, which he absolutely should have had? There aren't any. One culture out of 20 was positive for *Staphylococcus epidermidis;* that organism used to be known as *Staphylococcus albus*. Dr Weinstein discusses, in his section on bacterial endocarditis in Braunwald's text,[3] some of the reasons for negative cultures in active endocarditis. One of the possible reasons is the high level of antibactericidal antibody in the patient's blood which interferes with the growth of the organism in the culture; and Dr Weinstein mentions a technique for removing complement from the blood so that the organisms might grow in culture. I think that is what had been going on here. There is plenty of evidence for increased antibody formation; his serum globulin is well above normal, though no latex fixation test was done, which is positive in 50% of patients with bacterial endocarditis. This test might have helped determine a diagnosis. The serum protein electrophoresis showed that the gamma globulin was quite high in this patient. It is not stated specifically, but I imagine that the gamma globulin was a broad-based fraction. The results of tests for such factors as Raji cells or antibody-antigen complexes would have been of interest, but they are not terribly important, and I never ask for such tests myself.

What about the infecting organism? If this patient had an eight or nine month illness due to staphylococcus infection, the infecting agent would have had to be *Staphylococcus epidermidis*. This is not a lethal or destructive organism; a coagulase test is usually negative. Dr Weinstein reported on several of his patients with endocarditis who were ill for as long as eight or nine months with this organism.[3] Certainly *Staphylococcus aureus* would have killed this patient long before he came to this hospital. I think that the positive culture with *Staphylococcus epidermidis* is not a false-positive finding; it is a very significant finding. I am inclined to believe that this patient had staphylococcal endocarditis involving the tricuspid valve, with a great many small lung abscesses that were too small to be seen, below the level of resolution of the chest film.

The results of the gallium scan of the thorax indicated some increase of lymphocytes and polymorphonuclear leukocytes in the mediastinal nodes; this is not sufficient to form the basis of a diagnosis of AIDS or any other specific infection. I believe this finding is the result of tiny lung abscesses with drainage into the mediastinal nodes.

What about the so-called penicillin allergy? It is mentioned in the history, and then all of a sudden it disappeared. Later in his hospital stay, he is given ampicillin and oxacillin, and nothing happens. The penicillin allergy is probably not real; it is not real in many patients who claim such an allergy. Since the medical treatment for endocarditis, if this is what he had, includes a non-penicillinase-susceptible penicillin and gentamicin, the matter of a penicillin allergy was important and I would certainly in a desperate situation try out a course of desensitization.

I am disinclined to think that penicillin therapy would have been effective in a person who was sick for so many months and who was so terribly ill. If the diagnosis of bacterial endocarditis could be proven more strongly during life, it might have been helpful to excise the tricuspid valve, a procedure which is successful in a reasonable number of patients.[4] In addition, an echocardiogram might have helped. I am not sure how valuable it is for tricuspid disease; it is not as useful with the right side of the heart as with the left, but certainly it would have been worth a try.[5] Not only should an echocardiogram have been done, but this patient also should have undergone a right-sided cardiac catheterization. Looking at the results of the Swan-Ganz study, the pressures on the right side of the heart are very high, including the right atrium, the right ventricle, and the pulmonary artery. I would suspect that he had a tricuspid insufficiency.

When endocarditis is not obvious from a medical history and physical examination, very often the diagnostic investigation for that condition stops; tuberculosis, which I think was not reasonable to suspect in this case, continued to be investigated and its treatment gave the patient an isoniazid-rifampin hepatitis. That treatment was stopped and then started again, because a few acid-fast bacteria were found in a mediastinal node biopsy.

In a patient with a chronic illness which is suppurative and lasts for months, and given the symptoms of a bowel disorder, a diagnosis of amyloidosis should be considered. Little is known about the life history of amyloidosis. I am aware of one patient who had proven amyloidosis that was first suspected serendipitously based on the finding of globulin spike on electrophoresis and later confirmed by an oral biopsy. That patient had no other symptoms for a period of 10 years. Amyloidosis can be a long slow disorder. Why can't it also develop more acutely? I will suggest that the patient has small bowel amyloidosis. A biopsy from the lower bowel during a sigmoidoscopy could have been examined and might have shown signs of amyloidosis. The pathologist would have to be told to look for amyloid; otherwise, the sample will not routinely be stained with Congo red, and the diagnosis can be overlooked. The striking deficiencies of calcium and magnesium reinforce the concept of absorption defects in the small intestine.

It is amazing that the urine had a specific gravity of 1.011 in the setting of a febrile wasting disease. It would seem to me that the patient had difficulty with renal concentration. This could have been due to renal involvement if amyloidosis was present. What about the urinary dribbling? He did not have an enlarged prostate, but judging by his life style, I think he might have had a urethral stricture. I would be inclined to suspect that as a result of a urethral stricture he would have hydronephrosis, not too severe, but at least some would be present.

In summary, this patient had right-sided *Staphylococcus epidermidis* endocarditis involving the tricuspid valve. There are at least 500,000 intravenous drug abusers and not all of them develop bacterial endocarditis, so there must be some other factor involved that makes it possible for some of them to develop endocarditis. I am not sure what it is. In addition, I wonder if he did not have early amyloidosis. I also suspect that he has a urethral stricture with some hydronephrosis and that he has what used to be called focal embolic glomerulonephritis, although not too severe.

CLINICAL DIAGNOSES

Bacterial endocarditis, right-sided, involving the tricuspid valve, due to *Staphylococcus epidermidis*.

Myocardial abscesses, embolic, secondary to bacterial endocarditis.

Lung abscesses, multiple, small, embolic.

Focal embolic glomerulonephritis.

Possible amyloidosis, secondary to chronic infection, with intestinal involvement and resulting malabsorption syndrome; with possible renal involvement, proteinuria, and hyposthenuria.

Urethral stricture with partial lower urinary tract obstruction.

PATHOLOGY PRESENTATION

Belinde Aftalion, MD (Staff Pathologist): At autopsy, the body was that of an emaciated man, with multiple antecubital scars from intravenous drug abuse. There was a left thoracotomy wound from the mediastinal biopsy and a chest drainage tube. The autopsy was limited in the sense that the patient was suspected of having AIDS, so that the organs were inspected and sampled only, except for the heart and brain. All remaining organs were buried. The heart blood and all organs were cultured.

The heart weighed 407 grams, which is larger than usual. There were pericardial adhesions related to the surgery. Right ventricular hypertrophy was present. The right heart chambers presented the typical endocarditis of the drug abuser (Fig 2). At the entrance of the superior vena cava into the right atrium there was a string of bacterial vegetations. The tricuspid valve was studded with vegetations. There were also bacterial vegetations attached to the right endocardial outflow tract. On microscopic study, the tricuspid valve revealed exuberant masses of gram-positive cocci (Fig 3). The pulmonary valve was covered with bacterial vegetations resembling a necklace (Fig 4). Similar vegetations could also be seen within the main pulmonary trunk (Fig 4). On microscopic examination all

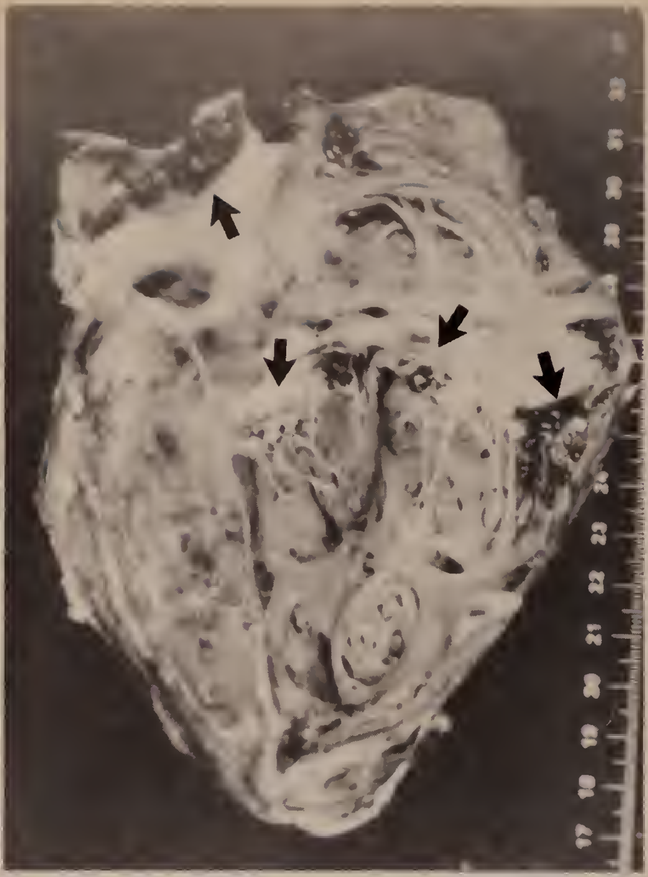


FIGURE 2. Inflow tract of right side of heart, with vegetations at junction of superior vena cava and right auricle (upper arrow), and on tricuspid valve (lower arrows).

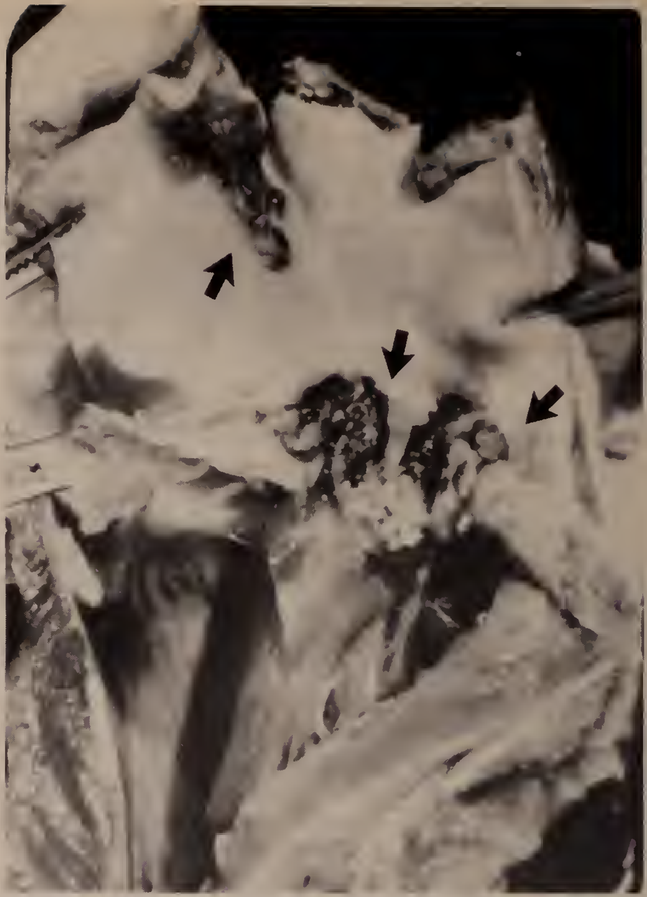


FIGURE 4. Outflow tract of right ventricle, with vegetations on pulmonary valve (lower arrows), and in main pulmonary artery (upper arrow).

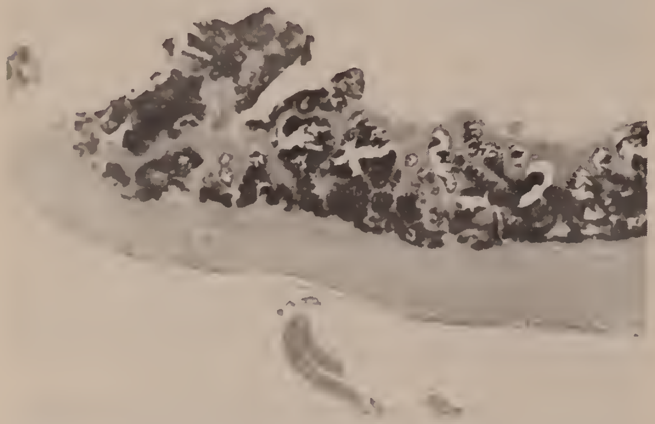


FIGURE 3. Bacterial vegetations on tricuspid valve. (hematoxylin and eosin stain; original magnification × 10)

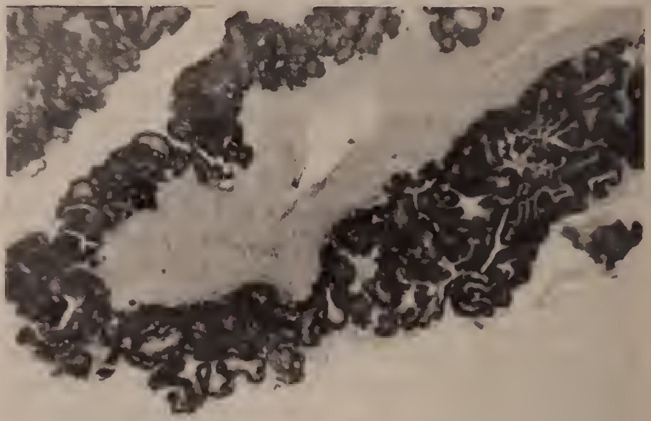


FIGURE 5. Pulmonary valve vegetations containing gram-positive cocci. (Gram stain; original magnification × 25)

vegetations contained gram-positive cocci (Fig 5). Postmortem cultures of the tricuspid valve, all organs, and the heart blood yielded *Staphylococcus aureus* organisms.

It is postulated that narcotic addicts induce right-sided endocarditis on endocardium damaged by the filler material in oral drug preparations which are injected intravenously. The filler material is said to act in the form of small foreign bodies bombarding the intact endocardial surface, and causing roughening of the endocardial lining with subsequent platelet and fibrin deposition; finally, bacterial growth produces the larger vegetations. The left side of the heart had unremarkable mitral and aortic valves. There was mild endocardial fibrosis of the left ventricle. The coronary vessels were all patent, and only the left anterior descending branch of the left coronary artery showed slight stenosis due to atherosclerotic changes.

A study of 351 cases of endocarditis in drug abusers indicates that the tricuspid valve is the most frequently involved (52%).[6] Combined involvement of both the tricuspid and pulmonic valves was rare. The organism most commonly isolated was *Staphylococcus aureus* (62%); other organisms cultured included all varieties of streptococcus (21%), gram-negative organisms, and *Candida*. Among the drug abusers who had previous cardiac valve damage, the most common organism is *Streptococcus viridans*. The myocardium in this case appeared grossly unremarkable, but on microscopic study revealed multiple abscesses resulting from septic emboli. These were predominantly present in subendocardial locations and could be seen in the columnae carneae of both ventricles (Fig 6).

The lungs were bulky and stiff, showing a diffuse "honeycomb" appearance. There was a right-sided pleural effusion. The left lung revealed fibrous pleural adhesions secondary to the surgery. Microscopic examination of the lungs showed areas of exudate, fibrosis, and septic emboli. Septic vascular emboli produced occasional mycotic aneurysms. The pulmonary vasculature also contained foreign-body granulomas and the foreign material could be seen in the lumina of smaller pulmonary blood vessels. It was identified as talc (magnesium sulfate) by virtue of its intense birefringence under polarized light (Fig 7).

Prominent features of the autopsy findings were pulmonary fibrosis and clubbing of the fingers. It is well known that heroin addicts may develop acute pulmonary edema and diffuse alveolar damage. A cocaine user has also been reported to develop fatal acute pulmonary edema with alveolar damage.[7] I would like to postulate that the pulmonary fibrosis in this case was a late consequence of drug-induced diffuse alveolar damage (Fig 8). The pulmonary edema present at autopsy was due to congestive heart failure.

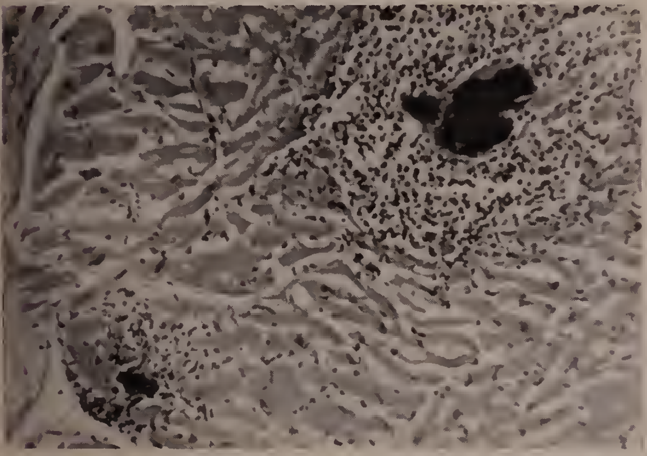


FIGURE 6. Myocardial abscesses in wall of left ventricle. (hematoxylin and eosin stain; original magnification × 50)

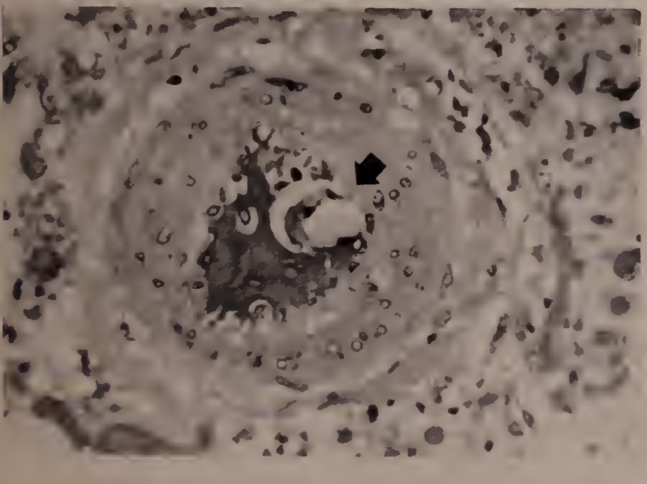


FIGURE 7. Birefringent intravascular foreign body material viewed under polarized light. (hematoxylin and eosin stain; original magnification × 100)

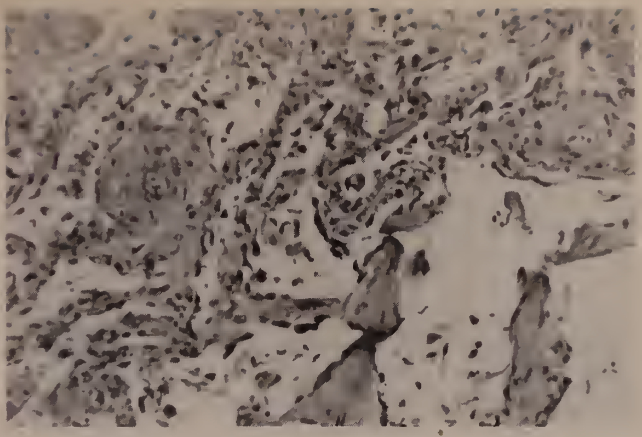


FIGURE 8. Interstitial fibrosis of right lung. (hematoxylin and eosin stain; original magnification × 50)

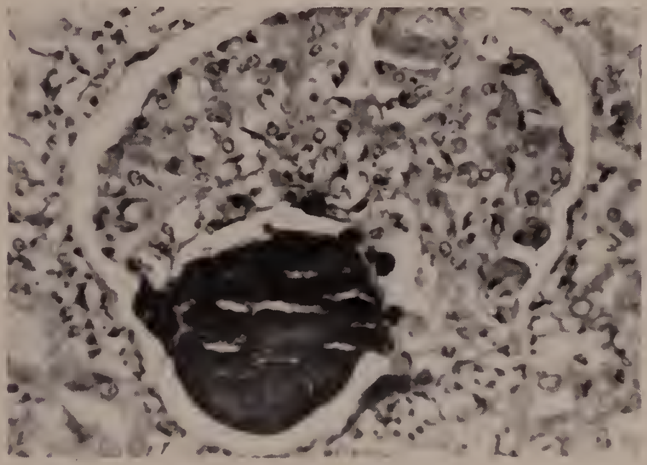


FIGURE 9. Bacterial embolus in renal glomerulus. (hematoxylin and eosin stain; original magnification × 100)

The hilar lymph nodes removed at autopsy were identical to the biopsied material. They contained an abundance of lymphocytes and plasma cells without granulomas. An acid-fast stain showed multiple acid-fast organisms; however, cultures for acid-fast organisms were overgrown by other organisms. The axillary and inguinal lymph nodes were small, lymphocyte-depleted, and infiltrated with plasma cells. They were negative for acid-fast organisms. Such lymphocyte-depleted lymph nodes can be seen in any patient with sepsis.

Septic emboli with cocci were found in the kidneys, as Dr Shapiro predicted, with abscess formation (Fig 9). Septic emboli with abscess formation were also found in all viscera, including the pituitary gland and brain. The cerebral vessels also contained birefringent foreign material.[8] The liver was firm and showed bile duct proliferation and periportal fibrosis, probably secondary to alcohol abuse, on histologic examination. The gastric mucosa was studded with petechiae on the villous crests. The mucosal villi of the small bowel were not atrophic. Patients with severe sepsis may develop diarrhea, the mechanism of which has not been established. It is suggested by Ogbuawa[9] that staphylococcal toxemia may induce diarrhea.

The bone marrow contained many cells, with erythroid hyperplasia and a shift to the left of the myeloid cells.

Henry D. Isenberg, PhD (Chief, Microbiology): You asked about the stool culture. About 10 days before death, a stool specimen was submitted for culture; we isolated *Staphylococcus aureus* and brought that fact to the clinicians' attention, especially since it was a methicillin-resistant organism. The organisms which we isolated from all the organs of the patient, but especially from the valve vegetations, at autopsy were methicillin-resistant *Staphylococcus aureus.* The antimicrobial suspectibility patterns were the same; the staphylophage pattern for all isolates was 3A/3C/84/71/6/85/83A/59/53/42E/77/47/81/-75. The finding of methicillin-resistant *Staphylococcus aureus* was the one microbiologic result of significance that might have influenced the treatment.

Dr Shapiro: What would you do in a future situation like this, where the blood cultures remain negative?

Dr Isenberg: The present methodology for blood cultures, the DuPont Isolater technique, removes all of the host complexes and antimicrobial agents that might interfere with bacterial growth in the cultures. All host cells are lysed before a blood culture concentrate is cultured in various agar media. Therefore, neither complement nor antigen-antibody complexes should interfere with the growth of organisms in these blood cultures. Why the staphylococcus was not recovered cannot be explained. It may be that we received the blood cultures only early in the patient's hospital course and never received them later when they might have been positive. I don't understand why *Staphylococcus aureus*, which usually grows so profusely, should not have grown out by the methods which we use.

Edward J. Hotchkiss, MD (Attending Physician): I want to point out that had an autopsy permission not been obtained on this patient, we would have all missed an important chance to learn a great deal. The autopsy examination has been a tried and true friend of the clinician, and this case is an excellent example of what benefit we can all derive from it.

Harry Steinberg, MD (Chief, Pulmonary Medicine): I would like to comment about the finding of the acid-fast bacilli in this patient. We would all be missing the flavor of this case if we did not keep in mind that this patient was febrile, had had almost 20 negative blood cultures, and had a markedly enlarged left hilar node which was positive on gallium scan and which at first microbiologic examination was loaded with acid-fast organisms. I think that the clinicians felt at that time that they were facing a systemic atypical mycobacterial infection in a patient who had an opportunity to undergo localized proliferation —during a terminal illness when the patient was in an immunocompromised state and unable to mount a granulomatous tissue reaction.

Leonard B. Kahn, MBBCh (Chairman, Department of Laboratories): I was involved in the evaluation of the frozen section of the mediastinal lymph node, which by the way was not actually enlarged. What was interesting about that node was its total lack of reaction to acid-fast bacteria; it contained only a great many plasma cells. Also of interest was that the acid-fast stain demonstrated literally hundreds, perhaps thousands, of organisms in one segment of the node. In other parts of the node, these organisms were not seen. I favor the concept that these bacilli may have been locked away until such time as they had an opportunity to undergo localized proliferation —during a terminal illness when the patient was in an immunocompromised state and unable to mount a granulomatous tissue reaction.

Dr Shapiro: Do we have the facilities to determine the helper-to-suppressor lymphocyte ratios?

Herbert Diamond, MD (Chairman, Department of Medicine): Yes, we have the facilities, but one of the difficulties in such an acutely ill patient is that the results of such a test may not be very meaningful.

Rabia Mir, MD (Attending Pathologist): The cardiac lesion in this patient is rather unusual for an endocarditis due to *Staphylococcus aureus.* As you saw, there were multiple vegetations, but the underlying valve was not destroyed. Such an endocarditis would be more appropriate for a *Staphylococcus epidermidis* infection. I would like to propose that the initial infection started with *Staphylococcus epidermidis* and the *Staphylococcus aureus* infection developed later.

Dr Isenberg: I assure you that staphylococci are not only illiterate but they also don't listen. Just because there are numerous papers in the medical literature which describe how *Staphylococcus aureus* ought to behave in lesions, that does not necessarily mean that the staphylococcus will behave that way. Secondly we have to keep in mind that this is not the usual *Staphylococcus aureus;* reports in the literature of basic microbiology contain repeated statements that organisms that are resistant to antimicrobial agents do not behave exactly like the wild-type organism. The fact that this organism carried around with it several resistance determinants not only to a number of the usual antimicrobials but also to the beta-lactamase-resistant beta-lactams, may indicate that it may not display the traditional staphylococcal potential as a pathogen. That to me is a more logical explanation.

I should mention that we saw a large number of acid-fast organisms on a blood smear. We were questioned by our colleagues in the pathology department as to whether the water supply might be contaminated by acid-fast organisms; but subsequent tissue studies of this patient demonstrated numerous acid-fast organisms in the areas studied. As to what kind of acid-fast bacterium this is, I am sorry that I cannot tell you as yet. Certainly it is not one of the faster-growing mycobacteria. It may be an avium-intracellular type, but I cannot tell. From the microscopic morphology of the organisms, I am highly suspicious that this is going to turn out to be *Mycobacterium tuberculosis*.

FINAL DIAGNOSES

Severe infective *Staphylococcus aureus* endocarditis in narcotic (cocaine) addict, involving the tricuspid and pulmonary valves, superior vena cava, and main pulmonary artery trunk.

Bacterial myocarditis; visceral and cerebral bacterial emboli with abscess formation.

Pulmonary fibrosis secondary to remote drug-induced diffuse alveolar damage; clubbing of fingers.

Heart failure; pleural effusion and ascites; fibrosis of liver (acohol abuse).

REFERENCES

1. Mills J, Drew D: Serratia marcescens endocarditis: a regional illness associated with intravenous drug abuse. *Ann Intern Med* 1976; 84:29–35.
2. Williams WJ, Beutler E, Erslev AJ, Lichtman MA (eds): *Hematology*. New York, McGraw-Hill, 1983, p 156.
3. Weinstein L: Infective endocarditis, in Braunwald E (ed): *Heart Disease*. Philadelphia, W B Saunders, 1980, pp 1198–1201.
4. Arneborn P, Björk VO, Rodriguez L, Svandbom M: Two-stage replacement of tricuspid valve in active endocarditis. *Br Heart J* 1977; 39:1276-1278.
5. Andy JJ, Sheikh MU, Ali N, et al: Echocardiographic observation in opiate addicts with active infective endocarditis. Frequency of involvement of the various valves and comparison of echocardiographic features of right- and left-sided cardiac valve endocarditis. *Am J Cardiol* 1977; 40:17–23.
6. Reisberg BE: Infective endocarditis in the narcotic addict. *Prog Cardiovasc Dis* 1979; 22:193–204.
7. Allred RJ, Ewer ST: Fatal pulmonary edema following intravenous 'freebase' cocaine use. *Ann Emerg Med* 1981; 10:441–442.
8. Openshaw H: Neurological complications of endocarditis in persons taking drugs intravenously. *West J Med* 1976; 24:276–281.
9. Ogbuawa O, Fam WM: Comparison of staphylococcal and nonstaphylococcal endocarditis in narcotic addicts. *South Med J* 1979; 72:1557–1558.

FROM THE LIBRARY

SUBACUTE STREPTOCOCCUS ENDOCARDITIS

In discussing the symptoms of the disease you will note that, apart from those due to intercurrent conditions, they are produced in the following ways: We have first the symptoms due to the original valve defect, and of that disease as influenced by the fever of the disease, the anemia, the exhaustion, and the complications. These I call cardiac or, better, myocardial symptoms. Then there are the symptoms due to the infection. These are of two kinds: first, those due to the toxemia, and, second, those due to the vegetations in the valve. Due to the toxemia we have the fever with all its manifestations, including usually a marked anemia. Due to the change in the valves we have what I have termed the endocarditic symptoms. These include all manifestations due to breaking off of larger or smaller vegetations on the valves. Such symptoms are petechiae, tender cutaneous nodules, embolisms, embolic aneurysms, and purpura. Purpura may, however, also be produced, I believe, by the toxemia and anemia. The subject of purpura in cardiac disease of all kinds needs elucidation.

The onset of the disease, as I have said before, is usually insidious. The patient may be up and around for a long time. We usually reckon the disease as lasting from four to eighteen months, but longer and much shorter cases have been observed by other authors and by myself. The earliest symptoms are of the greatest variety. In fact, we could spend all the time at our disposal today in going over the many modes of onset of the clinical picture. I may later do this in a brief fashion. The most common symptoms at the onset are lassitude, vague pains, loss of appetite, feverishness, chills or chilliness, vertigo, headache, cough, and, less often, cardiac symptoms. The constant symptom in all the cases is fever. Occasionally the disease is acute in onset, the first symptoms being a high temperature, accompanied or not by a chill, or an embolism of one of the organs or of one of the peripheral vessels. It is very probable that in all such cases there has been a fairly long preceding period during which the patient was suffering from the infection, and either did not feel ill or did not feel ill enough to consult a physician. I remember seeing a woman who, ten days before being seen by me, walked almost ten miles in the Catskill Mountains in company with her physician. She took sick with vague abdominal symptoms, diarrhea, and fever, one or two days later, and was brought to New York on the day before that on which I saw her. She was suspected of having an atypical typhoid infection. Because of the presence of an exquisitely tender sternum without evident cause I examined her heart with great care, and finally satisfied myself that the mitral orifice was stenosed. After a prolonged search I found one petechia. A blood-culture was ordered, and the next day an anhemolytic streptococcus was found. On the same day the patient died, with symptoms of pulmonary infarction.

The possibility of patients going around for a long time and attending to their duties and not realizing that they are sick is a very important one in connection with a part of this subject that will occupy us much in future conferences—namely, the bacteria-free stage of the disease. It explains how it is possible for us to encounter patients who have become spontaneously free of the infection and come to us only later with the after-results of the infection.

E. LIBMAN, M.D.
(*Med Clin NA* 1918; 2:117–151.)

HISTORY

Medical education in New York City in 1866–1867

A student's notebook of Professor Charles A. Budd's lectures on obstetrics at New York University—Part II

BERT HANSEN, PhD

In the two lectures by Professor Budd, one can appreciate his teaching style and his commitment to medicine. This article examines a series of 54 shorter samples of his teaching on a much wider range of topics. The following passages are presented in the order they occur within the nine weeks of the course. The descriptive headings, bracketed commentaries, and footnotes were added. In a few cases, passages have been taken out of order so that comments on one topic could be grouped together.

1. Frequency of spontaneous abortion. By the term abortion we signify the expulsion of the foetus from the womb before it has attained a period of development sufficient to maintain its own existence, which is generally prior to the sixth month of utero-gestation . . . . Abortions are much more frequent than is generally credited. Statistics [from Europe] have shown that only one in eight of married women pass through life without aborting at one time or another. It is certainly not less in this country. Abortion is also more apt to occur in those pregnant for the first time; and males are the subject of it more than females. (pp 3–4)

2. Women's fashion. Tight-lacing, I am satisfied, is another cause of abortion. Our American women are too much inclined to emulate the wasp, forcing all the viscera down, thereby keeping the uterus from ascending as it should. This gives rise to conjestions [sic] and premature excitement of the uterus.* (p 8)

3. Requests for abortion. Then we have those causes [of abortion] dependent on the male, which are termed accidental; but I am sorry to say, from their great frequency at present, they belie the name accidental. Certain medicines as ergot, tansy, rue, savin, aloes, etc. are taken with a view of abortion. But if all the parts are healthy and the mother has no constitutional disease, I am convinced that in the early months of pregnancy at least they have not the slightest effect whatever unless administered in doses that would produce effects on anyone.

Ladies are continually calling at my office until I am sick at heart, married ones, and, with the greatest nonchalance imaginable, ask me to do something to relieve them. But gentlemen, the laws of God and man put it as akin to murder. When you put the question to them in its proper aspect, "What do you want this done for?", some will tell you they are unmarried and wish to avoid the disgrace. Others beat around the bush in that way that only those can do who would murder their offspring. I tell them and plainly, too, that those who dance must pay the fiddler.† (pp 12–13)

4. Use of electricity for abortion. When I was engaged making autopsies at the Coroner's office of those who died from abortion, I frequently found metritis and metro-peritonitis, which were undoubtedly caused by undue violence. I have known an electrical current to be passed through the uterus, several times in one person, to destroy the vitality of the ovum and cause abortion, which has resulted in fatal metro-peritonitis. (pp 16–17)

5. Acute infectious diseases in pregnancy. I desire to call your attention to the prognosis when the case [of haemorrhage] is dependent on some of the acute diseases, es-

---

Address correspondence to Professor Hansen, Department of History, New York University, 19 University Place, New York, NY 10003.

Grateful acknowledgment is made to the Institute for Advanced Study, Princeton, New Jersey, for financial support of this research by a fellowship for 1984–85.

This is the second installment of a two-part article which analyzes extracts from a rare set of student lecture notes. The 366-page manuscript reveals aspects of teaching and medical practice not generally witnessed in such documents as published textbooks, journal articles, or celebratory speeches. Because the notebook contains a complete verbatim account of 26 lectures, one is able to enter a medical school classroom of more than a century ago and eavesdrop on an esteemed practitioner's advice to students. Part I of this study in the August issue of the *Journal* sketched the historical setting for Professor Budd's teaching and presented the complete text of two of his lectures.

* For recent analyses of doctors' opposition to corsets and other constricting fashions, see Schwarz[1] and Haller and Haller.[2]

† By this hostile equation of abortion with murder, Budd was taking a stand that was not yet standard for his profession, though it would come to be so over the next several decades.[3] Mohr has shown that "by the middle decades of the nineteenth century . . . abortion had shifted from being a marginal practice of the desperate few to being a quantitatively significant factor in the effort of American women to regulate their own fertility."[4] His study reveals further that "the nation's regular physicians, those who favored formal education and scientific research, mounted a major campaign to stamp out the epidemic of abortion . . . [and that] the abortion issue combined for many nineteenth-century physicians both their ideological world view and their professional self-interest."[5]

Budd's last sentence is interesting in another regard, for the sarcastic phrasing implies that these women, in choosing to "dance," were seeking sexual pleasure for themselves. This runs somewhat counter to the widespread interpretation (held both by Victorians and by some historians) that women of this era were generally "passionless" and took little pleasure in sex. Among the many recent historical analyses of 19th-century women's sexuality, see Cott,[6] Degler,[7] Haller and Haller,[8] Shade,[9] Smith-Rosenberg,[10,11] and Smith-Rosenberg and Rosenberg.[12]

pecially small-pox, scarlatina, and measles. A woman five or six months advanced in pregnancy contracts one of these diseases, labour sets in, and she aborts. In this case the prognosis is exceedingly grave. It is the disease, however, that kills the patients, the abortion only being a complication. The abortion is an indication of the severe form of the disease. I have seen women recover who have aborted in typhus, but never in measles, scarlatina, or small-pox. (pp 19–20)

6. Cautery to prevent miscarriage. A woman comes to you and says she has miscarried several times previously; you examine and find the neck ulcerated. Bennet cauterizes it with the same impunity as in the unimpregnated condition and says no evil results follow.[‡] Now I beg to differ with him. I have seen a miscarriage come on after one cauterization of the neck; but I have also seen it successful and prevent the abortion. I would advise you to treat the ulceration always, whether pregnant or not. [See also Excerpt 37 on cautery.] (pp 20–21)

7. Syphilis and mercury. Syphylis [sic] can only be treated constitutionally. I know instances where abortion has occurred four or fives times consecutively dependent on syphylis, and after treatment carry the product of conception to the full period. Do not avoid giving mercurials because of pregnancy. (p 22)

8. Remedies for severe haemorrhage during pregnancy. First place your patient in the recumbent position, on a hair mattress, and insist on mental as well as physical rest; allow none but the nurse to see your patient. Administer opium per rectum; suppositories will do very well. Keep her head low and let her have cooling drinks, lemonade or cold tea. Be sure she has no hot drinks or food. If the haemorrhage continues, you will have recourse to astringents acting through the blood. The acetate of lead answers best. Give it in the form of [a] pill—two grains combined with one of opium every two or three hours. If it still persists, abortion will come on in spite of all you can do; and then it should be accomplished as rapidly as possible. Some women will dribble away for weeks and not abort. But when this affects the constitution causing anemia and exhaustion, we should induce the expulsion of the foetus. Your textbooks will recommend turpentine, ergot, etc., but if all these fail and you even find part of the ovum has come away, your only recourse is the tampoon [sic]. It is nothing in reality but a plug which causes blood to coagulate and so stop the haemorrhage. We have the alum plug which is retained by a sponge or handkerchief. (pp 24–25)

9. Carbolic acid as a disinfectant. [Regarding the use of tampons and tents to stop uterine haemorrhage.] Recently I have tried a new method, which is far superior to anything before. I generally select a piece of good sponge, boil it in slightly acidulated water, and then immerse it in mucilage with a few drops of carbolic acid added to it. I generally order four or five drops to the ounce of mucilage.This is a very powerful disinfectant. I have allowed the tent to remain in 48 hours which was prepared in this way, and then on its removal detect but little foetor.[§] (pp 33–34)

10. Predicting the date of delivery. Women generally calculate labor to come on in 40 weeks or 280 days after conception, or four and a half months after the first sensation of life within. Naegela[‖] gives a very good rule; he says calculate three months back from the date of the last menstruation and then add 7 days. This is one I generally adopt and teach. Meigs only calculates three months back from the date of the last menstruation. You will prove yourselves which is the most correct.[¶] (p 35)

11. Explaining the commencement of labor. But it just all amounts to this, that labor comes on (as one of the ancients has said) on the 280th day by the Grace of God.[#] (p 39)

12. Protecting your reputation. The first stage [of labour] varies greatly in duration. Sometimes it occupies but a few minutes and in others many, many hours. Most of the text books lay down six to eight hours as a rule, which is positively absurd. Supposing you are sent for to a case of labour and find the os but little dilated and the pain seemingly not increasing in force and you go away with the intention of returning in an hour or two. But while you are away, the child is born and perhaps some injury has been sustained in consequence. This subjects you to severe criticism, and happening to a young practitioner would be almost fatal to him. I may say, nothing is so variable in the practice of midwifery as the duration of the first stage of labour. (pp 43–44)

13. Suffering and sympathy. You are to give your patient all the encouragement and comfort in your power. Inform her that every thing is for a wise object. Man little knows how woman suffers under these circumstances, and she is indeed entitled to our most profound sympathy. (p 46)

14. Clinical experience. As labour progresses and the cervix dilates, you pass your finger in and meet a rounded smooth surface and then proceed to diagnose the position. When you are studying these things, with a pelvis and foetal head before you, it may appear to you as an easy matter to recognise the position during labour; but take my word for it, this requires a good deal of experience. In your early practice do not be discouraged when you find yourself mistaken. Make the diagnosis carefully, never jumping at any conclusion too soon, for it is not an easy matter, teachers and text books to the contrary.

I have seen eminent practitioners positively assert that so and so was the position of the head and finally find themselves much mistaken. (p 70)

15. Post-partum diet. We were studying yesterday the duties of the accoucheur to the mother and child. You are also at your first visit to enquire if she has had any sleep, if the bandage is all right, also about the lochial discharge. There is a popular opinion that the mother needs some stimulants after labour, and you will find that without your advice she is dosed with wines and cordials. Now as a rule nothing can be more injurious than this practice. No stimuli or animal food are at all indicated. Farinaceous diet is preferable: rice, toast, crackers, tea, etc. If, after your orders, you find that the nurse has been administering stimulants, call her to account.

[‡] James Henry Bennet[13] of London, not "Bennett" as in Garrison[14] and Jameson.[15]

[§] Note that the "disinfectant" is used only to prevent odor, not to avoid infection. The writings of Oliver Wendell Holmes[16] on the contagiousness of puerperal fever (1843, reprinted 1855), of Ignaz Semmelweiss[17] on the etiology of childbed fever (1861), and of Joseph Lister[18] on antisepsis (1867 et seq) had relatively little impact on American medical practice prior to the late 1870s.[19] The introduction of aseptic and antiseptic technique in obstetric practice in New York City hospitals and the resistance it met from the late 1870s through the mid-1880s have been briefly outlined by Heaton[20] and by Ricci.[21] Carbolic acid was Lister's first disinfectant. See also Excerpt 49.

[‖] An error for Franz Karl Naegele, 1778–1851.[22]

[¶] Charles D. Meigs, 1792–1869, was Professor of Obstetrics at the Jefferson Medical College in Philadelphia.[23,24]

[#] Budd may well owe this idea to Dewees' statement that, "Avicenna, centuries ago, declared that labour was a law of God, and that it came on at the appointed time. I would ask, has any hypothesis since that period, enlightened us more upon this subject, than the humble confession of this good old man?"[25]

Generally, I direct the patient to be kept on a light diet till the fourth day. I then allow her some oysters, chicken broth or beef tea, and after the fifth or seventh day let her take some solid food. (pp 121–122)

16. Endorsed products for after-pains. With regard to the after-pains I have a few words to say. Frequently women dread these more than labour itself, and occasionally they cause a good deal of mischief. In this way the sleep which it is requisite she should have is disturbed.

The mouths of the utero-placental vessels, being open, pass blood into the uterus, which coagulates and as a foreign substance excites contraction. As a rule they do not suffer much from these after-pains in first labour. They are more felt in second labour than in first, and in the third than the second, and so on.

Now it is desirable, you see, to bring her under the influence of some anodyne. Opium is by far the most popular, in the shape of Dover's Powder, McMunn's Elixer, Paregoric, etc.[**]

The only trouble from its administration is the liability it has to act as an excitant and induce nausea and vomiting. Some cannot take opium in any form without having temporary delirium. Dewees recommends camphor and I know of nothing which is so efficient. His camphor julep (Rx Pulv camph 1 dram; Pulv Cassia, Pulv sacch alb ana 2 oz; Aqua 6 oz) is the best form to take the drug [camphor, cassia, white sugar, and water]. A tablespoonful is given every two or three hours. I am in the habit of adding an ounce of tincture of hyosciamus [sic] and discarding an ounce of water.[††] (pp 122–124)

17. Nurses don't know how to feed the baby. Some nurses make a mixture or paste which they call pap. I think it is bread, sugar, water and perhaps a little gin. She will put several teaspoonfuls of this into the child's stomach. This will make the child restless and worrisome and the nurse thinks she has not given it enough. Of course the child continues to be fretful and the nurse will then take it into her head that there is wind in the child's stomach and will give it some sort of vegetable tea, with perhaps a little more gin in it, which all results in the child's vomiting. Then comes more pap. All this is to be strongly reprehended.

Another habit which is recommended by some physicians is to give the child some molasses and water, with the idea of purging away the meconium, but this is simply disgusting. The meconium is purged away quite naturally. The rule you should bear in mind is to put the child to the breast as soon as labour is completely over and there it gets what is necessary viz. the colostrum or first milk that comes away; this purges the meconium in the proper manner. If the child requires some sustenance from the mother not being able to give it milk, approximate nature as closely as possible. Fresh cow's milk with a little sugar and water will answer well. The nurses frequently, when heating the milk, have a habit of putting the spoon into their own mouth to see when it is warm enough and in this way some of the nurse's saliva finds its way into the child's stomach. This is altogether unnecessary. (pp 128–130)

18. Excess lactation. A few words with reference to the management of the breasts in an ordinary case. About the third day the mother will tell you she has more milk than she requires; now to prevent the occurrence of mammary abscesses, the breasts should be drawn off with the husband's mouth or by the aid of one of the many breast pumps used. A newborn puppy, blind and with its feet tied, will draw off the milk as well as anything else. [Figs 1, 2] (pp 133–134)

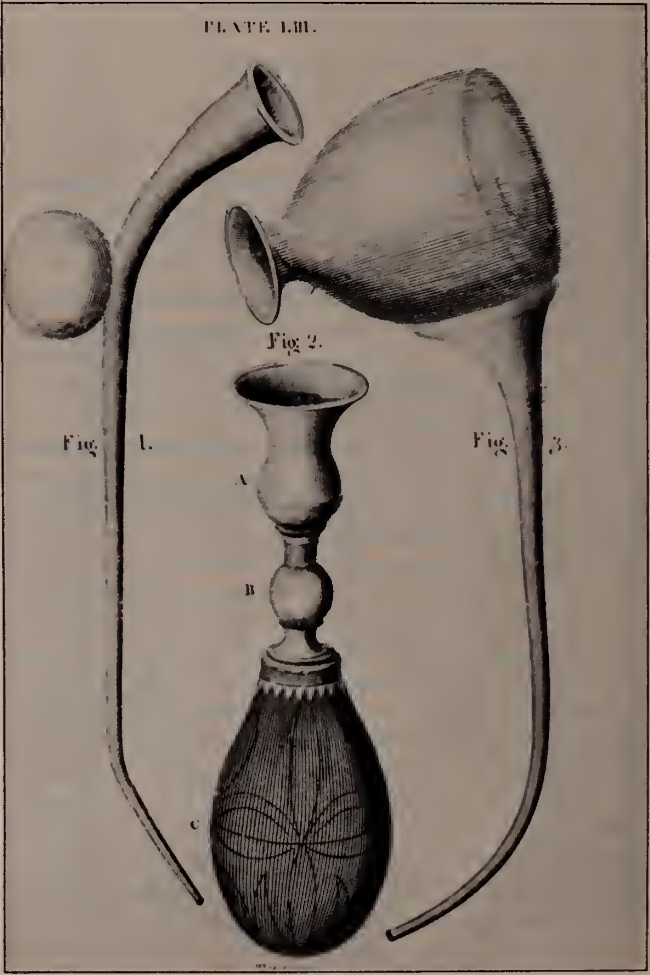
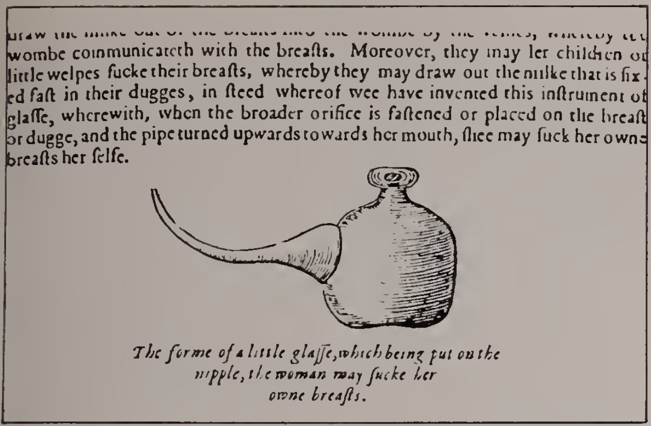


PLATE LIII.
Fig. 2.
Fig. 1.
Fig. 3.
A
B
C

FIGURE 1. A breast pump and two glasses for drawing milk, illustrated in Bell's *System of Surgery*, a late 18th-century work. Budd is probably referring to the device of the type shown in the middle, which has a glass cup (A) joined by a brass tube (B) to "a bag of elastic gum" (C). The glass on the right is of a very old style; see Fig 2. From Plate 53 of Vol. 5 in Benjamin Bell, *A System of Surgery*, 7th ed, Edinburgh, 1801, 7 vols.

[**] According to Haller and Haller, Dover's Powder is ipecac and opium and McMunn's Elixir is denarcotized laudanum prepared in ether.[26]

[††] Dewees (1832)[27] has a somewhat different prescription: 2 drams of gum camphor, alcohol, 3 drams of gum arabic, and sugar mixed with 6 oz of water. Perhaps Budd was using a different edition.



wombe communicateth with the breaſts. Moreover, they may let children or little welpes ſucke their breaſts, whereby they may draw out the milke that is fixed faſt in their dugges, in ſteed whereof wee have invented this inſtrument of glaſſe, wherewith, when the broader orifice is faſtened or placed on the breaſt or dugge, and the pipe turned upwards towards her mouth, ſhee may ſuck her owne breaſts her ſelfe.

The forme of a little glaſſe, which being put on the nipple, the woman may ſucke her owne breaſts.

FIGURE 2. The great Renaissance surgeon Ambroise Paré's breast glass from his book on generation first printed in Latin in the 16th century. This woodcut appears on p 919 in the English translation of his collected works printed in London in 1634. Observe its striking similarity to Bell's illustration of two centuries later (in Fig 1). Note, too, that Paré, like Budd, suggests one can also use a puppy to draw off excess milk.

19. Bloodletting of the infant.[28,29] The next cause [of suspended foetal animation] is cerebral conjestion [sic]. The most prolific source of this is in face presentations: the encircling of the cervical vessels, by the cord being tightly bound around the neck. Retarded delivery of the shoulder will often cause this. The condition of the face under these circumstances is of a purplish gorged appearance; the lips are everted and swollen and every evidence of hyperoemia of the head. The first thing you are to do in this case is to bleed the child, not from the arms, but cut the cord without tying. I have allowed 3–4 ounces to be taken away, but you will generally find 4 or 5 teaspoonfuls enough. (pp 148–149)

20. Cases of recovery of suspended foetal animation in Cincinnati and Yonkers. Now we see that no matter what the suspended animation be due to, the main point is to give it fresh air or in reality oxygen. I want you to bear in mind that under no circumstance ought you to avoid resuscitating a child if it is within an hour of the birth. I have seen animation return, where more than an hour had elapsed after the birth. A case is reported in France of a woman who buried her child, and 45 minutes afterwards it was disinterred and survived. Another case occurred in Cincinatti [sic], where forty five minutes had elapsed and after thirty minutes labour and patience, it was resuscitated. Dr F. Jenkins of Yonkers reports a case where the child recovered after being two hours born and where symptoms of suspended foetal animation were noticed long before.[‡‡] (pp 149–150)

21. Warning the family in cases of face presentation. The labour is always in a face presentation longer, more difficult, and more severe both to the mother and child than vertex presentation. Up to the time of Mad. Lachapelle it was deemed an impossibility to bring a child alive into the world where the face presented. If the case is let alone and you watch patiently, you may succeed without any injury either to the mother or child.[§§]

Always take the nurse or husband aside and say that the face is presenting; that experience shows how difficult labour is under this circumstance; and that the child will be almost sure to be disfigured, for indeed after a child is delivered when the face presented, its features are bloated and it has more the appearance of a young prize fighter. Also, be sure and tell them that in a day or two this will all disappear. In such a case, you may use a lotion of brandy and water. [It has been reported that in 19th-century England, the new babies of wealthy parents were commonly washed in brandy.[36]] (pp 163–164)

22. Pelvic presentations—what to do and what *not* to do. Prognosis. As a rule, presentations of this extremity are as safe to the mother as those of the cephalic. I know they are less severe than those of the face. If it is a first child the labour will be more difficult than a vertex presentation, but if not, it is more easier and quicker.

Not so with respect to the child. Pelvic presentations are more severe on the child than any other. Madame Lachapelle [identified in Excerpt 21] had, out of 804 cases, 115 still births and a hundred and twelve born in a feeble condition, making the mortality rather more than one in seven. Another report makes it far less: 668 only out of 26,698 being fatal or about one in thirty. When the breech offers first about 1 in 8½ are still born. In footling presentations about 1 in 6½, and when the knees, about 1 in 4½.[‖‖]

The causes of this are numerous, the most prolific being asphyxia from pressure on the cord. Another is where the head is retained in the vagina and the uterus being in a state of vacuity crumples the placenta as it contracts, separating it. There are other causes, but it is generally from one of these.

Suffice it to say, the mortality is frightful. These are the statistics, but I think in private practice it will not be found to be near so great. I have found by consultation with other eminent accoucheurs that one in eleven or twelve is quite large enough for private practice in this country. In Europe they don't receive the necessary care in the hospitals where these statistics come from.

With reference to the management—and I wish you to bear in mind that the great majority of ordinary labour cases will do better without any assistance—but in pelvic presentations this is not so, for they require the most careful management.

The first thing you should do is to send for your forceps. No man can say he will deliver the head, after the body is expelled, by manipulation alone; and if this fails, every second of time is of the utmost importance to the child and perhaps before your forceps can be got the child is dead. Always send for them as soon as you recognise or even suspect a pelvic presentation.[¶¶]

[‡‡] I would guess that Budd probably heard this report by J. Foster Jenkins, 1826–1882[30] at a medical society meeting, as I could not find it in the only possibly relevant publication[31] on the list in Jenkins' obituary,[32] which ought to be complete since it was written by "a long time intimate friend of Jenkins."[33]

[§§] Madame Louise Dugès Lachapelle, 1769–1821, became resident directress of the Maternité in Paris in 1795 and was its first instructor in midwifery. After her death, her notes were published in three volumes.[34] Garrison says Lachapelle's book, "with its statistical deductions from 40,000 labor cases, had a good deal to do with the establishment of a proper norm or canon of obstetrical procedure at the time."[35]

[‖‖] These statistics, unlike the figures in Excerpts 43 and 53, do not derive from Churchill.[37]

[¶¶] Note that near the beginning of Lecture VII, Budd admonished his students not to bring the forceps along with them initially, as they would then be more tempted to use them unnecessarily.

Do not tell the nurse or anybody else the state of affairs, but say that it is a cross birth or "coming double" and that one in ten or eleven cases the child is still born. By doing this you relieve yourself of a good deal of blame that otherwise might be attached to you; and if it lives you get all the more praise. (pp 172–176)

Some men, the minute they recognise a pelvic presentation, will go to work and pull down a foot or leg as much as they can. In this way the arms become caught along side of the head and the force that can be then employed will not deliver. Some even have used force enough to separate the head from the body.##

If the woman is greatly exhausted or the haemorrhage severe and it is evident she should be delivered immediately, you are obliged to interfere and expedite delivery; but otherwise let it alone. It is a tempting position for a young accoucheur, after having been up a night or two previous to know that by a little manipulation he can accomplish delivery, but do nothing but support the perineum and allow the limbs to drop out spontaneously. After the limbs or breech is out, feel for the cord, pass up your finger and bring down a loop, then watch it carefully; so long as it pulsates let everything alone; if it is in a tight place, push it into a part where there is more room. Much more is to be said with respect to the management of pelvic presentations, which I shall resume again. (pp 176–178)

23. Attitudes toward sexuality and race. We find in this country twin birth to occur about once in sixty cases. A colored woman gave birth to two children. . . . One was black and one was a mulatto; and it was ascertained that after her husband had left her, she allowed the caresses of a white man. Another is told by Mosely [presently unidentified], where a white woman gave birth to a white and [a] black child; and it was found that a negro had entered her rooms and, threatening her life unless she consented to his wishes, he had connection with her immediately after her husband had left her room. (pp 188–189)

24. Ergot—the basics. I shall say something this morning on oxytoccics [sic]—that class of remedies which produce uterine contractions. At the head of the class stands the ergot, secale cornutum or spurred rye. When given in small doses to the male it gives rise to no effects, but would seem to have specific qualities when given to the parturient female.

It has been known for many centuries, but only to possess deleterious and poisonous qualities; and has been employed on the continent by female midwives as a promoter of labour pains for nearly 150 years. In 1808 Dr John Stearns of Saratoga county, this state, called the attention of the profession to its peculiar effects. He alluded to it as the Pulvis Parturiens.[39,40,41] It is nothing more than a diseased state of the common rye in which it is attacked with a spur, which soon bursts from its husk and assumes a lengthened form slightly curved. Its hue is in the first instance red, but soon changes to a dark violet or blackish colour; it is generally about one half to one inch long. In fact, its general appearance resembles the spur of a cock.

There are certain conditions where it is desirable to effect uterine contractions as soon as possible. There is probably no other medicine whereby we hope to arrest haemorrhage, dependent on any cause whatever and none more frequently given in the various forms of menorrhagia than ergot. (pp 197–199)

Can we always depend on the drug? Will it act on the uterus during parturition with the same certainty that castor oil will on the bowels? No, it is an uncertain remedy.

Suppose a country practitioner has in his office a quantity of this spurred rye. A certain case of labour comes along and deeming its administration indicated, he gives it and finds it produces no effects. In a few days, he has another case where he also thinks it indicated and administers it. In this case, perhaps, he will find that 2 or 3 grains will have more effect than 50 in the other case. Then again in a third case, 2 or 3 grains may only partially produce its effects. It is clearly then an uncertain remedy. It seems to require some peculiar unexplained condition of the nervous system.

Its administration will, as a rule, increase the frequency, force, and violence of the uterine contractions. (pp 200–201)

25. "Patience is a peculiar requisite of the obstetrician," or, ergot is dangerous and must not be used to save you time. Is there any difference in the contraction produced by this drug and the normal uterine contraction? In an ordinary labour pain, the contraction comes on and passes off at intervals; in other words, they are distinctly intermittent or paroxysmal. The contractions produced by the ergot are more powerful and violent and there is scarcely any interval. The pain begins, goes to a certain point and gradually recedes, but barely, before the recurrence of another. The main point then is the failure of the pain and contraction to subside and allow the patient that necessary relief and quiet that nature does—and you see immediately that it is by no means a safe remedy. With the natural contractions, the nervous system of the woman is not so much exhausted or prostrated; it also allows of these important changes in the soft parts.

In certain conditions therefore the administration of this drug would be very disasterous to the mother. Take a case where a woman has been in labour 10 or 20 hours, and the principal cause of the delay is in the rigidity of the soft parts. The interval such as labour allows is what is most wanted under these circumstances. But under the influence of ergot, this is not attained, and the cervix is lacerated or torn off; or as I have myself seen, the organ ruptures.

It is universally recognized now that children are extremely liable to be still-born where the drug has at all been indiscriminately used, as the cord or placenta are pressed upon to such a degree that asphyxia ensues or as some practitioners maintain, it gives rise to a poisonous condition of the mother's blood and this also causes asphyxia from imperfect aeration of the blood.

We will now study the conditions which contraindicate its administration and then those where it may be given satisfactorily.

The first rule I would lay down for you is never to give it with a view of relieving yourselves of time and trouble. Patience is a peculiar requisite of the obstetrician. (pp 201–204)

Note that Budd is here criticizing male doctors for this inappropriate interference and pulling. Shorter has recently attacked midwives for these practices as if only midwives had pulled at limbs or ever separated the head from the body.[38]

26. Opium given for rest may induce contractions. Let me say a few words on what I consider the rational treatment of debility. What would you do with a tired horse? Put him in the stable, give him a dose of oats, and let him rest. Spur him on to renewed work or beat him and you will do great injury to him. So it is with a woman. Give her a dose of opium and allow her to have an hour or two of rest and in this way you may save both mother and child.

Never leave a woman shortly after the administration of a dose of opium whether given with a view of taking the edge off excruciating pain or to cause relaxation of the soft parts. I have found it to produce an effect not only sedative, but not unlike the ergot, viz. of increasing the force of the uterine contraction. I have seen a woman dozing apparently sound and in a few minutes labour come on, and delivery be affected very quickly. Therefore in no case leave your patient. (pp 206–207)

27. Another danger of ergot. The next rule I would lay down is never to administer ergot unless the os uteri be fully dilated. I remember seeing a perfect ring of the cervix uteri which had been torn off from the violent uterine contractions causing the head to force its way down. This was presented at a Medical Society. Never administer it where you cannot reach the presenting parts. The membranes may rupture and down comes a shoulder, you introduce your hand, and try to get the feet; this you cannot accomplish from the violent uninterrupted contractions. (p 207)

Finally, as a rule you had better not give ergot to primiparae because the soft parts have not been subjected to that distension which is in the multiparae. (p 209)

28. When ergot may properly and safely be used. I have now stated on what conditions it is contraindicated, and you see there are not many cases left where it can be satisfactorily given. If the labour does not make any progress simply from inertia and there is none of the contraindications, you may administer it with benefit. Also where the head or breech present and there is none of the contraindications.

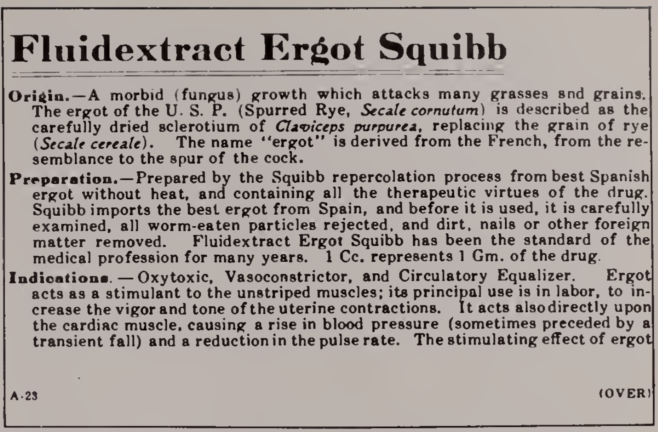
We may embrace all under the following rule. If there is no condition present save deficiency of uterine contraction, and that not dependent on uterine debility, and where you think the child will not be born before an hour, you may give ergot with impunity.

As a prophylactic against puerperal haemorrhage where it is at all alarming, I generally administer a full dose, just before the delivery of the placenta. There seems to be an inherent peculiarity in this drug, in some women, to quell this most efficiently. It has been my fortune to have several cases where women have come from a great distance to put themselves under my care when their life has been saved by this drug in previous labours.

Ergot may be given in substance, in vinous or alcoholic tincture, in the form of fluid extract or what is known as the oil of ergot, and the alkaloid ergotine.

If you give it in powder, be sure it is freshly powdered and unadulterated. You may prescribe 1 dram to be divided into 3 powders, one to be given every 20 minutes, or until its specific effect is produced. If it has no effect, send for another specimen. So with the tincture. It is a popular method in the country to make an infusion. (pp 209–211)

29. Product endorsement for Squibb. The fluid extract [of ergot] as prepared by Dr. Squibb is perhaps the most reliable; it seems to keep its quality for a longer period than any other preparation. I always use it when accessible.*** [Fig 3] (p 212)



Fluidextract Ergot Squibb

Origin.—A morbid (fungus) growth which attacks many grasses and grains. The ergot of the U. S. P. (Spurred Rye, *Secale cornutum*) is described as the carefully dried sclerotium of *Claviceps purpurea*, replacing the grain of rye (*Secale cereale*). The name "ergot" is derived from the French, from the resemblance to the spur of the cock.

Preparation.—Prepared by the Squibb repercolation process from best Spanish ergot without heat, and containing all the therapeutic virtues of the drug. Squibb imports the best ergot from Spain, and before it is used, it is carefully examined, all worm-eaten particles rejected, and dirt, nails or other foreign matter removed. Fluidextract Ergot Squibb has been the standard of the medical profession for many years. 1 Cc. represents 1 Gm. of the drug.

Indications.—Oxytoxic, Vasoconstrictor, and Circulatory Equalizer. Ergot acts as a stimulant to the unstriped muscles; its principal use is in labor, to increase the vigor and tone of the uterine contractions. It acts also directly upon the cardiac muscle, causing a rise in blood pressure (sometimes preceded by a transient fall) and a reduction in the pulse rate. The stimulating effect of ergot

A-23 (OVER)

FIGURE 3. Partial entry from a pharmacy file card issued by Squibb for their fluid extract of ergot. The other side of this card indicates the average dosage as 30 minims (2 cc): "Orally, its action begins in about fifteen minutes and continues for one-half hour." (Courtesy of the Library and Archives of the Squibb Institute for Medical Research, Princeton, New Jersey.)

30. Bloodletting of the mother.[28,29] Rigidity of the os I look upon as the most frequent cause of delayed labour. This may occur under a variety of circumstances. It is more apt to be met with in very young girls or in those well advanced in life but in their first labour. It is often, very often, found when the dilatation is retarded by disease of the os. No matter how long labour may be in the first stage, no danger may be apprehended except a certain amount of fatigue etc. There are a host of means employed with a view of remedying a protracted labour dependent on this cause. I regard a labour which occupies over 24 hours for its accomplishment a protracted labour. Bloodletting is, I suppose, the oldest remedy, and has attained the most extensive use both here and in Europe. Plethora of course may give rise to a rigid os, but not unfrequently we will find rigidity recurring in those who are very anemic. You perceive then, we are to discriminate between anemic and plethoric individuals if we resort to this. I believe it is rare now to bleed a parturient woman at all. I can remember when every woman who had a protracted labour had her arm tied up and a vein opened. When, however, your patient is very robust and plethoric, as some of you will meet with, you may bleed with great advantage. I have seen a rigid os melt down within an hour after opening a vein; such cases are exceptional, however. (pp 214–217)

31. Drugs to induce relaxation of the os. Tartar emetic . . . has been given many years with a view of relaxing contracted muscular fibre, but I have employed it not unfrequently and must admit I have never seen any good effects follow. It produces depression and a constant nausea, with

---

*** Edward Robinson Squibb, 1819–1900, was a surgeon and the founder of a laboratory in Brooklyn to prepare high quality pharmaceuticals, which grew into the modern multi-national corporation.[42,43]

a sensation as bad as that of sea sickness, which added to the other distress is truly a miserable condition to be in.[†††]

The administration of tobacco, in the form of enemata, with the same view of tartar emetic has been spoken of by some; but I look upon it as entirely out of the question. I have never used [it] nor shall I in any form as far as woman is concerned. (pp 217–218)

The internal administration of opium has also been advised; and it [like belladonna] under certain circumstances acts favorably. (p 219)

Let us consider if there are any means by which, as a rule, we can effect relaxation. I think there are. Place the patient under the influence of chloroform and the fibres will certainly relax. Another means, and one which the older authors deprecate is manual dilatation. This properly done is one of the most certain methods I know of. (pp 219–220)

32. Belladonna defended at a Medical Society meeting. There is an old adage that an ounce of prevention is better than a pound of cure; now let me tell you that, with a view of preventing rigidity of the os, I have for several years been in the habit of administering belladonna for a month or six weeks before the time of the expected labour, when the woman has previously had a rigid os. I forget the name of the gentleman with whom this treatment originated, but Dr Fordyce Barker was the first to speak of its good effects.[‡‡‡]

I commence giving a quarter of a grain three times a day and watch its effects. I am anxious if I do not find any constitutional evidence of the action of the drug, viz. dryness of the throat, flushed face, dimness of vision. I generally suspend its administration for a day or two and then resume giving it in the same doses that produced its effects before. Its administration has a peculiar and powerful effect on the cervix uteri. I have given it in more than 100 cases and have yet to see the first case where it has failed.

Interrogations have been put to me at Medical Society meetings. "Have you never seen it produce any evil effects on the child?" I answer "No! Never!" "Does not its administration have the effect of producing relaxation to such an extent as to cause haemorrhage during labour?" "No! Never!" I therefore regard it as a certain prophylactic against rigidity of the os during labour. (pp 223–225)

33. Colleagues criticized for rupturing the membranes. The membranes protrude and the liquor amnii is distinctly full in them, but they refuse to rupture. Now the too early intentional rupture of the membranes is to be deprecated. Practitioners often do this when there is not the slightest call for it whatever.[§§§] They act an important part in labour as a distensible wedge; they assist the os to dilate, and instead of the hard rim of the rigid os for the head to come against, the membranes form a pliable substance. Their intentional rupture then, as a rule, cannot be too strongly reprehended. When, however, the os is fully dilated, or only partially so but dilatable, and the presentation recognised, then if you have reason to suppose the delayed labour dependent on this cause, you are to rupture them, either by scratching with your nail or by a pocket probe, which can be conveniently done. Do not let your patient see the instrument, but bear it up on one side of your finger; some use a hair pin or tooth pick. (pp 229–230)

34. Gaining your reputation. Another cause [of delayed labour] is the imprisonment of the anterior lip of the uterus between the bones of the head and those of the pelvis, so that at each contraction it acts as a band keeping the head back. You make an examination and find the diameters of the pelvis large enough and the parts in the proper position, and you are apt to overlook this. It is an accident of frequent occurrence and the man who understands how to relieve it most satisfactorily will have more cases terminated satisfactorily, and quicker, and will bear that name in the community. The use of instruments is seldom required in cases of delayed labour dependent on this cause. (pp 233–234)

35. Banker's error. The various mental emotions will give rise to delayed labour. These act very powerfully on the nervous system of some persons. We have a melancholy instance of this within the last 24 hours in this city. The President of the North American Bank was taken with a fatal stroke of apoplexy on being informed of a reverse which happened to the bank through carelessness of him.[‖‖‖] The sudden announcement of news, whether exhilirating or depressing in character. [The sentence is incomplete in the manuscript.] I have seen instances where the appearance of a strange medical man has caused a complete cessation of the uterine contractions; and also cases where disappointment on account of the sex of the child has given rise to fatal haemorrhage from the contractions ceasing. You must therefore exercise discretion in informing the woman on any topic and be careful that nobody else says anything to her.[¶¶¶] (pp 239–240)

36. Chloroform used lightly. Suppose a woman is lingering hour after hour and [there is] no reason why the labour should not progress but the constant worrying of the woman. In such a case, chloroform is one resort, no matter what the mental excitement is due to. You need not carry the influence of the drug to its full extent, but just enough to put her in that state in which her thoughts lose connection. (pp 240–241)

[†††] Antimony and potassium tartrate, or tartar emetic, first became a popular medicine in the 1600s, and by the 19th century its undeniable powers had achieved a variety of uses. Notably, it could be substituted for bloodletting (especially for weak patients) and for ergot. In obstetrics it might also be used along with bleeding to relax the os uteri, to control violent labor, to calm puerperal convulsions, and to relax obstructed mammae.[44]

[‡‡‡] Benjamin Fordyce Barker, 1818–1891, was a founding faculty member of the New York Medical College from 1850 until 1860, when the entire faculty was replaced and Budd became their new Professor of Obstetrics; in 1861 Barker became Professor of Obstetrics and Diseases of Women and Children (later Professor of Clinical Midwifery and Diseases of Women) at the new Bellevue Hospital Medical College, and remained there until his death in 1890. He and Budd were colleagues on the staff at the Charity (or City) Hospital and as district physicians of the New York Asylum for Lying-in Women. Barker remained a stubborn opponent, even in the 1880s, of the introduction of antiseptic procedures to prevent puerperal sepsis.[45,46] Kelly and Burrage claim he was "instrumental in introducing the hypodermic syringe into America."[47]

[§§§] Shorter has criticized this practice among midwives, apparently unaware of how common it was—at least in Budd's view—among doctors as well.[48]

[‖‖‖] Under a headline proclaiming "Excitement in Wall-St.," the *New York Tribune* of Friday, January 25th, 1867 (the same day as this lecture), reports that Mr Yelverton, the President of the Bank of North America, was struck with apoplexy Wednesday evening at his home following the discovery that a check he had approved for over $200,000 did not have sufficient funds behind it, dying the next morning.[49]

[¶¶¶] Suitor observes from her reading of nearly 150 19th-century books on midwifery that "authors frequently told of cases where labor had been temporarily arrested by the entrance of a man to the lying-in room."[50] For example, the famous Dewees observed: "Every accoucheur has experienced a temporary suppression of pain upon his first appearance in the sick chamber."[51]

Near the beginning of Lecture VII, Budd also warns against whispering in the woman's presence for fear of distressing her. Such apparently sound advice, when directed to midwives, has been given a much different interpretation by Shorter, who recently wrote of "the chronic fatalism and fear of supernatural forces that one finds among traditional peasants. All were preoccupied with what could go wrong. 'Do not whisper in front of the mother,' to avoid alarming her, commanded article 8 of Kaufbeuren's 1737 midwife ordinance."[52]

37. Clinical instruction. Many authors object to the application of the actual cautery to the neck during the early months of pregnancy on the ground that cicatricial tissue will form there. Now experience is of more value than all theories. I have had cases myself, as I have told you in the Clinic, where I have applied it during pregnancy with good results and the dilatation of the neck not at all interfered with. Thereby showing that cicatricial tissue will melt down under the parturient act. (pp 242–243)

The diagnosis of a prolapsed cord is very easy. You feel a fold of slippery material, perhaps several inches long, but generally not as long as your thumb, which cannot be mistaken for anything else. It is also generally pulsating. I have found it hanging out like festoons, so much of it being prolapsed, and have been called by students when they thought the organ had ruptured and a fold of intestine descended. (p 261)

Apply your forceps guided by the same general rules and imitate nature as closely as possible in your movements; after you have effected the delivery of the head you feel so delighted that you undertake to pull off the forceps. Now do not loose your brains and wits and attempt to shake them off, as I have seen students do; but patiently unlock them.[###] (p 345)

38. Don't trust the nurse. In every case of delayed labour dependent on any cause whatsoever, watch the bladder; examine the hypogastric region every five minutes. Do not let the nurse's assertions on this point satisfy you. She may not be intending to deceive you, but does most assuredly. This can be accounted for by the patient thinking she has the desire to urinate when she asks the nurse for the chamber. The nurse looks at it and sees a little discolored water which she thinks the woman has passed, when perhaps 24 hours has elapsed since a single drop passed.[****] (pp 248–249)

39. Budd consults on a student's or former student's case. A member of our class last year called me to see a case where he had been in attendance a long while; he informed me on my arrival there was a face presentation and hydrocephalic head. I was rather inclined to ridicule his statement, but on careful examination found he was right. It was a very pretty diagnosis for a young student to make. (pp 253–254)

40. Studying even the unlikely problems. You must bear in mind, however, that all these causes of dystocia are comparatively rare, and require a vast amount of sound judgement and nicety to discriminate them. It is, however, of the utmost importance that you should know the appropriate remedy for each. (pp 255–256)

41. Mortality statistics for prolapse of the funis. If an individual has his larynx compressed he dies from strangulation; so does a child die from strangulation when the cord is compressed. As a general thing we find the umbilical cord stored away in the nooks and inequalities formed by the foetal body, so that its presence does not give rise to trouble. But sometimes it does. This morning I propose calling your attention to prolapse of the cord, the dangers attending to it, and its treatment. You will read of funis presentations. This is a misnomer. I do not recognize any presentation of the cord, but I do, prolapse. You know that it is frequently twisted around the body several times; this would seem to be a provision of nature against compression.

Let us look at the frequency of mortality in new born children from prolapse of the cord. Dr Churchill states that out of 365 [sic] cases where this occurred 220 were lost.[††††] In these, 107 were delivered by version and 37 by forceps [These figures agree with Churchill's table, p 420]. McClintock and Hardy report 29 cases of prolapse of the cord, where 25 died [in *Practical Observations in Midwifery and the Diseases Incident to the Puerperal State* (Dublin, 1843)]. And Dr Ramsbotham reports 1822 still births from all causes, where 128 were of prolapse of the funis, about 7 percent of the still births.[‡‡‡‡]

When we see that pelvic presentations occur in round numbers once in 50 cases and prolapse only one in 250, we may say that this is the most prolific source of death to the new born infant. As to the frequency of prolapse of the cord, Churchill reports it to have occurred 320 times in 90,983 or one in 280 labours.[§§§§] Dr Collins[56] reports 97 cases occurring in 16,652 [error for 16,654] births at the Dublin Lying-in Hospital [where Budd had studied after getting his MD in New York City], so that it is not of very unfrequent occurrence. (pp 256–258)

42. Innovation and a priority dispute. But experience has shown that all the instruments ever devised will not keep the [prolapsed] cord retained. Sir Richard Croft[‖‖‖] suggested and carried into use the method of introducing the entire hand and carrying the cord up as far as the fundus to hitch it on to the feet. But while the hand is up there why not make version and deliver the child at once.

About eleven years ago Dr Thomas of this city introduced his postural method. Some Germans and English have claimed priority, but he taught it unquestionably first, and the results as originally suggested by him have been of the most satisfactory character. Instead of three-quarters dying now of prolapse, it has become an exceptional thing to find a single death. Some 6 or 7 years ago he reported 47 cases where the treatment was carried out after his method and 37 were successful. Subsequent experience has shown that even a larger proportion than this can be saved. He compared the attempt to keep the cord in its place to keeping an eel in a bag with its mouth downwards and I think it a very fit comparison. Following out this homely example, he thought that if in any way he could reinvert the inclined plane of the uterus, that the result would be successful, so the idea occurred to him of turning the woman upside down or placing her on the knees and hands. This was tried and found to be successful.[¶¶¶¶] (pp 265–267)

[###] See also the Prof Gilman anecdote in Excerpt 47 and the recent study of clinical training by Atwater.[53]

[****] See also Excerpt 53 regarding urination, the nurses, and the catheter.

[††††] Fleetwood Churchill, 1808–1878, practiced in Dublin, publishing *Operative Midwifery* in 1841 and *Theory and Practice of Midwifery* in 1842. In Churchill (1848),[37] the cases in the table on p 420 add up to 385, but the text on p 421 says 220 of 355.

[‡‡‡‡] In Ramsbotham (1851),[54] 109 cases of prolapsed funis in 2263 still births, or 4.8%, are reported on p 535; perhaps Budd was using a different edition.

[§§§§] Churchill (1848)[55] indicates 431 cases in 98,512 as 1:245½; perhaps Budd was using a different edition.

[‖‖‖] English royal accoucheur, 1782–1818, who committed suicide after Princess Charlotte died in childbirth.[57,58]

43. A quiz question? Trunk presentations occur about as often as prolapse of the cord. Madame Lachapelle reports 68 cases in 15,652 labours, or about 1 in 230 [These figures appear in Churchill,[37] p 404]. Dubois found it 13 times in 2,020 deliveries. Dr Merriman found it to occur 1 in 255 cases in his private practice. And Dr Joseph Clark only 1 in 212. Naegela 1 in 180, and Collins 1 in 416.[37,62]

If the question is asked you, and it will, I assure you "How often may you expect to meet with presentations of the trunk?", answer 1 in 250.#### (pp 272–273)

44. Advice on trunk presentations. The intervention of art is absolutely necessary. Baudelocque, I told you, compared the foetus-in-utero to an olive in a long necked bottle. If the olive offered transversely, either one of the extremities must be substituted or one of two things will take place; the bottle will rupture, or the bottle [clearly an error for "olive"] be destroyed.*****

So with the child if it offers transversely. One of three things must take place; either the child be extracted piecemeal, the uterus rupture, or, by the introduction of your hand, the substitution of either the head or breech.

We are never to hope for the successful termination of a case without the intervention of art. Spontaneous version occasionally, though rarely, occurs. The shoulder ascending and the breech coming down or descending and the head coming down. In other cases we will find the body forced through to the inferior strait in a doubled up condition. This is denominated spontaneous evolution.

This is spoken of a great deal, and some men are fools enough when they find such a state of affairs to adjourn into an adjoining room and hope and pray. I say, there is about as much chance for this spontaneous evolution to occur as for her to be struck with lightning. It is almost a physical impossibility. We have no right as intelligent men to anticipate or scarcely hope for this; and if we do not know how to interfere skilfully, we should not be there.

You will read in medical journals of women being in labour 24, 48, or 72 hours or *days* with this presentation and finally the head or breech come down and the woman be safely delivered, but this is sheer nonsense. If the child is only of 6 or 7 months utero-gestation, it may double up in all shapes, and in such a case only, may you hope for spontaneous evolution. (pp 281–284)

45. Version as "the surgery of midwifery." This morning we commence studying what may be appropriately denominated the surgery of midwifery. We will first take up version or that operation by which one of the two extremities of the child is brought to the superior strait, and this we divide into cephalic, where we bring down the head, and podalic, where the inferior extremities are brought down to engage in the superior strait. This is done either by introducing the hand into the uterus, or by external manipulation.

The external manipulation has received a great deal of attention of late years; but it cannot be done in all cases. When the abdomen is lax and you can clearly trace out the position of the child, it is an operation attended with far less danger. (pp 284–285)

46. Cephalic version has advantages over podalic version, but the family must be advised in all cases. The operation of cephalic version is one which for many years had fallen into disuse; the podalic being almost entirely substituted for it. In certain circumstances it is very difficult. You can easily see how much easier it would be, when everything is tense and hard, to grasp the inferior extremities, than the slippery head, which recedes from your hold.†††††

But experience has demonstrated that more children are lost where the podalic version is performed than where the cephalic. This can be done provided the head is not too far up, the liquor amnii all evacuated, or the parieties [sic] of the abdomen too tight. Experience has also shown that even where the forceps are used in cephalic version more viable children are born than where recourse is had to podalic version. There are certain precautions to be observed before attempting version, no matter what object leads us to it.

It is universally admitted that the patient or some member of the family should be informed. If you are dealing with a nervous apprehensive woman, and from your cognizance of it you know the announcement of it to her would be detrimental to labour, it would be decidedly wrong to inform her. In this case let the husband know it. Then subject her to the influence of an anaesthetic. (pp 286–288)

Sometimes where the liquor amnii has not been evacuated and the abdominal parieties [sic] are lax and soft parts dilated, this operation can be performed with the utmost facility.

But look at the case if the liquor has been drawn off for many hours and the uterus is contracting with rapidity and force where the slightest attempt to introduce your hand gives the greatest pain, it is almost an impossibility to introduce the hand without administering an anaesthetic. (pp 286–289)

47. Anaesthesia very useful and very safe. Without the administration of an anaesthetic [version is] one of the most difficult operations in midwifery, and I might say in general surgery. . . . Only those who have had their hand in a contracting uterus can thoroughly comprehend the immense power it possesses. Nearly every obstetrician has had his hand paralysed at one time or another.

Professor Gilman was in the habit of narrating a case to the class, where he was standing along side of a medical

---

¶¶¶¶ Note that this same position was ridiculed as the habit of the "common Irish" in Budd's Lecture VII.

Theodore Gaillard Thomas, 1831–1903, was an adjunct lecturer for obstetrics at the University Medical College from 1855 to 1860; adjunct professor, then professor at the College of Physicians and Surgeons; and in 1868 he published his *Practical Treatise on the Diseases of Women*, which went through several editions and translations.[59,60] In his centennial history of American obstetrics and gynecology in 1876, Thomas gave this third-person account of the innovation: "In 1858, Gaillard Thomas published an essay (*Transactions of the New York Academy of Medicine*, Vol 12, p 21) upon the treatment of prolapse of the funis by gravitation developed as a remedial measure by placing the patient in the genupectoral position." He noted in 1876 that the procedure had formerly been used, but was so forgotten that for ten years after his innovation, its prior existence remained unknown to him.[61]

Lachapelle was identified in Excerpt 21. Paulin Dubois, 1795–1871, published *Traité complet de l'art accouchements* 2 vols (Paris, 1849, 1860) and served as head of the Maternité in Paris,[63] where Budd studied in 1856. Samuel Merriman, 1771–1852, published *A Synopsis of the Various Kinds of Difficult Parturition* (London, 1814; with many later editions and translations).[64] Joseph Clark, 1758–1834, was master of the Dublin Lying-in Hospital from 1786 to 1793. "Naegela" is an error for Naegele, who is identified above in Excerpt 10. Robert Collins, 1801–1896, married Joseph Clark's daughter and served as master of that same Dublin hospital from 1826 to 1833.[65] Collins published a remarkable treatise on the management of non-normal labors, based on his analysis of 16,654 births.[66]

***** Jean Louis Baudelocque, 1746–1810, played a major role in the establishment of a midwifery school at the Maternité in Paris.[34]

††††† Cutter and Viets credit Marmaduke Burr Wright (1854) with the American revival of cephalic version, which in Europe had generally been out of favor since Paré's writings in the 16th century.[67]

student who was attempting to produce [version], and he did not know who was bellowing the loudest, the woman or the student.[‡‡‡‡‡]

Whether we use ether or chloroform is entirely immaterial. It is strange that the first case of death from the administration of chloroform to a parturient woman has yet to be recorded.[§§§§§] (pp 289–290)

48. The doctor's discomfort with the woman's friends. We must now put her in a proper position [for effecting version]. This varies in different countries. In Great Britain she is placed on her left side, brought to the edge of the bed with the legs flexed on the thighs, and these again on the abdomen. On the continent of Europe she is placed cross-wise on the bed, the hips brought down to the edge of the bed, and the feet put on chairs. Call for three chairs. Place one foot on one, one on another, and the operator takes his position on the middle chair.

Three assistants are required, two to hold the limbs and one to administer the anaesthetic. It is preferable to have a professional man to administer the anaesthetic, but not necessary. The individuals to whom the limbs are entrusted are generally women, those garrulous old women of whom I have spoken not a little. You choose two who seem the most intelligent. Sometimes you get stuck with those who cannot hold their tongues, and so soon as you take your position, the first thing they do is to speak up, and perhaps tell you what to do. Now to avoid this, just set them down with their backs to you. (pp 291–293)

49. Non-aseptic procedures. [With the three chairs and the assistants so arranged], then prepare yourself. Take off your coat and roll up your sleeves. It is a hard road to travel often. Also take off any ring you have on your finger as you might lacerate the parts or lose it in the cavity of the uterus. A story is told of a Frenchman who lost a very valuable ring and had the nurse arrested. Some days subsequently it was found in the lochia. [Note that removal of a ring was not mentioned in any of the other discussions of internal examination.]

Lubricate your arm and back of hand thoroughly; do not put any oil on the palmar aspect of your hand as you want that to grasp the child, which is slippery enough under any condition. The doctor should also protect himself with a sheet or apron; it preserves his clothing from coming into contact with the liquor amnii and looks as though you were in no hurry. Another point. After you have got all these things arranged, particularly if you are in good society, spread some old clothing on the carpet. It looks cool and cleanly. [See also Excerpts 9 and 53.] (pp 293–295)

50. Errors of young practitioners. Ordinarily [in version] the young practitioner experiences difficulty in getting the bulk of his hand in, and will violently twist and push; but gentle and steady rotation will efface the folds of the vagina and allow the whole hand to enter. (p 301)

51. Patience is needed in version to avoid dangers to mother and child. With regard to the extraction, ought it to be done immediately, or having rectified the malposition, should it be left to nature? Suppose it is a shoulder that presents, and the feet are delivered. Feel the cord, if pulsating vigorously leave the case alone; but if not, and you have performed the operation with a view of curtailing the duration of labour in consequence of rupture, haemorrhage, convulsions, etc., you should deliver as speedily as possible.

Some practitioners so soon as they have a knee, rupture the membranes and perform evolution. This is to be deprecated. Although you are compelled to keep your hand in the uterus 5 or 10 minutes longer to enable you to reach the feet, the delivery will be accomplished more easily and satisfactorily and with less danger to mother and child. (pp 305–306)

52. Even doctors get sick (and joke about it). Dr Budd stated at the onset of the lecture that he was suffering from aphonia (which was quite evident) and was afraid that his attempt to lecture would be a practical illustration of an abortion; he was obliged to curtail any further remarks [regarding cases of an unduly short cord], but stated that this was a subject which was fully dilated on in the College text book, Cazeaux.[ǁǁǁǁǁ] (pp 308–309)

53. The forceps and chloroform. Yesterday [in Lecture XXIII on the use of the forceps], I was speaking of the preliminary precautions to be observed before employing the forceps. With reference to the administration of chloroform: some always use it, some seldom, and some never. It appears to me that on the grounds of humanity alone it should be used. This is a point on which a discussion was held a few years ago between two eminent men, Dr Simpson of Edinburgh and Dr Meigs of Philadelphia. The latter stating that under its influence the practitioner could not tell whether any of the soft parts were involved in the blades of the forceps or not. Simpson replies that he is sorry the students are taught in such a way as to have this accident befall them; and I think logically and scientifically he is correct.[¶¶¶¶¶]

The patient is to be brought to the edge of the bed in the same manner as directed for version. The practitioner ordinarily takes off his coat or protects himself. He then proceeds positively and accurately to ascertain the relation of the child to the maternal parts. The bladder, as I told you, is always to be evacuated; do not trust the statements of the nurse, but use the catheter. [On catheterization, see also the second paragraph of Lecture VII and Excerpt 38.]

[‡‡‡‡‡] Chandler R Gilman (1802–1865) had been Professor of Obstetrics at the College of Physicians and Surgeons[68,69] and American editor of Maunsell.[70] Gilman and Budd were colleagues in 1862 when both served as district physicians of the New York Asylum for Lying-in Women.[71]

[§§§§§] Here Budd would seem to be simply wrong and a few years behind the medical news. According to Duffy, "By 1863 a total of 123 deaths were attributed directly to chloroform in Great Britain alone, and the Royal Medical College, urged on by *The Lancet* and other medical publications, appointed a committee to investigate the subject. This committee . . . issued a long report in 1864, conceding that chloroform was potentially dangerous and recommending that a search be made for a safer substitute."[73] But in contrast to Duffy's pessimistic picture, Cassedy's recent study of statistical evaluations of anesthesia draws a conclusion that seems more in accord with Budd's view: "By 1848 . . . masses of favorable evidence for both [ether and chloroform] had generally swept away the initial skepticism. Widespread trials, both in the United States and in Europe, showed that they posed little danger even when used under poorly controlled conditions. Although a need for continued testing of anesthesia remained, individual American surgeons, dentists, and obstetricians by and large had no more doubts about its safety at midcentury than did the thousands of citizens who had taken to sniffing chloroform recreationally."[74]

[ǁǁǁǁǁ] In fact, this lecture is about as long as any of the others. Pierre Cazeaux, 1808–1862, published his major work in 1840, which appeared in English translation as *A Theoretical and Practical Treatise on Midwifery, Including the Diseases of Pregnancy and Parturition* in 1850 with further editions in 1857, 1863, 1866, 1868, 1875, and 1881.

[¶¶¶¶¶] Sir James Young Simpson, 1811–1870, had an international reputation and is especially famous for introducing chloroform as a childbirth anesthetic in 1847; in 1853 Queen Victoria accepted chloroform for the birth of Prince Leopold.[75,76] See Excerpt 10 for the identification of Meigs.

The forceps are to be warmed for the double purpose of taking the chill off them, and of cleansing them so as to make sure against contagion. For this purpose, immerse them in warm water, as it gives a more equal temperature; then lubricate them.

Ought we under any circumstances to have recourse to the forceps until the os is fully and thoroughly dilated? This is a point which gave rise to discussion for a long time. The old English school taught it as an axiom that they should not be employed until the head has descended sufficiently low to perceive an ear. Now you will see that this excludes a great number of cases. This practice was in vogue, and inculcated, as I say, in all the old textbooks on the subject. Craniotomy was the resort in all the secluded cases.

It is interesting to compare statistics of various obstetrical writers and observe to what an extent the destruction of foetal life was resorted to as the only means of safety to the mother. [All the following ratios are given in one table in Churchill's book.[77]] In Dublin Dr Clark used the forceps 1 in 728 cases and the perforator 1 in 248. Dr Collins in the same institution used the forceps 1 in 716 [error for 617] and the perforator 1 in 141. Siebold of Berlin used the forceps in [1] in 7 cases, the perforator 1 in 2,093. [Gap in manuscript, Budd] probably said Carus] of Dresden 1 in 14, perforator 1 in 283. Lachapelle 1 in 293, perforator 1 in 1,854. Baudelocque 1 in 501 [error for 561] and the perforator 1 in 2,898.#####

Americans are eclectics in many respects; we glean all that is good from the old schools and discard all that is bad. You will find that the perforator is used in this country not more than 1 in 1500 or 2000 cases, while the forceps are used 1 in 100 cases, or even less. Therefore if any contingencies arise which demand instrumental interference, although the head is at the superior strait, and you can introduce them without laceration, have recourse to the forceps before the perforator. (pp 323–329)

I hear I have not sufficiently dilated on the necessity of you always using the disengaged hand as a guide. Let me illustrate this by a case that occurred with a practitioner of very high standing not a thousand miles from here about a year or two ago. He was the attending obstetrician of a large hospital, and there was a case in which instrumental delivery was demanded. This gentleman announced that he had a new dodge of introducing the forceps; and went on to advise the class not to practice it, as they were not experienced. The dodge merely consisted in using only one hand [without employing the other as a guide]. He succeeded in introducing them and effecting a lock; but in the course of 48 hours the woman died, and a post mortem revealed a laceration into the peritoneum. (pp 333–335)

54. Sexual humor in the medical school classroom. They tell a good story of a distinguished obstetrician who was in the habit of narrating the following to his class. He was engaged to attend a woman who was in her first labour. As the case progressed he was frequently annoyed by the husband poking his head in at the door and enquiring about the case. At last delivery was accomplished; but there was a very slight laceration of the perineum. The husband, on enquiry, was informed that he was the father of a fine boy, and that everything was well save a slight tear which necessitated him to put in a few stitches. The husband had hardly shut the door when he returned asking, "How many stitches did you say doctor?" "Two!" was the answer. "Can't you put in three? That place was always a little too capacious." (pp. 343–344)

Acknowledgments. The author is especially grateful to Ms Marion Hanscom, Head of Special Collections at the University Library of SUNY-Binghamton, for invaluable assistance. For help in finding illustrations used in Part II, I am pleased to acknowledge Mr Gilbert Clausman, Librarian of the New York Unversity Medical Center, and Dr W. B. McDowell, Archivist of the Squibb Institute for Medical Research. A complete list of persons and institutions to whom I am indebted for other assistance in this research is given at the end of Part I, which appeared in the August issue of the *Journal*.

Drs Clark and Collins are identified above in Excerpt 43. Siebold of Berlin refers to Adam Elias von Siebold, 1775–1828, professor of obstetrics at Berlin and not to his more famous son, Eduard Caspar Jacob von Siebold, 1801–1861, the author of a magisterial history of obstetrics.[78] Carl Gustav Carus, 1789–1869, was professor of gynecology at Dresden, a Naturphilosoph, and a friend of Goethe; his *Lehrbuch der Gynäkologie* was published in 1820.[79] Lachapelle is identified in Excerpt 21 and Baudelocque in Excerpt 44.

Cassedy's recent study of statistics in American medicine considers the use of obstetrical statistics only as they were created by American practitioners and reported in medical journals, without noting the significant role European figures, especially the large aggregations of births in the Dublin and Paris hospitals, played in American midwifery textbooks and teaching.[80] Budd's use of statistics appears as well in Excerpts 22, 41, and 43.

REFERENCES

1. Schwarz GS: Society, physicians, and the corset. *Bull NY Acad Med* 1979; 55:551-590.

2. Haller JS Jr, Haller RM: *The Physician and Sexuality in Victorian America.* New York, WW Norton and Co, 1977, pp 146–174.

3. Mohr JC: *Abortion in America: The Origins and Evolution of National Policy 1800–1900.* New York, Oxford University Press, 1978.

4. Mohr JC: Patterns of abortion and the response of American physicians, 1790-1930. In Leavitt JW (ed): *Women and Health in America.* Madison, University of Wisconsin Press, 1984, p 117.

5. Ibid, pp 118–119.

6. Cott NF: Passionlessness: an interpretation of Victorian sexual ideology, 1790–1850. *Signs: J Women in Culture and Society* 1978; 4:219–236.

7. Degler CN: What ought to be and what was: women's sexuality in the nineteenth century. *Amer Hist Rev* 1974; 79:1467–1490, with criticism and response, 1975; 80:1079–1080.

8. Op cit, Haller JS Jr, Haller RM, pp 92–102.

9. Shade WG: "A mental passion": female sexuality in Victorian America. *Int J Women's Studies* 1978; 1:13–29.

10. Smith-Rosenberg C: The female world of love and ritual: relations between women in nineteenth-century America. *Signs: J Women in Culture and Society* 1975; 1:1–29.

11. Smith-Rosenberg C: The hysterical woman: sex roles and role conflict in 19th-century America. *Social Research* 1972; 39:652–678.

12. Smith-Rosenberg C, Rosenberg C: The female animal: medical and biological views of woman and her role in nineteenth-century America. *J Amer Hist* 1973; 60:332–356.

13. Bennet JH: *A Practical Treatise on Inflammation of the Uterus and its Appendages, and on Ulceration and Induration of the Neck of the Uterus.* 2nd American ed. from the 2nd London ed. of 1849. Philadelphia, Lea and Blanchard, 1850.

14. Garrison FH: *An Introduction to the History of Medicine*, 4th ed. Philadelphia and London, WB Saunders Co, 1929, p 508.
15. Jameson E: *Gynecology and Obstetrics* (Clio Medica Primers on the History of Medicine, 17). New York, Hafner Publishing Co, 1962 (rept. of 1936 ed.), p 61.
16. Clendening L: *Source Book of Medical History*. New York, Dover Publications, 1960 (rept. of 1942 ed.), pp 605–606.
17. Ibid, pp 606–610.
18. Ibid, pp 611–621.
19. Richmond PA: American attitudes toward the germ theory of disease, 1860–1880. *J Hist Med and Allied Sciences* 1954; 9:447–453.
20. Heaton CL: Obstetrics at the New York Almshouse and at Bellevue Hospital. *Bull NY Acad Med* 1940; 16:45–46.
21. Ricci JV: A glance into the history of the City Hospital and into the lives of its gynaecologists and obstetricians. *Contributions from the Department of Gynaecology of the City Hospital New York [Blackwell's Island, later Welfare Island]* 1945; 1:49–50.
22. Cutter IS, Viets HR: *A Short History of Midwifery*. Philadelphia and London, WB Saunders Co, 1964, p 204.
23. Meigs CD: *Obstetrics: The Science and Art*. Philadelphia, Lea and Blanchard, 1849, 1852, 1856, 1863, 1867.
24. Op cit, Cutter IS, Viets HR, pp 159–160.
25. Dewees WP: *A Compendious System of Midwifery*. 1st ed. Philadelphia, Carey and Lea, 1832, p 147.
26. Op cit, Haller JS Jr, Haller RM, p 275.
27. Op cit, Dewees WP, p 199.
28. Sidall AC: Bloodletting in American obstetrical practice, 1800–1945. *Bull Hist Med* 1980; 54:101–110.
29. Seigworth GR: Bloodletting over the centuries. *NY State J Med* 1980; 80:2022–2028.
30. Kelly HA, Burrage WL: *Dictionary of American Medical Biography*. New York and London, D Appleton and Co, 1928, pp 659–660 and 408.
31. Jenkins JF: Report on spontaneous umbilical hemorrhage of the newlyborn. *Trans AMA* 1858; 11:263–318.
32. Fisher GJ: Obituary of J. F. Jenkins. *Trans Med Soc State of NY* 1884, pp 369–387.
33. Op cit, Kelly HA, Burrage WL, p 660.
34. Op cit, Cutter IS, Viets HR, pp 90–94.
35. Op cit, Garrison FH, p 605.
36. Smith FB: *The People's Health, 1830–1910*. New York, Holmes and Meier, 1979, p 83.
37. Churchill F: *On the Theory and Practice of Midwifery*. 3rd Amer. ed. Philadelphia, Lea and Blanchard, 1848. (Other editions 1843, 1846, 1855, 1860, 1862, 1863, 1866.)
38. Shorter E: *A History of Women's Bodies*. New York, Basic Books, 1982, pp 79–81.
39. Thoms H. John Stearns and pulvis parturiens. *Am J Obstet Gynecol* 1931; 22:418–423.
40. Thoms H: *Classical Contributions to Obstetrics and Gynecology*. Springfield, IL, Charles C Thomas, 1935, pp 21–25.
41. Op cit, Clendening L, pp 186–188.
42. Serendipity in our files: In memoriam Edward Robinson Squibb, M.D. (1901). *NY State J Med* 1982; 82:1271-1272.
43. Blochman LG: *Doctor Squibb. The Life and Times of a Rugged Idealist*. New York, Simon and Schuster, 1958.
44. Haller JS Jr: The use and abuse of tartar emetic in the 19th-century materia medica. *Bull Hist Med* 1975; 49:247–252.
45. Walsh JJ: *History of Medicine in New York: Three Centuries of Medical Progress*. 5 vols. New York, National Americana Society, 1919; 4:17–18.
46. Op cit, Ricci JV, pp 10–11, 21–22, 24.
47. Op cit, Kelly HA, Burrage WL, pp 60–61.
48. Op cit, Shorter E, pp 58–60.
49. Excitement in Wall-St. *The New York Tribune* January 25, 1867, p 8.
50. Suitor JJ: Husbands' participation in childbirth: a nineteenth-century phenomenon. *J Family History* 1981; 6:290.
51. Op cit, Dewees WP, pp 107–108.
52. Op cit, Shorter E, p 54.
53. Atwater EC: "Making fewer mistakes": a history of students and patients. *Bull Hist Med* 1983; 57:165–187.
54. Ramsbotham FH: *The Principles and Practice of Obstetric Medicine and Surgery in Reference to the Process of Parturition Illustrated with 148 Figures*. 6th American ed. Philadelphia, Blanchard and Lea, 1851.
55. Op cit, Churchill F, p 420.
56. Collins R: *A Practical Treatise on Midwifery, Containing the Result of Sixteen Thousand Six Hundred and Fifty-four Births, Occurring in the Dublin Lying-In Hospital During a Period of Seven Years Commencing November 1826*. Philadelphia, Haswell, Barrington, and Haswell, 1838, pp 170–177.
57. Op cit, Cutter IS, Viets HR, pp 187–188.
58. Donegan JB: *Women and Men Midwives: Medicine, Morality, and Misogyny in Early America*. Westport, Greenwood Press, 1978, pp 193–194.
59. Op cit, Walsh JJ, 5:455–458.
60. Op cit, Jameson E, p 62.
61. Thomas TG: Obstetrics and gynaecology, in Clarke EH, Bigelow HJ, Gross SD, Thomas TG, Billings JS: *A Century of American Medicine, 1776–1876*. Philadelphia, Henry C. Lea, 1876, p 250.
62. Op cit, Collins R, p 43.
63. Op cit, Cutter IS, Viets HR, p 207.
64. Ibid, pp 211–212.
65. Ibid, pp 121–122.
66. Op cit, Collins R.
67. Op cit, Cutter IS, Viets HR, pp 172–176.
68. Op cit, Ricci JV, pp 6–7.
69. Op cit, Kelly HA, Burrage WL, pp 468–469.
70. Maunsell H: *The Dublin Practice of Midwifery, With Notes and Additions by Chandler R. Gilman*. New York, William A. LeBlanc, 1842.
71. Op cit, Ricci JV, p 24.
72. Duffy J: Anglo-American reaction to obstetrical anesthesia. *Bull Hist Med* 1964; 38:36–37.
73. Ibid, p 38.
74. Cassedy JH: *American Medicine and Statistical Thinking, 1800–1860*. Cambridge, MA, Harvard University Press, 1984, pp 89–90.
75. Op cit, Jameson E, pp 57–59.
76. Op cit, Clendening L, pp 373–377.
77. Op cit, Churchill F, p 340.
78. Op cit, Cutter IS, Viets HR, p 203.
79. *Neue deutsche Biographie*. Berlin, Duncker and Humblot, 1953–; 3:161–163.
80. Op cit, Cassedy JH, pp 80–83.

Perinatal aspects of high multiple gestation

JALLEH VAFAI, MD, DONALD L. SHAPIRO, MD

One in 41 million couples conceive quintuplets; one in 729,000 conceive quadruplets. Such multiple gestations are unique experiences for the parents and physicians. The arrivals are often heralded by the mass media. In 1981 the Perinatal Center of the Strong Memorial Hospital experienced the exciting challenge of helping to manage the pregnancy, labor, delivery, and postnatal care of quintuplets. The purpose of this paper is to report observations on the delivery. The infrequent occurrence of high multiple gestation is reflected in the small number of articles on this topic. Most published papers concern obstetrical management. In the current era of perinatal care, such high risk pregnancies and deliveries must involve close cooperation between the obstetrician and the neonatologist if successful outcomes are to be achieved.

CASE REPORT

A 28-year-old woman sought treatment for secondary infertility. She had had one previous pregnancy which resulted in a healthy full-term infant three years prior to this consultation. She was treated with menotropins (Pergonal®), and she conceived within the same cycle.[1] Evaluation of the pregnancy by ultrasound at approximately 10 weeks gestation revealed a quintuplet pregnancy. The pregnancy proceeded without complications until approximately 20 weeks gestation when there was onset of uterine activity and some cervical dilation. The patient was admitted to the hospital, and intravenous ritodrine therapy was begun. Uterine activity stopped, and the patient was maintained on oral ritodrine and complete bed rest.

Anemia and problems of skin care secondary to prolonged bed rest developed while the patient was in the hospital. Repeated ultrasound examination showed all fetuses to be growing in the 25 to 50 percentile. At 32 weeks gestation, labor began which could not be suppressed by intravenous ritodrine. A primary cesarean section was performed. All the newborn were delivered within a two-minute period. One infant required intubation in the delivery room. All five infants had some degree of respiratory distress. Apgar scores and admission data are summarized in Table I.

DISCUSSION

Incidence. The conception of twins occurs in approximately 1 out of 90 pregnancies in the United States. The incidence is different for monozygotic and dizygotic types. Dizygotic twinning is in part genetically determined. There is a definite familial inheritance pattern, and its incidence increases with maternal age and parity. Monozygotic twinning is not related to any known factors and has a frequency of 3.5 in 1,000.[2] The incidence of triplets is approximately 1 in 8,000 pregnancies; which is the square of twin incidence (1 in 90)^2.[3] By using Hellin's hypothesis, the incidences may be calculated for quadruplets—(1 in 90)^3, quintuplets—(1 in 90)^4, and sextuplets—(1 in 90)^5. Further calculations may be made up to the highest reported multiple birth, nantuplets.

Quintuplet pregnancy occurs spontaneously once in every 41 million births. The incidence of multiple gestation has increased in the past two decades due to extensive use of drugs which induce ovulation, such as human pituitary gonadotropins (HPG), human menopausal gonadotropins (HMG), and clomiphene.[4] Treatment by ovulation inducing drugs increases the multiple gestation rate to 10% to 30%. A quintuplet pregnancy following clomiphene induction of ovulation was reported in 1975. Three cases of quintuplet livebirths following gonadotropin therapy, and five quintuplet pregnancies after induction of ovulation with clomiphene and HMG-HCG are found in the literature. This report adds another case of quintuplet pregnancy after treatment with menotropins and HCG.

Several studies suggest a correlation between oral contraceptive use and multiple births. For instance, it has been found that women who had taken birth control pills sequentially for a period of six months or longer had ap-

TABLE I. Admission Data and Hospitalization Periods of Quintuplets

Quintuplets	Weight	Apgars 1,5 Minutes	Peripheral Hematocrit (%)	Hospitalization Days
Boy A	1,540	2,6	60	58
Boy B	1,540	5,7	62	47
Girl C	1,280	5,7	67	58
Boy D	1,500	7,8	54	38
Girl E	1,380	7,9	53	64

From the Division of Neonatology, Department of Pediatrics, Creighton University School of Medicine, Omaha, Nebraska (Dr Vafai), and the Division of Neonatology, Department of Pediatrics, University of Rochester School of Medicine, Rochester, New York (Dr Shapiro).

Address correspondence to Dr Vafai, Instructor in Pediatrics, Division of Neonatology, Department of Pediatrics, Creighton University School of Medicine, 601 North 30th St, Omaha, NE 68131.

proximately twice the expected rate of conceiving twins soon after cessation of usage.[5] Another group showed a possible correlation between maternal age and multiple gestation after contraceptive use. In this study women aged 35 and older had a higher multiple pregnancy rate than the control groups and than younger women who had also used oral contraceptives.[6] The increased use of oral contraceptives may be partially responsible for the increased rate of multiple births in the last two decades.

Diagnostic criteria for multiple gestation. A family history of twins or multiple pregnancy and a history of drug-induced ovulation may be suggestive of multiple pregnancy. The clinical suspicion of multiple gestation arises from finding a uterine size significantly large for expected dates, polyhydramnios, auscultation of two or more heart beats which differ by 10 or more beats per minute, or palpation of multiple fetal parts. Maternal serum levels and urinary excretion of human chorionic gonadotropin and human placental lactogen are elevated in multiple gestations.[7] In general, however, laboratory values are not reliable criteria for evaluation of multiple gestation.

When the diagnosis of multiple gestation is suspected, ultrasound should be used to confirm the diagnosis. The first multiple pregnancy diagnosed by ultrasound examination was reported in 1967. Since then, with improvement in the method of ultrasound and the availability of expert personnel, ultrasound has become the method of choice for the diagnosis of multiple gestation.[8,9] It should be noted, however, that for a variety of reasons the failure to obtain an accurate fetal biparietal measurement is almost three times greater for a multiple versus a single pregnancy.

With the advent of ultrasound, a new phenomenon has been observed with sequential studies. One or more gestational sacs have been seen to be reabsorbed during the pregnancy without any apparent harm to the remaining fetus.

Psychological factors affecting the parents. With the diagnosis of multiple births, parents are faced with a considerable amount of emotional stress. The families' regular lifestyle may be interrupted by a prolonged hospital stay and bed rest for the mother. During the pregnancy, specific anxieties arise concerning the future of the enlarged family. Stress factors include the actual pregnancy as well as concern for the future care of the infants, scheduling and the necessity for assistance, as well as long-term concerns such as education and the economic problems of a large family.

TABLE II. Inverse Correlation Between Mean Birthweight, Gestational Length, and Increasing Multiplicity[2,7]

Type of Pregnancy	Gestational Length (Days)	Mean Birthweight (grams)
Singleton	280.5	3,377
Twin	261.5	2,395
Triplet	247	1,818
Quadruplet	237	1,395
Quintuplet	224	1,448

Duration and other problems of pregnancy. There is an inverse correlation between the duration of the pregnancy and increasing multiplicity. Table II shows this relationship and shows the decrease in mean birth weight of the multiple births up to quadruplets. Data from the quintuplets reported on here are added. It is clear that as the number of fetuses in the uterus increases, both the mean birth weight and the gestational length decrease. Prematurity and intrauterine growth retardation are the primary causes of perinatal mortality and morbidity in multiple gestation.

Frequent maternal complications are associated with multiple pregnancies. Anemia (hemoglobin level less than 10 g/100 mL), polyhydramnios, hypertension, and pre-eclampsia occur 2 to 3 times more often than with a single pregnancy. Additional maternal problems include a higher incidence of bleeding in early pregnancy (40%) and a higher occurrence of spontaneous abortion (36.7%). Other problems which are more common in multiple gestation include malpresentation, placenta previa, premature rupture of the membranes, and prolapse of the umbilical cord. The incidence of malformations also increases with multiple births.[7,10]

Prenatal management. The early diagnosis of high multiple gestation is important because careful medical management is necessary throughout the pregnancy. The risks of multiple pregnancy, such as prematurity, growth retardation, fetal asphyxia, and antepartum fetal death, are known, and the goals of the medical treatment are to decrease these complications and to support the mother during the pregnancy.

Prevention of premature labor is the first priority in the management of a high multiple gestation pregnancy. Early bed rest during gestation may be advantageous. A recent study explored the effect of bed rest in relation to neonatal deaths and found that those women who had bed rest from the 28th week had fewer infant fatalities.[11] Although no increase in gestational length has been associated with bed rest, a potential positive effect is a rise in fetal weight due to an increase in uterine blood flow caused by bed rest. Table II indicates that this may have been a factor in the quintuplets reported here. Additional data are required to substantiate this hypothesis.

Another controversial method of extending the gestational period is the elective application of a cervical suture at 12 to 15 weeks of pregnancy.

The early use of oral beta-mimetic agents to prevent premature labor in selected circumstances is increasing, particularly in multi-fetal pregnancy.[12] Relaxation is effected by the stimulation of beta receptors in the uterus. If uterine contractions are already in progress, agents can be administered intravenously, adjusting the dose depending on the the effect of the drug on uterine contractility. One side effect of beta-mimetic agents is maternal and fetal hyperglycemia. It has been observed that fetal growth may be accelerated by the hyperglycemic state. Another potential advantage of beta-mimetic agents is the decrease in respiratory distress syndrome resulting from accelerated fetal lung maturation. It is important to be aware of the added risk of possible maternal fluid retention caused by beta-mimetic agents; when given in conjunction with corticosteroids, the risk of pulmonary edema increases.[13]

Pharmacologic induction of fetal lung maturation has been recommended in high multiple gestation pregnancies. In some reports, betamethasone has been given on a prophylactic basis at 26 to 28 weeks gestation. The dose of betamethasone is the same in both single and multiple gestations (12 mg every 24 hours for two doses); one dose per week is administered up to the time of delivery.

The use of amniocentesis to detect surfactant production is difficult in multiple gestations. It is not sufficient to accept the results from one test, since there may be significant variations between the fetus' lung maturation. There is one report of a successful amniocentesis for genetic diagnosis in a triplet pregnancy. The procedure was performed using indigo carmine as a marker, and it was performed under sonographic control.[14]

Management of labor and delivery. Because there is an increased incidence of complications such as dysfunctional labor, malpresentation, and early and late postpartum hemorrhage, a cesarean section also minimizes the possibility of asphyxia resulting from delayed delivery of any of the infants. The establishment of a combined obstetric-neonatal team is important for advanced delivery preparation. In order to provide adequate emergency treatment, it is necessary to anticipate potential complications. With this patient preparations were complete by the 25th week of gestation. Delivery was deferred by complete bed rest and beta-mimetic agents until 32 weeks of gestation when labor occurred and could not be suppressed. During this period five standby teams of neonatal personnel were ready. It was predetermined that each was assigned to work with a specific infant, and each team was numbered accordingly.

In addition to maintaining personnel at all times during the six-week period, equipment was also made ready to receive the five newborns. Despite peak periods in the intensive care nursery, it was necessary to constantly ensure the availability of facilities in units of five. The delivery room was organized to provide a place for five warmer-beds equipped with oxygen, suction, monitors, and resuscitation sets for each infant.

All five infants were born within two minutes. As each baby was removed, color coded cord clamps were used to coordinate each infants' umbilicus with his or her attachment to the placenta. There were separate chorioamnionic sacs for each infant. The quintuplets were brought to the intensive care nursery in transport isolettes. On arrival in the nursery, each infant was placed in an assigned warmer-bed. Organization was a key element in the successful delivery.

Neonatal management. Perinatal mortality is increased in high multiple gestation pregnancies because of prematurity and low birth weight. There is no significant difference between neonatal mortality and morbidity in a single birth versus multiple births when the infants are of comparable weight and gestational age. Since respiratory distress syndrome is inversely related to gestational age and prematurity, it occurs more frequently in multi-fetal births. During the first month of life the mortality rate continues to be higher in multiple births. The rate for twins is 5 times higher than that for a single birth and is approximately 20 times higher among triplets. The rate of stillbirths is also greater in multiple gestation. It is twice as high for twins, three times as high in triplets, and four times as high in quadruplets, as compared to single births.[2]

Once successful delivery has occurred, the prime concern of neonatal care is the treatment of prematurity and respiratory distress syndrome. These may be complicated by other problems of multiple gestation, such as birth asphyxia and interfetal transfusion syndrome. A major concern is to have sufficient personnel and equipment to provide care for the simultaneous arrival of a group of sick premature babies in a normally active intensive care nursery.

These quintuplets overcame problems ranging from respiratory distress syndrome, patent ductus arteriosus, and apnea in the least sick infant to one infant who suffered severe respiratory distress syndrome, sepsis, bilateral pneumothorax, intraventricular hemorrhage, and mild hydrocephalus. The four other infants had normal head development based on ultrasound examinations. All the babies had a patent ductus arteriosus which was closed by indomethacin, or it closed spontaneously following fluid restriction. All the babies were free of apnea and bradycardia for a period of time prior to their discharge from the hospital. However, on reviewing the follow-up of the quintuplets, baby A died of Sudden Infant Death Syndrome at age five months. At this point in time all the other babies were readmitted to the hospital for examinations. They were monitored at home until they reached eight months of age. There was no further occurrence of apnea. We suggest home apnea monitoring in cases of high multiple gestation. Table 1 indicates the length of the hospital stay for each infant.

In summary, the early diagnosis of high multiple gestation is crucial. Any instance in which a maternal factor might increase the chance of a multiple birth should initiate early investigation by ultrasound examination. If a pregnancy is diagnosed as a high multiple gestation, a team of neonatologists and obstetric perinatologists should be involved with the perinatal care and delivery decisions. The mother should have adequate bed rest and should be monitored for nutritional and circulatory status. Tocolytic drugs and steroid therapy should be considered in treatment. Pending delivery, neonatal teams should be organized for each baby in the delivery room. Cesarean section is the optimal procedure for prevention of birth asphyxia and birth trauma. Organization and cooperation are the keys for successful management of high multiple gestation pregnancies.

REFERENCES

1. Muechler EK, Huang K-E: Plasma estrogen and progesterone in quintuplet pregnancy induced with menotropins. *Am J Obstet Gynecol* 1983; 147:105–106.
2. Cetrulo CL, Ingardia CJ, Sbarra AJ: Management of multiple gestation. *Clin Obstet Gynecol* 1980; 23:533–548.
3. Behrman RE, Vaughan VC: The fetus and the neonatal infant, in Nelson WE (ed): *Textbook of Pediatrics*, 12th ed. Philadelphia, WB Saunders, 1983, p 340.
4. Schenker JG, Laufer N, Weinstein D, Yarkoni S: Quintuplet pregnancies. *Eur J Obstet Gynaecol Reprod Biol* 1980; 10:257–268.
5. Rothman KJ: Fetal loss, twinning and birth weight after oral-contraceptive use. *N Engl J Med* 1977; 297:468.
6. Harlap S: Multiple births in former contraceptive users. *Br J Obstet Gynaecol* 1979; 86:557–562.
7. Ganesh V, Apuzzio J, Iffy L: Clinical aspects of multiple gestation, in Iffy L, Kaminetyky H (eds): *Principles and Practice of Obstetrics and Perinatology*, Vol 2. New York, John Wiley & Sons, 1981, p 1183.
8. Bailey D, Flynn AM, Kelly J, O'Conor M: Antepartum fetal heart rate and monitoring in multiple pregnancy. *Br J Obstet Gynaecol* 1980; 87:561–564.
9. Powell-Phillips WD, Wittmann BK, Davison BM: Real-time ultrasound B-scan as an aid to antepartum fetal hart rate monitoring in multiple pregnancy case report. *Br J Obstet Gynaecol* 1979; 96:666–667.
10. Farr V: Prognosis for the babies early and late, in MacGillivray I, Nylander PPS, Corney G (eds): *Human Multiple Reproduction*. Philadelphia, WB Saunders, 1975, pp 191–192.
11. Loucopoulos A, Jewelewicz R: Management of multifetal pregnancy: Sixteen years' experience at the Sloane Hospital for Women. *Am J Obstet Gynecol* 1982; 143:902–905.
12. Richter R: Evaluation of success in treatment of threatening premature labor by betamimetic drugs. *Am J Obstet Gynecol* 1977; 127:482–486.
13. Fuchs F: Prevention of premature birth. *Clinics in Perinatology*, 1980; 7:3–15.
14. Hausknecht RU, Yeh HC, Godmilow L: Prenatal genetic diagnosis in a triplet gestation. *Obstet Gynecol* 1981; 58:382–385.

See also p 527.

Rounded atelectasis

GIL HAUER SANTOS, MD, HELEN MOREHOUSE, MD

Folded lung syndrome or rounded atelectasis is not a well known entity. Awareness of the clinical history and roentgenographic picture of this localized pulmonary parenchymal infolding could enable its diagnosis without the need for a thoracotomy. If a diagnosis is not made before surgery and a thoracotomy is subsequently performed, an inspection of the area involved and local decortication will be sufficient for diagnostic and therapeutic purposes, without the need for further pulmonary resection.

CASE REPORT

A 64-year-old man with a history of alcohol abuse was admitted to Bronx Municipal Hospital Center after suffering a head injury that resulted in left hemiparesis. He had also sustained two fractured ribs and a small hemothorax (Fig 1) that did not necessitate insertion of a chest drainage tube. His neurologic symptoms regressed under steroid medication and he was discharged from the hospital after four weeks.

Nineteen months later he was readmitted to the hospital with extensive cellulitis of the right leg in which there were maggots. A chest film at this time demonstrated a rounded, well-defined parenchymal density with pleural thickening in the lower lobe of the right lung. Other roentgenographic changes were not fully appreciated before surgery (Fig 2). Considering that this was a new finding not present on the previous recent admission, an exploratory thoracotomy was performed as soon as the leg infection resolved. Considerable thickening of the pleura was found in the area where the mass was expected to be. No pleural plaques were seen. Further inspection of the lung did not demonstrate other abnormal findings. At this point, chest films taken before surgery were reviewed, and the typical "comet tail" [1] pattern was identified. Careful decortication of the constricting pleura was performed, liberating the underlying folded lung parenchyma. Re-expansion of the lung after removal of the fibrinous peel showed complete re-expansion of the constricted area of atelectasis. Chest films taken after surgery revealed total disappearance of the "mass." A pathology report was consistent with pleural fibrosis. No asbestos bodies were seen, even with the aid of iron stains.

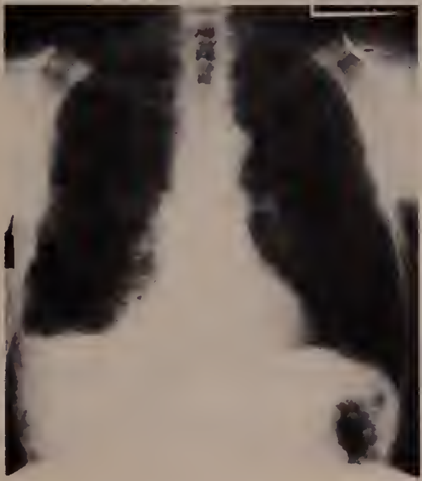


FIGURE 1. Posteroanterior chest roentgenogram taken on initial admission to the hospital. Fractured right sixth and seventh ribs (arrowheads) with associated hemothorax is demonstrated.

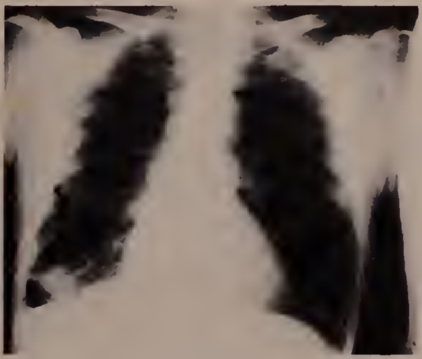


FIGURE 2. Posteroanterior chest film obtained on second admission to the hospital reveals a rounded, well-defined parenchymal density with associated pleural thickening in the right lower lobe. Vessels and bronchi of right lower lobe are drawn to a point (comet tail sign; open arrow). The right hilum and minor fissure (arrow) are lower, indicating volume loss.

DISCUSSION

The presence of a mass in the lung of a 64-year-old patient that was not detected on chest films taken 19 months before has to be considered to be a carcinoma until proved otherwise. This was our impression. The size of the density, 3 cm in diameter, and its shape were consistent with a fast growing lesion. However, other distinctive findings such as the "comet tail" sign[1-4] and volume loss in the lower lobe of the right lung reflected by the lowered minor fissure and right hilum, were not fully appreciated before surgery. If more detailed scrutiny of films and prompt awareness of the rounded atelectasis syndrome had occurred, surgery could possibly have been avoided in this patient.[1]

Rounded atelectasis is a peripheral form of atelectasis that is usually localized in the lower lobes of the lungs and is associated with pleural disease. According to several authors,[3-6] such pleural changes are most often associated with exposure to asbestos. In the patient presented here, however, the triggering factor could have been a pleural reaction following a hemothorax produced by fractured ribs. It appears that the fibrinous peel deposited around a limited area of the slowly infolding lung produced the picture of rounded atelectasis. Decortication of this area of lung parenchyma was first reported by Heine[7] in 1962. According to Hanke,[1] the possibility of entrapment of small portions of lung parenchyma due to localized pleural thickening had appeared in the German literature since 1928.

In 1971 Hanke presented a series of 22 patients with rounded atelectasis and, more recently, the same author[1] in association with Kretzchmer published reports of more than 80 cases of this entity. It is their belief that rounded atelectasis originates as a result of the presence of a pleural effusion—of any cause—followed by collapse of an area of lung above the lower edge of the lung. This lower part of lung, still aerated, will float up and fold over the area of atelectasis, in this way starting the events that will produce infolding of the lung. The high fibrin content of the pleural effusion will then be responsible for the trapping of this folded lung. There was no mention in their paper of asbestosis as a causative agent.

From the Departments of Surgery (Dr Santos) and Radiology (Dr Morehouse), Albert Einstein College of Medicine, Bronx, New York.

Address correspondence to Dr Santos, Albert Einstein College of Medicine, 1300 Morris Park Ave, Bronx, NY 10461.

Recently Mintzer and Cugell[4] proposed to emphasize the relationship of rounded atelectasis with asbestos-induced pleural disease as previously done by other authors,[3] by identifying 15 patients with "historical information confirming asbestos exposure" in a group of 18 patients with rounded atelectasis. They failed, however, to demonstrate the presence of pleural or pulmonary asbestos-induced disease. The only radiologic sign that was used to support their claim was "thickened intralobar fissure, a manifestation of asbestos-induced pleural disease." Evidently a thickened intralobar fissure is neither restricted to nor demonstrative of asbestosis.

Based on the reports available and our own experience, it is proper to say that rounded atelectasis is an infolding of the lung usually in the lower lobes, possibly tethered in that position by constricting forces produced by localized pleural fibrosis.[1,8-10] It is detected on chest films as a solitary lung mass in the lower lobes associated with lowering of the fissure, lowering of the hilum, pleural thickening, and the "comet tail" sign. This sign is produced by crowding of vessels and peripheral bronchi arriving at the area of atelectasis. When the diagnosis is made, invasive procedures are not necessary.[5,9] If, however, the diagnosis is not made prior to surgery, a simple decortication will establish the true nature of the "mass."[1,5,9] A lobectomy is not advised.[5] Once the lung is exposed, a detailed inspection of the suspicious area will not reveal a hard mass but only a thickening of the visceral pleura. Once this pleural peel is removed, the lungs will re-expand, confirming the diagnosis of rounded atelectasis.

REFERENCES

1. Hanke R, Kretzschmar R: Round atelectasis. *Semin Roentgenol* 1980; 15:174-182.
2. Schneider HJ, Felson B, Gonzalez LL: Rounded atelectasis. *AJR* 1980; 134:225-232.
3. Mintzer RA, Gore RM, Vogelzang RL, Holz S: Rounded atelectasis and its association with asbestos-induced pleural disease. *Radiology* 1981; 139:567-570.
4. Mintzer RA, Cugell DW: The association of asbestos-induced pleural disease and rounded atelectasis. *Chest* 1982; 81:457-460.
5. Case Records of the Massachusetts General Hospital (Case 24-1983). *New Engl J Med* 1983; 308:1466-1472.
6. Blesovsky A: The folded lung. *Br J Dis Chest* 1966; 60:19-22.
7. Heine F: Faltungsphanomene der Lunge. *Internist* 1962; 3:357-363.
8. Payne CR, Jaques P, Kerr IH: Lung folding simulating peripheral pulmonary neoplasm (Blesovsky's syndrome). *Thorax* 1980; 35:936-940.
9. Dernevik L, Gatzinsky P, Hultman E, Selin K, William-Olsson GW, Zettergren L: Shrinking pleuritis with atelectasis. *Thorax* 1982; 37:252-258.
10. Stark P: Rounded atelectasis: another pulmonary pseudotumor. *Am Rev Respir Dis* 1982; 125:248-250.

See pages 527-528.

Acute cholecystitis due to *Aeromonas hydrophila*

WILLIAM J. PARSONS, MD

Aeromonas hydrophila is a gram-negative organism well recognized as a primary pathogen in diseases of marine and warm-blooded animals.[1] Human diseases associated with this organism include cellulitis, wound infections,[2-4] septicemia in immunocompromised patients,[4,5] urinary tract infections,[1,3,4] and diarrheal syndromes.[2,3,6]

A hydrophila has been isolated from patients with hepatobiliary disease.[7,8] Its role as a potential pathogen in this setting is supported by the following report of a patient with acute cholecystitis.

CASE REPORT

A 47-year-old Navajo man was admitted to the hospital in 1983 with epigastric pain, nausea, and vomiting after ingesting a large fatty meal. Examination on admission revealed a temperature of 37.6°C, mild epigastric tenderness, and diminished peristalsis. Laboratory studies disclosed a white blood cell count of 15,300/mm^3, normal serum alkaline phosphatase and bilirubin levels, and a marginally elevated SGOT value. An ultrasound of the gallbladder showed a thickened wall and an echodense homogeneous bile, without evidence of gallstones. The patient's temperature rose to 38.4°C on the next day. Initial treatment included intravenous ampicillin; gentamicin was added to the regime when his temperature remained elevated. Laparotomy on the fourth hospital day revealed an inflamed gallbladder which was filled with purulent material and was acalculous. The liver appeared grossly normal. Cholangiograms obtained during laparotomy were unremarkable. The patient had an uneventful recovery, receiving intravenous gentamicin through the seventh day after surgery.

Histologic examination of the gallbladder revealed acute cholecystitis and an intramural abscess with incipient perforation, measuring 1.0 cm in diameter. The cystic duct was patent and of normal size, without histologic abnormality.

Aerobic bile cultures grew an oxidase-positive, gram-negative rod identified at the New Mexico State Laboratory as *Aeromonas hydrophila*. Anaerobic bile cultures were negative. Blood cultures drawn prior to beginning gentamicin therapy grew *A hydrophila*. Isolates from both bile and blood were resistant to ampicillin and cephalothin and were sensitive to gentamicin, amikacin, tetracycline, and chloramphenicol. An *E coli* was also isolated from the blood cultures drawn prior to gentamicin therapy which was resistant to ampicillin and sensitive to gentamicin.

DISCUSSION

The clinical presentation and intraoperative findings of this patient were consistent with acute cholecystitis due to *A hydrophila*. Bacteremia and histologic evidence of incipient perforation of the gallbladder wall by an acute pyogenic necrotizing process implicate a virulent biliary infection.

The presence of a second organism in the patient's blood of known pathogenic potential is of note. However, it is unlikely that it could cause the acute necrotizing process observed and then escape identification in the bile (*E coli* was specifically looked for).

From the Departments of Internal Medicine, Gallup Indian Medical Center, Gallup, New Mexico, and the University of Rochester Medical Center, Rochester, New York.

Address correspondence to Dr Parsons, Department of Internal Medicine, Strong Memorial Hospital, 601 Elmwood Ave, Rochester, NY 14642.

A hydrophila has been isolated from the blood and ascitic fluid of patients with cirrhosis.[2,3,7] The organism has been noted in the bile of patients with sclerosing cholangitis[3,5] and in patients with biliary obstruction due to pancreatic carcinoma.[4] From most of these bile specimens other organisms of known pathogenicity had been isolated. Cholecystitis associated with *A hydrophila* infection has been noted in three previous reports.[3,8] The bile was not cultured in one patient, and other organisms of known hepatobiliary pathogenicity were noted in the bile of the remaining two patients.

In this patient *A hydrophila* was isolated in pure culture from a focus of acute inflammation within the biliary system. The role of *A hydrophila* as a potential pathogen in hepatobiliary disease is supported by this finding.

Acknowledgments. The author appreciated the thoughtful comments of Dr Richard Hornick and Dr Raphael Dolin.

REFERENCES

1. Slotnick IJ: *Aeromonas* species isolates. *Ann NY Acad Sci* 1970; 174:503-510.
2. Davis WA 2nd, Kane JG, Garagusi VF: Human aeromonas infections: a review of the literature and a case report of endocarditis. *Medicine* 1978; 57:267-277.
3. Von Graevenitz A, Mensch AH: The genus *Aeromonas* in human bacteriology. *N Engl J Med* 1968; 278:245-249.
4. Washington JA 2nd: *Aeromonas hydrophila* in clinical bacteriologic specimens. *Ann Intern Med* 1972; 76:611-614.
5. Bulger RJ, Sherris JC: The clinical significance of *Aeromonas hydrophila*. *Arch Intern Med* 1966; 118:562-564.
6. Burke V, Gracey M, Robinson J, Peck D, Beaman J, Bundell C: The microbiology of childhood gastroenteritis: *Aeromonas* species and other infective agents. *J Infect Dis* 1983; 148:68-74.
7. Conn HO, Fessel M: Spontaneous bacterial peritonitis in cirrhosis: variations on a theme. *Medicine* 1971; 50:161-197.
8. Defronzo RA, Murray GF, Maddrey WC: *Aeromonas* septicemia from hepatobiliary disease. *Am J Dig Dis* 1973; 18:323-331.

Neonatal hydrops of the gallbladder

Evaluation by cholescintigraphy and ultrasonography

JOHN B. AMODIO, MD, EILEEN FONTANETTA, MD, MORRIS COHEN, MD, AMIEL Z. RUDAVSKY, MD, MURRAY J. ROSENZWEIG, MD

Hydrops of the gallbladder is a non-calculous, non-inflammatory distention of this organ. In childhood this entity may mimic intra-abdominal emergencies such as intussusception or appendicitis.[1] Hydrops of the gallbladder rarely occurs in the neonate. The radiologist must be aware of the possibility of this disorder in the neonate who presents with jaundice and a palpable abdominal mass. Diagnostic studies should include ultrasonography and ^{99m}TC-DISIDA (diisopropyliminodiacetic acid) cholescintigraphy. Though the disorder is frequently the result of sepsis, there may be no apparent cause. Spontaneous resolution usually occurs. A case of hydrops of the gallbladder in a premature infant of a mother with diabetes mellitus is presented.

CASE REPORT

The patient was a 1,720-gram female infant born at 32 weeks gestation. She was delivered by cesarean section because of uncontrolled maternal diabetes mellitus. The mother was a 39-year-old woman (gravida 4 para 2) who had been followed in the prenatal high risk clinic because of the diabetes. Spontaneous rupture of the membranes occurred 48 hours prior to delivery; the mother was not febrile.

Physical examination of the neonate revealed microtia of both ears and a mass in the right upper quadrant. Blood cultures for aerobic and anerobic organisms were drawn because of prolonged premature rupture of the maternal membranes and prematurity of the infant. The patient was initially placed on prophylactic antibiotics; these were discontinued when all cultures were reported to support no growth.

The infant developed hyperbilirubinemia on the second day of life. The bilirubin remained elevated for four days, reaching a maximum level of 16 mg% total and 1.4 mg% indirect. Multiparameter serum analysis was otherwise normal.

An initial plain film of the abdomen obtained immediately after birth was unremarkable except for a 3 cm × 1 cm mass in the right upper quadrant. A sonogram of the right upper quadrant, performed on the sixth day of life using a 7.5-MHz transducer at medium range focus, showed marked distention of the gallbladder without stones or sludge (Fig 1). The liver and right kidney were unremarkable.

^{99m}TC-DISIDA cholescintigraphy was performed on the eighth day of life using 670 Ci of ^{99m}TC-DISIDA. The gallbladder was not visualized within four hours despite

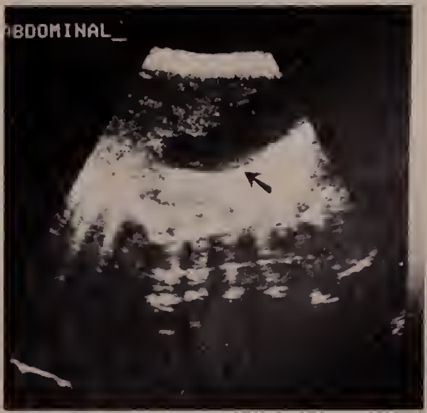


FIGURE 1. Longitudinal ultrasonogram of the right upper quadrant demonstrating a 3.5 cm × 1 cm distended gallbladder (arrow). No sludge or gallstones are present.

From the Departments of Radiology (Drs Amodio and Rosenzweig), Pediatrics (Drs Fontanetta and Cohen), and Medicine, Nuclear Medicine and Radiology (Dr Rudavsky), North Central Bronx Hospital, Montefiore Medical Center, and Albert Einstein College of Medicine, Bronx, NY.

Address correspondence to Dr Rosenzweig, Associate Attending Radiologist, Department of Radiology, Montefiore Medical Center, 111 East 210th St, Bronx, NY 10467.

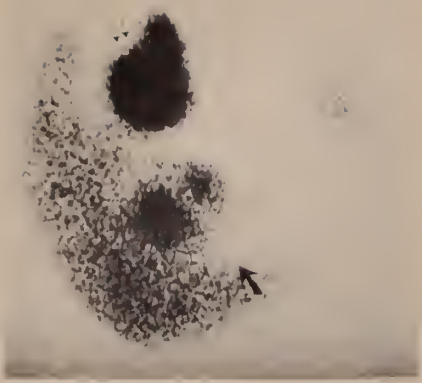


FIGURE 2. ^{99m}TC-DISIDA cholescintigram taken four hours after injection demonstrating good hepatic uptake and excretion into the common duct. The gallbladder is not visualized suggesting cystic duct obstruction. A photon deficient area in the region of the gallbladder fossa is present (arrow), probably representing the distended gallbladder. Reflux into the stomach is also present (double arrows).

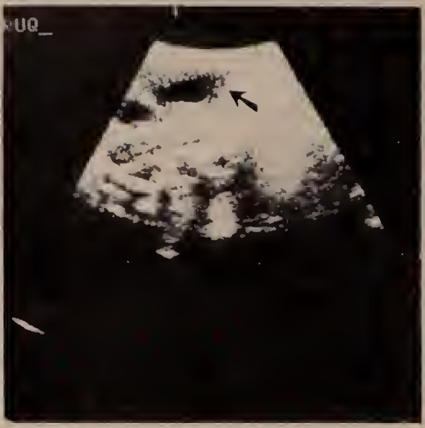


FIGURE 3. Longitudinal ultrasonogram of the right upper quadrant demonstrating a normal size gallbladder (arrow), indicating decompression has taken place.

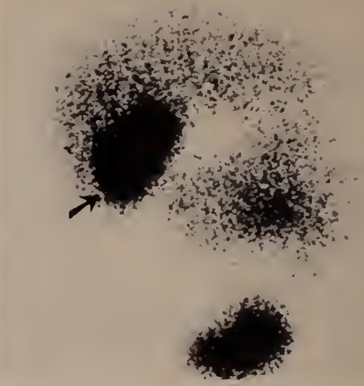


FIGURE 4. ^{99m}TC DISIDA cholescintigram demonstrating visualization of the gallbladder (arrow) 10 minutes after injection.

adequate fasting and prompt hepatic uptake and excretion into the biliary system (Fig 2).

From day eight to day 19 the infant had onset of mild congestive heart failure, and a diagnosis of ventricular septal defect was made by echocardiogram. The infant was able to be maintained on digoxin and lasix. The mass in the right upper quadrant gradually diminished in size, as determined by palpation, and the bilirubin level began to decrease.

A repeat sonogram, performed on the 19th day of life, revealed a gallbladder of normal size (Fig 3). DISIDA cholescintigraphy obtained on the same day demonstrated prompt visualization of the gallbladder, indicating cystic duct patency (Fig 4). These findings coincided with the disappearance of the right upper quadrant mass and a return of the bilirubin level to within normal limits.

DISCUSSION

Hydrops of the gallbladder is an uncommon problem in infants and children. Most reported cases occurred as complications of other illnesses. The majority of cases occurred in association with infections, including group A streptococcus, salmonella, and pseudomonas sepsis,[2] leptospirosis, and cervical adenitis. The disorder has also been reported as a component of the abdominal crisis of the mucocutaneous lymph node syndrome.[1,3] Congenital malformations of the cystic duct can also produce this condition. Dutta[4] reported a case of a small cystic duct in an infant with subsequent hydrops of the gallbladder due to periductal inflammation.

The mechanism of this disorder in infancy may be related to transient bile plugging of the ductal system. Robinson[5] described angulation of the cystic duct in this condition leading to obstruction of the duct. Non-visualization of the gallbladder at four hours after injection, as in this patient, suggests that some form of cystic duct obstruction is associated with hydrops of the gallbladder.

If the cystic duct straightens and the obstruction is relieved the gallbladder may decompress with prompt drainage. Spontaneous resolution can then occur.

Ultrasonography and DISIDA cholescintigraphy are the most useful diagnostic methods for hydrops of the gallbladder. Those patients who have dilation of the gallbladder on sonographic examination and failure to visualize the gallbladder on radionuclide studies should be suspected of having this disorder. In patients in whom cholescintigraphy demonstrates filling of the gallbladder despite sonographic findings of gallbladder dilation, other diagnoses should be considered. Inertia of the gallbladder, possibly analogous to the "colon inertia syndrome" described in infants born to mothers with diabetes mellitus, might be an explanation for such findings. Ultrasonography alone would not make this differentiation; sepsis should be excluded. In patients with sepsis, repeated sonographic and radionuclide studies should be utilized to document resolution with treatment. However, no cause for this condition may be apparent and spontaneous resolution may occur. When no resolution of the disorder has taken place, cystic duct abnormalities should be sought; hypoplasia of the cystic duct has been described. Moreover, cystic duct atresia has been associated with hydrops of the gallbladder,[6,7] requiring cholecystectomy.

REFERENCES

1. Bradford BF, Reid BS, Weinstein BJ, Oh KS, Girdany BR: Ultrasonographic evaluation of the gallbladder in mucocutaneous lymph node syndrome. *Radiology* 1982; 142:381-384.
2. Neu J, Arvin A, Ariagno RL: Hydrops of the gallbladder. *Am J Dis Child* 1980; 134:891-89.
3. Westfall CT, Masello TP: Kawasaki disease associated with hydrops of the gallbladder. A case report and review of the literature. *Z Kinderchir* 1981; 32:379-383.
4. Dutta T, George V, Sharma GD, et al: Gallbladder disease in infancy and childhood. *Prog Ped Surg* 1975; 8:109-118.
5. Robinson AE, Erwin JH, Wiseman HJ, Kordroff MB: Cholecystitis and hydrops of the gallbladder in the newborn. *Radiology* 1977; 122:749-751.
6. Scobie WG, Bently JF: Hydrops of the gallbladder in a newborn infant. *J Pediatr Surg* 1969; 4:457-459.
7. Swischuck LE: *Radiology of the Newborn and Young Infant*. Baltimore, Williams and Wilkins, 1980, p 470.

MSSNY Malpractice Fund Honor Roll

At the June 1985 meeting of the House of Delegates of the Medical Society of the State of New York, the delegates adopted a resolution intended to replenish the Society's special Malpractice Fund, which had been almost depleted during the previous several months by the combined public relations and legislative campaign for solutions to the medical liability insurance crisis.

The resolution called for the solicitation of voluntary contributions to the fund from MSSNY members and non-member physicians as well. The resolution also directed that these contributions be deposited in the Malpractice Fund to be used in the continuing efforts to correct the still-critical liability situation, and also to be used in connection with other public issues directly impinging on the practice of medicine.

Many of our members, and some non-members, have already contributed; MSSNY wishes to express its sincere gratitude for their generosity by listing their names in this Honor Roll.

George J. Lawrence, Jr., MD
Executive Vice-President

Sallah Uddin Abbasey, MD
Yousif M Abdel-Jawad, MD
R Thomas Abderhalden, MD
Robert Adolph Abel, MD
Abram Joseph Abeloff, MD
Irving Abelow, MD
Louis Charles Abelson, MD
Lambert R Abeyatunge, MD
David Abisch, MD
Brian I Abraham, MD
David A Abraham, MD
Martin Wm Abrams, MD
Charles N Accettola, MD
Murat Acemoglu, MD
Ramkrishna Achut Acharya, MD
A Bernard Ackerman, MD
Martin Ackerman, MD
Marvin Ackerman, MD
James Adam, MD
Gaspar Mathew Adamo, MD
Alvin Addes, MD
Irving M Ader, MD
Robert Ira Adler, MD
Dominick C Adornato Jr., MD
Alexander Afalonis, MD
Juan Simon Afif, MD
Galaa M Agban, MD
Andrew Lewis Ageloff, MD
Joseph S Agnello Jr., MD
Kavasseri S Agneshwar, MD
Shyam Narayan Agrawal, MD
Demetrio A Aguila Jr., MD
Gabriel Aguilar, MD
Saul G Agus, MD
Raphael Aharon, MD
Nargess Ahgarian, MD
Anwar S Ahmad, MD
Syed Kader Ahmed, MD
Ibrahim Al-Sinjari, MD
Anthony S Alaimo Sr., MD
Ireneo V Albano, MD
Joel Albert, MD
Joan Thysen Albin, MD
Valiere Alcena, MD
Bassam N Aldaia, MD
Catalina Alegre-Levine, MD
Steven K Alexander, MD
J Edwin Alford, MD
Kenneth M Alford, MD
Corazon Laygo Alialy, MD
Victor Alinovi, MD
Eugene Allen, MD
Diogenes Antipas Almonte, MD
Herman S Alpert, MD
Allen David Alt, MD
Edgar David Altchek, MD
Martin Altchek, MD
Ralph Altman, MD
Dolores B Amar, MD
Salvatore V Ambrosino, MD
Stanley Amelkin, MD
Gregory A Amendola, MD
Joseph Charles Amico, MD
Achala Hasmukh Amin, MD
Hussein Kamel Amin, MD
Rasiklal N Amin, MD
Margot Ammann, MD
Frank John Amodio, MD
Emmanuel Joseph Amoury, MD
William S Andaloro, MD
George Downey Anderson, MD
John Randall Anderson, MD
Albert F R Andresen Jr., MD
Francis Joseph Anello, MD
Basil Anghelis, MD
Martin Asare Anim, MD
Joseph Antenucci, MD
Kenneth David Anthone, MD
Anthony C Antonacci, MD
James V Aquavella, MD
Jose A Arandia, MD
Cecilia Tesoro Arboleda, MD
Joel Strom Archer, MD
Frank P Arena, MD
Dominic S Ariola, MD
Frank A Ariosta, MD
Sureyya Naci Arkun, MD
Herbert Stanley Arm, MD
Enrique Armenta, MD
Migdalia Arnan, MD
I M Frances Arndt, MD
Philip Roger Aronson, MD
Alexander John Arony, MD
Marianne W Arseneau, MD
Martin Robt Artman, MD
Matthew A Asbornsen, MD
Robert C Ascher, MD
John Joseph Asheld Jr., MD
Jeffrey Roy Ashkin, MD
Michael S Ashman, MD
William John Athos, MD
Marshall Erding Atwell, MD
Neil August, MD
Stanley August, MD
Anthony R Aurello, MD
Paul Auspitz, MD
Edward H Axelrod, MD
Murty S R Ayyala, MD
Arnold Leonard Bachman, MD
Paul Bachner, MD
Leo Bagg, MD
Bruce A Bagley, MD
Charles P Bailey, MD
Robert W Baird, MD
Bruce Edward Baker, MD
John G Baker, MD
Richard Holden Baker, MD
Virginia Harriet Baker, MD
Ronald A Balkin, MD
Peter Herbert Ballen, MD
Venkataraman Balu, MD
Charles Felix Banas, MD
Burton Banner, MD
Pasquale D Baratta, MD
Susan Veronica Barbieri, MD
Anthony Joseph Bardinelli, MD
Barbara Ann Barker, MD
A Addison Barman, MD
Gerard A Barnaby, MD
Jay Gold Barnett, MD
Richard John Baron, MD
James Vincent Barrett, MD
Eric R Barth, MD
Alberto Marcio Bartoli, MD
Neil Mark Barton, MD
Ernst Georg Bartsich, MD
Joseph Boutros Basile, MD
Naef Khoury Basile, MD
Joel David Baskoff, MD
William Batiuchok, MD
Lillian Batlin, MD
Biagio Battaglia, MD
Arthur F Battista, MD
Arthur Bauman, MD
Caroline E G Bauman, MD
Judah Bauman, MD
Walter Baurle, MD
Donald Henry Baxter, MD
Vincent Bayarri, MD
Douglas L Beards, MD
Robert Wayne Beasley, MD
Christopher J Beatty, MD
Fritz J D Beauboeuf, MD
Alfred Becker, MD
Glenn Adam Becker, MD
Michael Ira Beckerman, MD
Daniel Theodore Beer, MD
Donald Jay Behr, MD
A L Loomis Bell, MD
James Milton Bell, MD
Edward T Bello, MD
Marvin S Belsky, MD
Dominick Bendo, MD
Benedict Bruno Benigno, MD
Sacha Benjamin, MD
Vallo Benjamin, MD
Zachary H Benjamin, MD
Leslie G Bennett, MD
George W Benninger, MD
Seyton G Bentham, MD
Samuel L Beranbaum, MD
Dicran A Berberian, MD
Frank Berchenko, MD
Jonas Beregovich, MD
Abraham Berens, MD
Murray John Berenson, MD
Marc Steven Berenzweig, MD
Lawrence Berger, MD
Leland Sylvan Berger, MD
William Matthew Berger, MD
Edward P Bergin, MD
Robert B Bergmann, MD
Harold A Bergner, MD
Howard Berk, MD
Husnu Sinan Berkay, MD
Daniel Berkenblit, MD

Elena Rebeca Berkowitz, MD
Stanley D Berliner, MD
Jay B Berlowe, MD
Morton Henry Berman, MD
Richard Edward Berman, MD
Robert William Berman, MD
Robert William Bernard, MD
Viola W Bernard, MD
Bernard Bernhardt, MD
Harvey Eric Bernstein, MD
Nathan K Bernstein, MD
Robert Henry Bernstein, MD
Sidney A Bernstein, MD
Robert L Bernzweig, MD
Gerald J Berrafati, MD
Karla L Berry, MD
Frank A Bersani, MD
Bernard Harvey Berson, MD
Harold E Berson, MD
Robert W Bertcher, MD
Arthur P Bertolino, MD
Charles A Bertrand, MD
Charles J Bertuch, MD
Vicki C Beshlian, MD
Milton Best, MD
Allan Elie A Beyda, MD
Bernadette A Beyda, MD
Barbara Conley Beyer, MD
Milton Ronald Beyers, MD
Nellikunje G Bhat, MD, P.C.
Khalid Rashid Bhatti, MD, P.C.
Robert Lawrence Bialkin, MD
Anthony E Biancardi, MD
Quinton John Bianchine, MD
Hylan Arthur Bickerman, MD
Paul A Bienstock, MD
Marvin Wallace Bierman, MD
Samuel Richard Bierstock, MD
Charles Mayo Biller, MD
Lawrence G Bilsky, MD
Lee S Binder, MD
Robert Throne Binford Jr., MD
Giovanni Biondi, MD
Vera Birch, MD
Etta Gerstein Bird, MD
Eric Arac Birken, MD
Allan M Birnbaum, MD
Morton Birnbaum, MD
Laszlo Biro, MD
Merlyn Abel Bissell, MD
John C Bivona Jr., MD
Norman Sidney Blackman, MD
Charles R Blair, MD
Donald W Blair, MD
James Morgan Blake, MD
Howard Steven Blank, MD
Stephen Warren Blatchly, MD
Stanley M Blaugrund, MD
Paul Lautant Bleakley, MD
Michael Jay Blecker, MD
Alan Ralph Bleich, MD
Jody S Bleier, MD
Charles J Bleifeld, MD
John A Bleski, MD
Brian Block, MD
Jerome Mervyn Block, MD
Donald J Blodgett, MD
Berna Jean Romyn Blom, MD
Melvin Bloom, MD, P.C.
Alan Arthur Bloom, MD
Allan Ellia Bloomberg, MD
Randall D Bloomfield, MD
Charles Irvin Bloomgarden, MD
David Karl Bloomgarden, MD
Marvin Bruce Bloshinsky, MD
Curt Bluefeld Jr., MD
Alexander Blum Jr., MD
Gerald Myron Blum, MD
Harry Blum, MD
Lester Blum, MD
Hyman Blume, MD
Ralph Stuart Blume, MD
Jerome Harris Blumen, MD
Serge Blumenfeld, MD
Gerald Blumenthal, MD
Jerome B Blumenthal, MD
Mordecai Bluth, MD
Eugene Julius Bluto, MD
Alan Ashley Bober, MD
David L Bobker, MD
Barbara A Boccia, MD
Eugene L Bodian, MD
Martin Bodian, MD
Robert Kenneth Boese, MD
Michael A Bogdasarian, MD
Robert M Bogdasarian, MD
Richard J Bonanno, MD
Walter R Bonime, MD
Johan Pieter Bonk, MD
Robert Bonvino, MD
Judith Ann Boone, MD
Robert Lee Boothe, MD
John Richard Border, MD
Michael Borecky, MD
Louis Raymond Borelli, MD
Custodio R Borgueta, MD
Mark H Borkin, MD
Sonia Iclea F Borra, MD
Mario E Bortolussi, MD
Peter A Bossart, MD
Mounir N Boulos, MD
Nazmy S Boulos, MD
Michael Aaron Bowser, MD
Alden Kelsey Boyd, MD
Dewey McDonald Boyer, MD
Thel Gordon Boyette Jr., MD
Michele Helen Boyle, MD
James Rodgers Boynton, MD
Paul Aaron Bradlow, MD
Salim Braick, MD
John C Braico, MD
Kathleen T Braico, MD
Stephen Ira Braitman, MD
John B Branche, MD
Harold Brandaleone, MD
Joseph Ralph Brandy Jr., MD
Roger William Brassel, MD
Carl W Braun, MD
Manfred Braun, MD
Paul Wm Braunstein, MD
Paul Joseph Brayer, MD
Emanuel Brechner, MD
Melvin Jos Breite, MD
Kenneth Morton Bremer, MD
Andrew B Bremness, MD
William C Brender, MD
Joseph R Brennan, MD
Robert A Brenner, MD
Robert J Brenner, MD
Seymour Brenner, MD
Vero A Brentjens, MD
Henry Brill, MD
Arthur David Brimberg, MD
Leonard Joseph Bristol, MD
Thelma Brock, MD
Linda Susan Brodsky, MD
Donald Charles Brody, MD
Matthew Brody, MD
Bruce Louis Brofman, MD
Fred R Bromberg, MD
Melvin Bronstein, MD, P.C.
Burton Bronsther, MD
Kenneth Haskell Brookler, MD
Andrew R Brookner, MD
Donald Libolt Brooks, MD
Elba Nelly Brooks, MD
Michael Brooks, MD
Marie L Brown-Wagner, MD
Frederick Robt Brown, MD
Roy Hershel Brown, MD
Stanley M Brown, MD
Martin H Brownstein, MD
Ronald C Bruce, MD
Elana Bruckmann, MD
Joseph Brudny, MD
Donald F Bruhn, MD, P.C.
Keith Arthur Brumberger, MD
Houston Brummit, MD
Robert John Bruneau, MD
Hans J Bruns, MD
Annie Louise Brush, MD
Robert Boyce Bryant, MD
Wendell L Bryce, MD
Benjamin F Bryer, MD, P.C.
Ira H Buchalter, MD
Stephen V Buckley, MD
Charles Buckman, MD
Murray Budabin, MD
James L Budny, MD
Damiano Anthony Buffa, MD
William M Bukowski, MD
Isidro Munoz Bulatao, MD
John Bulova, MD
James T Bumbalo, MD
Mary A Burchard, MD
Russell Spencer Burdge, MD
James Paul Burdick, MD
George Joseph Bures, MD
F Vincent Burke, MD
Lillian Patricia Burke, MD
Matthew W Burke, MD
John Butler Burns, MD
Caroline Frances Burpeau, MD
Joseph J Burrascano Jr., MD
B Cullen Burris, MD
James Earle Burris, MD
Paul Nathan Burstein, MD, P.C.
Alan George Burstein, MD
Jacob Burstein, MD
Ruth Cecil Burton, MD
Giacomo J Buscaino, MD
Emile Joseph Buscicchi, MD
Raymond Kimmich Bush, MD
Lawrence T Butler, MD
Benjamin H Button, MD
Lubomyr Bylow, MD
John Chas Byrne, MD
Anthony Caccese, MD
Lawrence M Cadkin, MD
Duane Maynard Cady, MD
Michael Frank Cahn, MD
Michael Calabrese, MD
Joseph Edward Calderone, MD
Elethea H Caldwell, MD
Eliza Hunton Caldwell, MD
Robert Lord Caldwell, MD
William S Calem, MD
Emilio Flores Calica, MD
Lynn R Callin, MD
William C Calloway, MD
Victoria T Camacho, MD
Airlie A C Cameron, MD
Donald John Cameron, MD
M Edwina Campbell, MD
Michael V Canale, MD
Russell P Cancellieri, MD
Orlando Canizares, MD
Daniel Peter Cannucciari, MD
Anthony Joseph Cantore, MD
Anthony Thos Capello, MD
Clive David Caplan, MD
Salvatore Capone, MD
Dominic Cappelleri, MD
Frank F Cappelli, MD
Daniel Raymond Cappon, MD
Jos Danl Cappuccio, MD
Sandra Ann Caputo-Belmont, MD
Otto Anthony Carabba, MD
John M Caracappa, MD
V William Caracci, MD
Tano Salvatore Carbonaro, MD
John Charles Cardona, MD
F Taimour Carey, MD
Robert James Carey, MD
Joseph Carfi, MD
Henry Patrick Carls, MD
Sixto Ramon Caro, MD
Arthur S Caron, MD
Patrick Francis Carone, MD
Robert Carras, MD
Rosario Cortez Carreon, MD
Robert H Carrier, MD
Ethelbert Carrington Jr., MD
Robert E Carroll, MD
John H Carter, MD
Rodman Dana Carter, MD
John J Carthy, MD
Vincent G Caruso, MD
James Louis Carvelas, MD
Lawrence Vito Casamas, MD
Sharen Casazza, MD
Ralph Philip Caschetta, MD
Daniel David Casden, MD
Clarke T Case, MD
David Bartlett Case, MD
George R Caso, MD
Ronald E Cassara, MD
Sam Cassara, MD
Max Cassell, MD
Phillip Rayner Casson, MD
Daniel A Castellani, MD
Frank Xavier Castellano, MD
Thomas Charles Catalano, MD
Anthony S Catinella, MD
George Fitzhugh Catlett, MD
Francesco E Cavallo, MD
Vernal Gordon Cave, MD
Otto Enrique Caveda, MD
Mehmet Cavusoglu, MD
Sol B Cea-Perez, MD
Russell N A Cecil, MD
Arnold Jos Cerasoli, MD
Frank Cerasuolo, MD
Frank A Cerzosimo, MD
Reynold Cesar, MD
Robert A Cestari, MD
John R Cetner, MD
Rabin K Chakraborty, MD
Sobha Chakraborty, MD
Robert G Chaloner, MD
Chi Ho Chang, MD
Mitchell H Charap, MD
Leon I Charash, MD
Richard E Charney, MD
Stanley Marvin Charnoff, MD
Harold H Chaskey, MD
Norman Chassin, MD
Mahavir P Chaturvedi, MD
Meera Chaudhuri, MD
Pasquale Cherico, MD
Ramesh B Cherukuri, MD
Gloria Almira Chia, MD
John Chiappetta, MD
Joseph F Chiaramonte, MD
Barbara Elizabeth Chick, MD
Henry Chin, MD
Yin-Lee Chin, MD
Irving W Chiteman, MD
David Tak Wai Chiu, MD
Kwang Ho Cho, MD
Waled H Chowdhury, MD
Foo Chu, MD
Wyman-Wei Min Chu, MD
Scott R Chudow, MD
Sae Douk Chun, MD
Byung Rak Chung, MD
Lew Edgar Cibeu, MD
Frank Cifarelli, MD
Umbert Cimildoro, MD
Armand Joel Cincotta, MD
Peter Bernard Cinelli, MD
Vincent A Cipollaro, MD
Solomon Ciprut, MD
Peter Joseph Cirigliano, DO
William R Clark Jr., MD
Paul M Clark, MD
Robert Francis Clark, MD
David Lovell Clarke, MD
Hamilton J Clarke, MD
Samuel C E Clayton Jr., MD
William Frank Clayton, MD
Anthony Arnold Clemendor, MD
Edwin I Cleveland, MD, P.C.
Louis Carl Cloutier, MD
Conchita Lim Co, MD
Deborah Jill Coady, MD
Richard James Coburn, MD
Antonio Cocchiarella, MD
William Ashley Coch, MD
Corazon S Puno Cocuzza, MD
David Harry Coene, MD
Arnold Daniel Cohen, MD
Burton Harvey Cohen, MD
Daniel Harold Cohen, MD
Emanuel S Cohen, MD
Herbert I Cohen, MD
Irwin Jerome Cohen, MD
Irwin Louis Cohen, MD
Jeffrey I Cohen, MD
Jerome Craig Cohen, MD
Jules Cohen, MD
Kenneth R Cohen, MD
Lawrence Cohen, MD
Michael Lloyd Cohen, MD
Murray Cohen, MD
Noel Lee Cohen, MD
Richard P Cohen, MD
Ronald B Cohen, MD
Victor Cohen, MD
William Nathan Cohen, MD
Lawrence Jerome Cohn, MD
Steven Lawrence Cohn, MD
William J Cohn, MD
William Vito Colangelo, MD

Frank Alfonso Colella, MD
Morton Coleman, MD
William A Coleman, MD
Jerome S Coles, MD
Robert Sohn Coles, MD
David Carl Coletti, MD
Frank Raphael Collier, MD
John Arthur Collins, MD
Robert Joseph Collins, MD
Neil D Colman, MD
James Kilmartin Condon, MD
Barbara Ann Connor, MD
Carl R Conrad, MD
Michael Angelo Consiglio, MD
Elizabeth F Constantine, MD
Leonard Paul Constantine, MD
Andrew Conti, MD
Robert Walter Conway, MD
Harold Robert Cook, MD
Glen Robert Cooley, MD
Joseph William Cooney, MD
Anthony Jos Cooper, MD
Burton S Cooper, MD
Howard N Cooper, MD
Jonathan Edward Cooper, MD
Leslie Barry Cooperman, MD
Robert L Copeland, MD
Stanford S Copley, MD
Stuart M Copperman, MD
Paschal Anthony Corbo, MD
Robert Paul Cordone, MD
Robert Lewis Corn, MD
Hans Corneille, MD
James P Corsones, MD
Anthony Joseph Cosentino, MD
Anthony Richard Costa, MD
John Louis Costa, MD
Anthony V Costantino, MD
Robert Wayne Cotie, MD
Allan Cott, MD
Robert J Covas, MD
Martin Cowan, MD
Edward Michael Cox, MD
George Robert Cox, MD
Joseph Aloysius Coyle, MD
Thomas F Cozza, MD
Susan M Craig, MD
Irving Cramer, MD
Paul Marion Cramer, MD
Maria Antoinette Crea, MD
William Robert Cromwell, MD
James Theo Crosby, MD
David G Cruickshank, MD
Primitivo T Cruz, MD
Priscilla Lees Cummings, MD
F Mitchell Cummins, MD
John Edmund Cunningham Jr., MD
James F Cunningham, MD
Thomas Joseph Cuomo Jr., MD
Edward Chas Curnen, MD
Frank J Curran, MD
Harold P Curran, MD
Gordon D Currie, MD
Brian Joseph Curtin, MD
Seymour S Cutler, MD
Frank L Cutson, MD
Albert V Cutter, MD
Theresa A Cyzewski-Kutz, MD
Pietro D'Alba, MD
Robert Anthony D'Amico, MD
Vincent Mario D'Amico, MD
Peter Samuel D'Arrigo, MD
Cappy D'Avino, MD
Giovanni D'Emilia, MD
John L Dadey, MD
Arthur N Dadirrian, MD
Andrew Albert Dahl, MD
Thomas Hammond Dailey, MD
Joseph J Daly, MD
Harlan Richard Daman, MD
Wm Damanda, MD
Mircea Damsker, MD
Clifton Louis Dance, MD
Lea Carol Dann, MD
Sidney Dann, MD
Marvin Dannenberg, MD
Louise M Dantuono, MD
Andrew Walter Danyluk, MD
Juan Carlos Dardati, MD
Francis Henry Darragh, MD
Baldev G Das, MD
Binodeswar Das, MD
Marianna M Davidian, MD
Ralph Davidoff, MD
Anthony Lewis Davidson, MD
Robert Victor Davidson, MD
Edward Alfred Davies, MD
Fel Gale Davies, MD
John E Davis, MD
John Stewart Davis, MD
Paul Harvey Davis, MD
Richard F Davis, MD
Selvan Davison, MD
Carl Edward Day, MD
Guido Alfred De Blasio, MD
Vincent H De Caprio, MD
Zira De Fries, MD
Clayton Richard De Haan, MD
C A De La Rocha, MD
Frank V De Laus, MD
Paul Michael De Luca, MD
Anthony G De Martino, MD
Harry M De Pan, MD
Felix De Pinies, MD
Vincent Alan De Rosa, MD
Caesar De Rostaing, MD
Louis Joseph De Russo, MD
Peter N De Sanctis, MD
John Patrick De Simone, MD
Rocco Joseph De Simone, MD
Paolo Emilio De Stefano, MD
Nicholas John De Vito, MD
William Henry De Vries, MD
Dale L Deahn, MD
William Dean Jr., MD
Charles Howard Debrovner, MD
Daniel George Deckler, MD
Douglas Degelsmith, MD
Christine T Del Gaizo, MD
William Victor Delaney Jr., MD
Leo T Delaney, MD
Thomas Brendan Delaney, MD
Felix Antonio Delerme, MD
Joseph I Delimon, MD
David A Della Porta, MD
Michael Robert Delman, MD
Peter S Deluca, MD
Howard Bertram Demb, MD
Michael William Dempsey, MD
Edwin Leopold Demuth, MD
Richard Drury Dent, MD
John Roe Denton, MD
George Dermksian, MD
William Henry Dermody, MD
Andre Desgroseillers, MD
Myles R Desner, MD
Frederic H Deutsch, MD
Oscar Andrew Devera, MD
Anthony D Di Benedetto, MD
Guido J Di Benedetto, MD
Joseph Thomas Di Bianco, MD
Edmund Jos Di Lello, MD
Edmund Di Lello, MD
Dominick J Di Maio, MD
John B Di Matteo, MD
Janis Di Pietro, MD
Joseph A Di Poala, MD
Michael T Di Roberto, MD
Wadih Diab, MD
Frederick Saml Dick, MD
Lionel A Dick, MD
Arthur Hill Diedrick, MD
William C Diefenbach, MD
Gerard J Diesfeld, MD
Theodore Diktaban, MD
Robert E Dinsmore, MD
Marta E Soler Diorio, MD
Frederick W Dischinger, MD
George A Disney, MD
James Anthony Dispenza, MD
Steven Marc Divack, MD
Peter A Doane, MD
Dean Irwin Dobbin, MD
Chauncey H Dobson, MD
John A Dolan, MD
Aaron Dolinsky, MD
Lawrence A Dolkart, MD
David Hyman Dombeck, MD
Josefina Tan Domingo, MD
Rodolfo T Domingo, MD
David Eisen Doniger, MD
Stanley Samuel Donnenberg, MD
Paul Tilson Donohue, MD
H Patrick Dorr, MD
Roland Ernest Doucet, MD
Roy George Stuart Dougall, MD
Sibert Roy Douglas, MD
Leonard R Dourmashkin, MD
Norman J Dovberg, DO
Frederich R Downs, MD
Henry Watkins Doyle Jr., MD
Theodore S Drachman, MD
Rosario Frank Drago, MD
Eric Martin Dreyfuss, MD
Irving Speiser Dribben, MD
Lloyd Richard Dropkin, MD
Barry L Drucker, DO
Marjory Parsons Dube, MD
Richard Dubin, MD
Jerome Dubowy, MD
Charles L Dubuar, MD
Robert A Duca, MD
Simon Duckman, MD
David Joseph Ducore, MD
William D Dugan, MD
Jocelyn A Dummett, MD
Adelmo P Dunghe, MD
Edward A Dunlap, MD
Thomas James Dunne, MD
Joan Catherine Durocher, MD
Murray Dworetzky, MD
Joseph Y Dwoskin, MD
Consan Cu Dy, MD
Ruben Dybner, MD
Thomas S Eagan, MD
Frederick Allyn Eames, MD
Amy P Early, MD
Robert Marshall Easley Jr., MD
Joan Gay Easton, MD
Richard G Eaton, MD
Richard D Eberle, MD
Robert Charles Eberle, MD
Roger Madison Echols, MD
Marianne Horney Eckardt, MD
Kenneth Harry Eckhert Sr., MD
Alan Econ, MD
Eugene Edelman, MD
William Sterling Edgecomb, MD
Jay William Edison, MD
James A Edmond, MD
John Newell Edson, MD
Walter Elmer Eells, MD
Jack Egnatinsky, MD
Irving Danl Ehrenfeld, MD
Theodore Ehrenreich, MD
Paul M Ehrlich, MD
Walter F Ehrlich, MD
Seymour Einhorn, MD
George Eisele, MD
Seth D Eisen, MD
Bernard Eisenberg, MD
David Marvin Eisenberg, MD
James B Eisenkraft, MD
Emilio M Ejercito Jr., MD
Dominic R Elia, MD
Robert Carl Eliot, MD
Arthur H Elkind, MD
David S Ellison, MD
John Bingham Ellison, MD
Jesse S Ellman, MD
Leonard R Elmaleh, MD
Camilo Elsin, MD
James Jerow Elting, MD
Edgar Carpio Elum, MD
Robert Healy Elwell, MD
Russell Stuart Elwell, MD
Leonard John Emma, MD
Robert Walter Emmens, MD
Anthony John Emmi, MD
Joseph Baltus Enders, MD
Jay W Eneman, MD
Young F Eng, MD
Leroy Engel, MD
Lewis Edward Engel, MD
David M Engelhardt, MD
Ralph L Engle Jr., MD
Carl Englebardt, MD
Allen Marshall Epstein, MD
Barry Michael Epstein, MD
Jeanne A Epstein, MD
Joseph A Epstein, MD
Manfred David Epstein, MD
Nancy E Epstein, MD
Enrique Ergas, MD
Henry Ralph Erle, MD
Alfred Richard Ernst, MD
Thomas Joseph Errico, MD
Leo Esbin, MD
Frank M Esemplare, MD
Francisco Aquino Espino, MD
John Wilson Espy, MD
Jacob Esses, MD
Howard Essner, MD
David Milnor Essom, MD
Lourdes G Esteban, MD
Sounder Rajan Eswar, MD
Hungi P Eswara, MD
Irving M Etkind, MD
Bernardo S Evangelista, MD
Henrietta J Eve, MD
Sinfronio N Eway, MD
Stiles Dean Ezell, MD
Aldo Rocco Faga, MD
Daniel J Fahey, MD
John F Faigle Jr., MD
Jere Butler Faison, MD
Stephen A Falk, MD
Martin S Farber, MD
Morton G Farber, MD
Peter Birger Farnsworth, MD
R Linsy Farris, MD
Arthur Eugene Fass, MD
Milton Fastenberg, MD
Vivian Kudgus Fasula, MD
Daniel Faustin, MD
John T Fazekas, MD
Robert A Feeney, MD
Simeon L Feigin, MD
Lawrence Howard Fein, MD
George Feinbaum, MD
Arthur Warren Feinberg, MD
Barry I Feinblatt, MD
Donald B Feinsod, MD
Michael Feinstein, MD
Hector Raul Felbarg, MD
Norbert Felber, MD
William Campbell Felch, MD
Jerome W Feldstein, MD
Joseph Feliccia, MD
Anthony Benito Felice, MD
Elizabeth M Feller, MD
Marvin Feller, MD
James Augustus Feltman, MD
Walter A Fenstermacher, MD
Richard L Fenton, MD
Ignaz Ferber, MD
John Henry Ferger, MD
James William Ferguson, MD
Susan Carol Ferrary, MD
Mark Allen Fialk, MD
Barry E Field, MD
J William Fielding, MD
Vincent George Fietti Jr., MD
Gil Figueroa, MD
Howard Martin Fillit, MD
Bernard David Fine, MD
Mark Fingerhut, MD
Max Fink, MD
John A Finkbeiner, MD
Martin Saml Finkelstein, MD
Howard E Finklestein, MD
Maria L Finley, MD
Richard A Finley, MD
Russell R Fiore, MD
Hossein Firooznia, MD
Carl R Fischer III, MD
Andrew Fischer, MD
Thos Francis Fischer, MD
Daniel Albert Fisher, MD
Yale Lloyd Fisher, MD
Donald R Fishman, MD
Stanley Irving Fishman, MD
James Mark Flanagan, MD
Fredcrick J Flatley, MD
Jerome Harold Flatow, MD
Leslie Flatow, MD
Henry Fleck, MD
Adiel Fleischer, MD
Morton Fleischman, MD
Charles A Fleischmann, MD
Gisela Ebert Fleischmann, MD
Philip Robt Fleishman, MD
David Jonathan Fleiss, MD
Theodore Charles Flemming, MD

Frederick Joseph Fleszar, MD
Edward Fletcher, MD
F Wesner Fleurant, MD
Antonio Flores, MD
Ofelia R Flores, MD
Francis E Florio, MD
Joseph Florio, MD
John Thomas Flynn, MD
Maura J Lynch Flynn, MD
William Francis Flynn, MD
Francis Milton Fodor, MD
William Henry Foege, MD
Sander H Fogel, MD
Edwin Joseph Foley, MD
Stuart Franklin Folick, MD
Miroslaw Alexander Foltyniak, MD
Peter D Fong, MD
Arthur Hastings Foresman, MD
Claude Ellis Forkner Jr., MD
Thomas J Forlenza, MD
Charles Forman, MD
Donald Forman, MD
Giles H Forman, MD
Philip L Forster, MD
Eric Douglas Foster, MD
John Allan Foster, MD
Pearl Delphine Foster, MD
Anthony M Foti, MD
James W Fox, MD
Joshua Larry Fox, MD
Leonard Fox, MD
Maria Martorell Fox, MD
Mark L Fox, MD
Martin Jeffrey Fox, MD
Hugh F Frame, MD
Walter Alfred Franck, MD
Peter Paul Franco, MD
Thomas John Francomano, MD
Donald William Frank, MD
Jerry R Frank, MD
Liesel Christine Frank, MD
Morris Joseph Frank, MD
Arthur Frankel, MD
Marie Nocek Frankel, MD
Herbert L Franklin, MD
Moira Louise Franklin, MD
Max Fratkin, MD
Sidney Freed, MD
Howard Freedman, MD
Melissa M Freeman, MD
Richard John Freeman, MD
Sumner Lester Freeman, MD
Julia L Freitag, MD
Renata Frenkel, MD
Michael Earl Freshman, MD
Alfred Fred Fretz, MD
Leo David Freydberg, MD
Myron A Fribush, MD
Hildegard Friede, MD
Stephen Lester Friedland, MD
Edward Alan Friedman Jr., MD
Abraham H Friedman, MD
Alexander J Friedman, MD
Bernard Lane Friedman, MD
Harold Friedman, MD
Simon H Friedman, MD
Steven Ira Friedman, MD
Paul Friedmann, MD
Charles Carswell Fries, MD
Robert Frisenda, MD
David A Frucht, MD
Howard L Frucht, MD
Bernard Fruchtman, MD
Jacob Frumkin, MD
Seymour Fuchs, MD
Murray Fuhrman, MD
John D Fulco, MD
Curtis Charles Fuller, MD
Robert Fulop, MD
James B Gabriel, MD
Alexander G Gabriels, MD
Jerome Benjamin Gabry, MD
John J Gadomski, MD
Brian Gaffney, MD
Kathleen Agnes Gaffney, MD
Francis A Gagliardi, MD
J Richard Gaintner, MD
Lila Jane Gairns, MD
Bronislaus Jos Galdys, MD
Richard F Gallagher, MD
Myrna S Gallego, MD

Arnold J Gallo, MD
Gerald Charles Gant, MD
Andrew Robert Ganz, MD
Philip J Gara Jr., MD
Vahe Garabedian, MD
Allan Channing Garben, MD
Vincent P Garbitelli, MD
Emilio Garcia, MD
Joseph Anthony Garcia, MD
Milton Melvin Gardner, MD
Priscilla H Garlock, MD
Lowell Garner, MD
John A Garnish, MD
Michael Garofalo Jr., MD
Thomas John Garrett, MD
Irene Garrow, MD
William G Garson, MD
A R Garza-Vale, MD
Luzvimin Garzon-Adriano, MD
Frank Mario Gatti, MD
Robert Paul Gearhart, MD
John Kevin Gebert, MD
Harry Joseph Gegerson, MD
Jesse Gelband, MD
Philip Michael Gelber, MD
Irwin Milton Gelernt, MD
Maxwell L Gelfand, MD
Samuel M Gelfand, MD
Harold Geliebter, MD
Datta Baburao Gengaje, MD
Robert Francis Genovese, MD
Goffredo G Gensini, MD
Louis Alexis Gentile, MD
Ralph Louis Gentile, MD
Michael J George, MD
Lloyd W Georgeson, MD
Manuel Manikis German, MD
Hal Gerstein, MD
Stephen A Gettinger, MD
Warren Chas Gewant, MD
John Lory Ghertner, MD
Radu Ghiulamila, MD
Samir George Ghobrial, MD
Nicholas Giampetruzzi, MD
Alfred Libero Gianfagna, MD
Albert Giannone, MD
Joseph P Giardino, MD
Joseph F Giattini, MD
Donald Robt Gibbs, MD
Edmond Jos Gicewicz, MD
Charles P Giel, MD
Thomas Edward Gift, MD
Jack M Gigante, MD
Jerome Lester Gilbert, MD
Louis Jay Gilbert, MD
Stewart E Gilbert, MD
Jerald Giller, MD
Robert Maynard Giller, MD
A Marvin Gillman, MD
Arthur E Gillman, MD
Harry L Gilmore, MD
Abraham Gilner, MD
Francis James Gilroy, MD
James A Gilroy, MD
Benjamin Joseph Giminaro, MD
Martin Bernard Gingras, MD
Mejer Ginzburg, MD
Phillip Charles Gioia, MD
John Joseph Giordano, MD
Gino Louis Giorgini Jr., MD
Gino Louis Giorgini, MD
Stephen Wm Giorlando, MD
Josephine A Girard, MD
Joel Stewart Gitlin, MD
George Frank Gitlitz, MD
Paul Allan Gitman, MD
Wilma Gladstone, MD
Stephen Charles Gladysz, MD
Cecil Evelyn Glanville, MD
Gerald L Glaser, MD
Robert Glaser, MD
Burt Anthony Glass, MD
Irving Alfred Glass, MD
Walter Martin Glass, MD
Kenneth I Glassberg, MD
Seymour Glasser, MD
George Morton Glassman, MD
Peter G Gleason, MD
J Arthur Gleiner, MD
Jules Glenn, MD
Morton Bernard Glenn, MD

Steven Z Glickel, MD
Franklin S Glickman, MD
Barry S Gloger, MD
Martin Gluck, MD
Seymour M Gluck, MD
Julian Gnoj, MD
Ramon Garcia Gochuico, MD
Mark P Gold, MD
Theodore Gold, MD
Irwin Goldberg, MD
Morris A Goldberg, MD
Richard L Golden, MD
Barry Goldenberg, MD
Morton Goldfarb, MD
Abraham M Goldman, MD
George J Goldman, MD
Louis Goldman, MD
Mitchell Barry Goldman, MD
Lazari Goldmann, MD
Dan Nicu L Goldstein, MD
David Joseph Goldstein, MD
Edward N Goldstein, MD
Jonathan V Goldstein, MD
Marvin Goldstein, MD
Naomi Goldstein, MD
William Zev Goldstein, MD
Sanford E Goldzier II, MD
David Goler, MD
Jack Jason Goliger, MD
Manuel Gonzalez Gonzalez, MD
Raquel Mirta Gonzalez, MD
Neil C Goodman, MD
Seymour Goodstein, MD
Charles B Goodwin, MD
Richard E Goodwin, MD
Emil S Goodyear, MD
Onslow A Gordon III, MD
Bernard Gordon, MD
Jeffrey Harold Gordon, MD
Robert Emmet Gordon, MD
Sandra Lynn Gordon, MD
Lewis Chester Gordonson, MD
Stephen R Gorfine, MD
Ivan Jennery Gotham, MD
Svend Gothgen, MD
Irving I Gottesman, MD
Joseph L Gottesman, MD
Kenneth Steven Gottesman, MD
Helena Gottlieb, MD
Lawrence Arlen Gould, MD
Henry Abraham Gozan, MD
Frederick H Grabo, MD
Eugen Grabscheid, MD
Harold Leon Graff, MD
Louis William Graffeo, MD
Alexander Gralnick, MD
Henry George Grand, MD
Dean Jay Grandin, MD
Edward Joseph Grandt, MD
Carmen Hermena Grange, MD
Carl V Granger, MD
Roderick Alfred Granzen, MD
Ronald A Grecco, MD
Bernard Isadore Green, MD
Frank Murray Green, MD
Glenn J Green, MD
Joseph Mordecai Green, MD
Manuel Green, MD
Richard Michael Green, MD
Richard Wm Green, MD
Robert Pat Green, MD
Stephen Howell Green, MD
Alan Arnold Greenbaum, MD
Bernard Greenberg, MD
Carolyn P Greenberg, MD
Howard Joel Greenberg, MD
Larry J Greenberg, MD
Samuel Alvin Greenberg, MD
William Lester Greenberg, MD
Michael Noel Greenblatt, MD
Arthur S Greene, MD
Robt Bruce Greene, MD
Seth Saml Greenky, MD
Irwin Greenspan, MD
Joel Greenspan, MD
Leon Greenspan, MD
Martin Ely Greenspan, MD
Nathan M Greenstein, MD
Neal Steven Greenstein, MD
Richard Myron Greenwald, MD
Patricia A Greve, MD

Donald Gribetz, MD
Irwin Gribetz, MD
Solomon I Griboff, MD
Aubrey L Griffith, MD
Edward Albert Griggs Jr., MD
John Daniel Grillo, MD
John Paul Grimaldi, MD
Andrew W Grimes, MD
August Herman Groeschel, MD
Elliott George Gross, MD
Joseph Mayer Gross, MD
Lillian Gross, MD
Stanley L Grossman, MD
Scott Bradley Groudine, MD
Anita Grover, MD
Paul Robt Zanis Grube, MD
Ellis Gruber, MD
Paul Grunfeld, MD
Joseph James Guarrera, MD
Frank Robert Gudas, MD
Marina Lopez Guerrero, MD
Carmelo Gullace, MD
Thomas V Gullo, MD
Richard Francis Gullott, MD
John E Gundy, MD
Teresita M Gungon, MD
Necdet Gureli, MD
Erol Osman Gursel, MD
Raul Orlando Gutierrez, MD
Leonard Beryl Gutner, MD
Thomas Joseph Guttuso, MD
Henry Hosein Guy, MD
Ann Lindsey Haag, MD
Ray Allen Haag, MD
M Wilhelmina Haake, MD
Joseph Haber, MD
Jesse Michael Hackell, MD
Makroo Haghbin, MD
Jane Nancy Haher, MD
Enayat Hakim-Elahi, MD
Isaak I Halegoua, MD
Cyrille Rebecca Halkin, MD
Darryl Hartley Hall, MD
Seymour Lionel Halpern, MD
Ronald Halweil, MD
Ronald Solomon Hames, MD
Paul Eric Hammerschlag, MD
Myung Hak Han, MD
John P Handago, MD
Edward Hanin, MD
Andrew Paul Hannes, MD
Robert Joseph Hanss, MD
Nisarul Haque, MD
Aristide Haravon, MD
Joan Marasco Hardenbergh, MD
Michael John Hardies, MD
Mark Adam Hardy, MD
Mavidi K Hariprasad, MD
Robert Bruce Harper, MD
James L Harrington, MD
Alvin Herbert Harris, MD
E Jack Harris, MD
Irwin Lee Harris, MD
Leigh J Harris, MD
Aaron Harrison, MD
Leonard R Harrison, MD
Raymond Harrison, MD
Lawrence David Harter, MD
Alfred A Hartmann Jr., MD
Martin Harwin, MD
Syed Jaffer Hasan, MD
Mahjabeen Hassan, MD
Donald D Hassett, MD
A Richard Hatfield, MD
Raymond A Havrilla, MD
Katherine Ann Hawkins, MD
Graham Granger Hawks, MD
Alden H Hayden, MD
Charles W Hayden, MD
Leon C Haymes, MD
Stanley Mansfield Haymes, MD
Robert Chester Hays, MD
Maurice Maxwell Heath, MD
Hubert Hecht, MD
Alan Louis Heck, MD
Leroy Savin Heck, MD
Jos Francis Hederman, MD
Martin Lee Heilbraun, MD
Robert K Heineman Jr., MD
Thomas H Heineman, MD
Harold C Heintz, MD

Walter T Heldmann, MD
Janet Anne Heller, MD
Marc Elliott Heller, MD
Barry Harvey Hellman, MD
Frederick Helm, MD
Nagib Pierre Helou, MD
Herman Goodman Helpern, MD
Jose Francisco Helu, MD
Robert Charles Hendler, MD
Arthur Digby Hengerer, MD
Wallace Fetter Henion, MD
William A Henion, MD
Frank Reuben Henne, MD
Mary Ingeborg Henrich, MD
Steven Herman, MD
Waldemar F Hermann, MD
Stephen L Hermele, MD
John C Herrman, MD
Alexander Hersh, MD
Solomon George Hershey, MD
Jay Hershkowitz, MD
Ivan Herzog, MD
A Gordon Hewitson, MD
William J Hickey, MD
Thomas Patrick Higgins, MD
Theodore K Himelstein, MD
J Raymond Hinshaw, MD
Theodore Irino Hirokawa, MD
Douglas Franklin Hirsch, MD
Ernest Ludwig Hirsch, MD
Lissa Beth Hirsch, MD
Richard James Hirschman, MD
Martin Hirschowitz, MD
Frederick Shiu Fai Ho, MD
Kenneth Shiu Kee Ho, MD
Frank Charles Hoak, MD
Thomas Charlton Hobbie, MD
John Habib Hobika, MD
Diane Hochlerin, MD
James G Hoehn, MD
Abraham Charles Hoffman, MD
Daniel Paul Hoffman, MD
Saul Hoffman, MD
Louis Hoffner, MD
Jesse Edward Holland, MD
Bernard Hollander, MD
Milton M Hollander, MD
Warren Le Roy Hollis, MD
William C Hollis, MD
Frank F Holmberg Jr., MD
Nicholas Louis Holowach, MD
George Riley Holswade, MD
Milton Holtzman, MD
Donald A Holub, MD
Manoochehr Homayoon, MD, P.C.
Houshang Homayoon, MD
Ha Ryun Hong, MD
Soo Woong Hong, MD
Sunkyung Hong, MD
Alexander Sandor Honig, MD
Kenneth Everett Hoogs, MD
David Randall Hootnick, MD
Arthur G Hopkins, MD
Charles E R Hopkins, MD
Stanley Hoppenfeld, MD
James F Horn, MD
Lawrence Allan Horn, MD
Stephen William Hornyak, MD
Ernest Horowitz, MD
Peter Michael Horvath, MD
Raymond W Houde, MD
Mortimer Housberg, MD
Edgar Minas Housepian, MD
Daniel Bernard Hovey, MD
James Thomas Howard, MD
Lyn J Howard, MD
Romelee A Howard, MD
Harry E Howe, MD
William P Howley, MD
Christopher Dion Hoy, MD
Cher Young Hsu, MD
Helen C Winsor Hubbard, MD
Herbert B Hudnut Jr., MD
Charles J Hudson, MD
Otho C Hudson, MD
Jean Schmidlapp Humes, MD
John Hanley Huntington, MD
James William Hurley, MD
Jacob Hurowitz, MD
Nathaniel John Hurst, MD
Lawrence B Hurwitz, MD
Herbert Anthony Huss, MD
Musheer Hussain, MD
Edwin Peter Hutsal, MD
Ben Mau-Lian Hwang, MD
James Franklin Hyla, MD
Elliot Lewis Hyman, MD
Robert R Hyman, MD
Sonia W Hyman, MD
Yvette K Hyman, MD
Louis Jerry Iandoli, MD
Peter Joseph Iannuzzi, MD
Charles Louis Ihlenfeld, MD
John D Ilario, MD
Mark Max Imberman, MD
Joseph Francis Incavo, MD
Alfred Peter Ingegno, MD
Milton Ingerman, MD
Allan E Inglis, MD
Hollis S Ingraham, MD
Joseph Thomas Ingrassia, MD
Louis Albert Ingrisano, MD
Peter John Inguagiato, MD
David Alan Inkeles, MD
Herbert A Insel, MD
Nathan Ionascu, MD
Carlo A Ippolito, MD
Felix S Ira Jr., MD
Salvatore J Iraci, MD
Jed Hotchkiss Irvine, MD
Hani Amin Ismail, MD
Alexander Itenberg, MD
J Bruce Iuppa, MD
Jay Martin Izes, MD
Richard Izquierdo, MD
Gerard Nicholas Izzo, MD
Kenneth V O Jackman, MD
Hollace Dawne Jackson, MD
Irving Jackson, MD
Abraham Jacobs, MD
Bernard Jacobs, MD
Bernard J Jacobs, MD
Elliot William Jacobs, MD
Arnold Jacobsen, MD
Alan L Jacobson, MD
Ivan Jacobson, MD
Sherwood Jacobson, MD
William Vincent Jacobson, MD
Emma I Jacoby, MD
Ralph Berthold Jacoby, MD
Anthony Jos Jadhon, MD
Richard Glenn Jaeger, MD
Jose E Jaen, MD
Joseph J Jaksy, MD
Herbert Jalens, MD
William A James Jr., MD
Vitold Jankowski, MD
Joseph David Jannuzzi, MD
Henry David Janowitz, MD
Warren Todd Janus, MD
Charles I Jarowski, MD
Jerome Alan Jarvis, MD
John Julius Jasaitis, MD
Jong-Chu Jean, MD
Charles N Jeffries, MD
Bernard Peter Jensen, MD
Gerald J Jerry, MD
Nagalingam Jeyalingam, MD
Devaraj Jeyamitra, MD
Victor G Jimenez-Ponce, MD
Michael A Jimenez, MD
Pawan Kumar Jindal, MD
Lawrence Franklin Jindra, MD
Aida C Tanyag Jocson, MD
Ernesto Jose Jocson, MD
Adela Johnson, MD
Franklin Johnson, MD
Lewis Wm Johnson, MD
William Cecil B Johnston, MD
Sterling Jonas, MD
Aubrey W Jones, MD
Charles William Jones, MD
Glenn Eugene Jones, MD
Thelma Jones, MD
Vann Kinckle Jones, MD
Eric C Jorgensen, MD
Grace Gertrude Jorgensen, MD
Gerald Allen Joselson, MD
Barry Morton Josephson, MD
George F Josephson, MD
Martin M Josephson, MD
Prakash P Joshi, MD
Suresh K Joshi, MD
William David Ju, MD
Joseph Thomas Judge, MD
E A Juliano-Veloso, MD
Edith M Jurka, MD
Norman Bertram Juskowitz, MD
Bernard Kabakow, MD
Hyman Dave Kachalsky, MD
Eugene Howard Kagan, MD
Leonard M Kagan, MD
Miles Kahan, MD
Kenneth Robert Kahn, MD
Steven Kahner, MD
Stephen J Kaiser, MD
Paul Robert Kalkut, MD
George C Kalonaros, MD
Anne Marie Kalosh, MD
Rajanikant D Kamat, MD
Henry Kamenecka, MD
Bernard Kaminetsky, MD
William Floyd Kanar, MD
Paul David Kandel, MD
Stuart N Kandel, MD
Leonard Kane, MD
Rolf Kane, MD
David Myron Kanef, MD
Allan Irwin Kanter, MD
Alan Bernard Kantor, MD
Stratos G Kantounis, MD
Mitchell Lee Kaphan, MD
Eugene Alken Kaplan, MD
Jeffrey Gene Kaplan, MD
Kenneth Charles Kaplan, MD
Munir Kaplan, MD
Philip Kaplan, MD
Robert Gerald Kaplan, MD
Robert Joseph Kaplan, MD
Sanford Allen Kaplan, MD
Paul Elliott Karafiol, MD
Zenon Karatnycky, MD
Harvey D Karkus, MD
Gerald Franz Karnow, MD
Richard H S Karpinski, MD
Arnold Lewis Karsch, MD
Stuart Kase, MD
Manickavelu Kasinathan, MD
William Kass, MD
Aaron Kassoff, MD
Harry Kassop, MD
Elsa Sara Katz, MD
Frederick Harvey Katz, MD
Michael David Katz, MD
Nachum Monroe Katz, MD
Richard Jay Katz, MD
S Sheldon Katz, MD
Seymour Katz, MD
George Alan Kaufman, MD
Harry H Kaufman, MD
Ira H Kaufman, MD
Joseph J Kaufman, MD
Hans Kaunitz, MD
Paul Kay, MD
B Bernard Kaye, MD
Edward A Kearney, MD
Paul Kechijian, MD
Jack E Keiser Jr., MD
Robert Michael Kelleher, MD
Thomas E Kellie, MD
John Patrick Kelly, MD
John Raymond Kelly, MD
Joseph J Kelter, MD
Ronald Daniel Kelton, MD
Norman Kemler, MD
Michael Charles Kenin, MD
Richard John Kenline, MD
John J Kennedy Jr., MD
Robert J Kennedy, MD
Donald Seymour Kent, MD
Gary Curtis Kent, MD
Andrew D Kerr, MD
Benjamin G Kerr, MD
Lawrence P Kerr, MD
Luisa Kerschbaumer, MD
Dennis Mark Kesden, MD
Sherry Horn Kesden, MD
Hesaraghatta B Keshava, MD, P.C.
Howard J Kesseler, MD
P Arjen Keuskamp, MD
Geraldine T Keyes, MD
Rene A Khafif, MD
Mansho Tolaram Khilnani, MD
Joseph N Khoury, MD
Isaac Khutaina, MD
Michael S Kiken, MD
John Joseph P Kilgallen, MD
Edward James Killian, MD
John C Killian, MD
Byung Ki Kim, MD
Dong-Chul Kim, MD
Hyung Rin Kim, MD
Ill Soo Kim, MD
Seong Jun Kim, MD
Youg Jae Kim, MD
Harry Kimmel, MD
Andrew Philip King, MD
Harry King, MD
Richard W King, MD
Warren L King, MD
Marguerite Kingsbury, MD
Philip Michael Kirol, MD
Madhuri Kirpekar, MD
Bernard Kirsch, MD
Marcia A Cleary Kirsch, MD
Milton Kissin, MD
Charles H Kite, MD
Spyros D Kitromilis, MD
Stanley K Klausner, MD
Barnard Kleiger, MD
Daniel Klein, MD
Edmund Klein, MD
Emil F Klein, MD
Mark Joel Klein, MD
Morton Paul Klein, MD
Robert Martin Klein, MD
Steven A Klein, MD
William W Kleinhandler, MD
Martin Stuart Kleinman, MD
Max Kleinmann, MD
Hans J Kleinschmidt, MD
Kenneth V Klementowski, MD
William A Klepack, MD
Percy Klingenstein, MD
Milton Elihu Klinger, MD
Arthur A Knapp, MD
Jodi C Knight, MD
Russell F Knope, MD
Nancy S Knudsen, MD
Angel Oscar Koatz, MD
Lawrence William Koblenz, MD
Stuart Koblenz, MD
Myron Harris Koch, MD
Steven Paul Koenig, MD
Donald R Koerner, MD
Boris Kogan, MD
Robert M Kohn, MD
Maria Ljubica Kolak, MD
Harry Kolson, MD
Salem Kooby, MD
Pang Lay Kooi, MD
Arnold Koopersmith, MD
Alfred W Kopf, MD
Richard Steven Koplin, MD
Richard J Kops, MD
Burton I Korelitz, MD
Daniel Joel Koretz, MD
Gertrude K Kornblau, MD
Harry Kosovsky, MD, P.C.
Vijayasimha Kotha, MD
Robert B Kotty, MD
Meguerdich Kouyoumdjian, MD
Raymond P Koval, MD
Michael O Kovaleff, MD
Harold A Kozinn, MD
Michael Kozower, MD
John Damion Krafchuk, MD
Robert Lawrence Kraft, MD
William F Kraft, MD
Jeffrey Alan Kramer, MD
Mark M Kramer, MD
Oscar J Kranz, MD
Barry S Krasner, MD
Howard Evan Krasner, MD
Hans Kraus, MD
William Floyd Krause, MD
Stephen Mark Krigsman, MD
Milton Krinsky, MD
Benjamin Joshua Kripke, MD
Veeraswamy Krishnaraj, MD
Edith Lind Kristeller, MD
Stephen Kronenberg, MD
Irving G Kroop, MD
Newton Krumdieck, MD

Leo N Kuczmarski, MD
Robert Gordon Kulak, MD
John J Kulcsar, MD
Kevin B Kulick, MD
William Kuncewitch, MD
Dorothy Kunstadt, MD
Jerome Kupchan, MD
George Kurian, MD
Martin Kurman, MD
Stephen Brill Kurtin, MD
C Frederick Kurtz, MD
Lawrence Gerald Kushins, MD
Pauline L Kuyler, MD
Gerta Kwaszewska, MD
James P G Kwee, MD
Harry George La Forge, MD
John M La Mancuso, MD
Anthony L La Marca, MD
Daniel Denis La Roche, MD
Antonio F La Sorte, MD
James Joseph La Vine, MD
Robert Labinson, MD
Samuel Barry Labow, MD
Henriette Lackner, MD
Alberto Lacoius-Petruccelli, MD
Ellis Wayne Lader, MD
Constantine P Ladopoulos, MD
William Laurace Ladue, MD
Vito Pasqual Laglia, MD
Fouad Elias Lajam, MD
Corradino Michael Lalli, MD
Francois Roosevelt Lamarre, MD
Phillips Lambkin, MD
Thomas King Lammert, MD
Kent Butler Lamoureux, MD
Norman J Lampert, MD
Luis A Landeo, MD
Allen M Landers, MD
John Paul Landi, MD
Allison B Landolt, MD
Lewis B Lane, MD
John Adam Lang, MD
Leonard C Lang, MD
Richard Howard Lange, MD
Ira Joseph Langer, MD
Silvester Lango, MD
Daniel A Langone, MD
H Virginia Langworthy, MD
Dattatraya G Lanjewar, MD
Thomas Allen Lansen, MD
Joseph A Lanzone, MD
Douglas Larsen, MD
Charles W Lasky, MD
Jerry Julius Lasser, MD
Michael Gary Lasser, MD
William Edward Lathan, MD
Paul M Latonero, MD
John K Lattimer, MD
Allen S Laub, MD
Gajanan W Laud, MD
Niels Helth Lauersen, MD
Denis Louis Laurencin, MD
Joseph Lauterstein, MD
Joseph Laval, MD
Uri Lavy, MD
David Bruce Lawrence, MD
George J Lawrence, MD
Alexander John Lazar, MD
Nomenida Ang Lazaro, MD
Virginia Martinez Lazaro, MD
George Mitchell Lazarus, MD
Robert Hubbs Leaming, MD
Erwin Lear, MD
Jude Alphonse Leblanc, MD
Pinkas E Lebovits, MD
Mark Allen Lebowitz, MD
Michael Eric Lebowitz, MD
Ezicl Lederman, MD
William Forrest Lee Jr., MD
Ernesto Lee, MD
Manoon Lee, MD
Michael M Lee, MD
Mookeun Lee, MD
Rachel D Lee, MD
Andre Lefebvre, MD
Barry J Lefkowitz, MD
Louis Hirsch Lefkowitz, MD
David E Lehine, MD
Paul C Lehmuller, MD
Arthur Lehrman, MD
Ira W Leibner, MD
Morton M Leibowitz, MD
Sidney Leibowitz, MD
Henry P Leis, MD
Elliot Leiter, MD
John Joseph Len, MD
Maurice Lenarsky, MD
Harold H Lent, MD
Bernard P Leonard, MD
Robert D Leonard, MD
Wesley Edgar Leonard, MD
Anthony James Leone Jr., MD
Marvin Ivan Lepaw, MD
Marshall Lepidus, MD
John J Leppard III, MD
Lawrence Lercher, MD
David Stephen Lerner, MD
Hobart Abraham Lerner, MD
Joseph Lerner, MD
Leo Robert Lese, MD
Eugene V Leslie, MD
Joyce Leslie, MD
Gerson J Lesnick, MD
Eric Lessinger, MD
Louis J Lester, MD
Soon Jack Leung, MD
Anthony Peter Leuzzi, MD
Robert J Lev, MD
Maria E Levada, MD
Ronald Steven Levandusky, MD
Eli Allen Leven, MD
Edgar Allan Levenson, MD
Gerard A Levi, MD
Daniel B Levin, MD
Florence Carol Levin, MD
Howard Alan Levin, MD
Robin Karen Levin, MD
Sophie Polonska Levin, MD
Allen D Levin, MD
Arnold Jan Levine, MD
David B Levine, MD
Donald Jerome Levine, MD
Jerome M Levine, MD
Judith J Levine, MD
Lester V Levine, MD
Melvin S Levine, MD
Sol Levine, MD
Stephen Norman Levine, MD
Louis Levinstim, MD
Kenneth B Levites, MD
Harry Levitt, MD
Martin Maurice Levitz, MD
Arthur Stephen Levy, MD
Erwin Levy, MD
Harold Levy, MD
Leonard Levy, MD
Lewis Alan Levy, MD
Mark Robinson Levy, MD
Matthew Zachary Levy, MD
Robert N Levy, MD
Ronald Allan Levy, MD
Ross Stuart Levy, MD
Fred Harvey Lewis, MD
John Sydney Lewis, MD
Linda Donelle Lewis, MD
Michael Raymond Lewis, MD
William Victor Lewit, MD
Alan Jay Leyton, MD
Luke K Li Calzi, MD
Nicholas C Li Calzi, MD
Cassian Kai-Shun Li, MD
Joseph J Licata, MD
Stephen B Lichtenberg, MD
Stephen Marc Lichter, MD
Abraham Nathan Lieberman, MD
Harvey Michael Lieberman, MD
Theodore Wm Lieberman, MD
Peter S Liebert, MD
William A Liebler, MD
Lawrence M Lieblich, MD
Arlene Swan Lieneck, MD
Alfred Lilienfeld, MD
Byung Woo Lim, MD
Eng Chiong Tan Lim, MD
Esther Pei-Cheng Lim, MD
George Lim, MD
James Joseph Limacher, MD
Min-Chung Lin, MD
Robert G Lindon, MD
William S Ling, MD
Robert L Lipari, MD
Pamela R Lipkin, MD
Jack Irwin Lipman, MD
Marvin M Lipman, MD
William Frederick Lipp, MD
Lawrence Jay Lippert, MD
Abbott Lippman, MD
Paul Chas Lipschutz, MD
Lynne Miriam Liptay, MD
Oscar C Lirio, MD
Arnold Louis Lisio, MD
Leon Littman, MD
Brad Litwak, MD
Manuel M Liu, MD
Leonidas F Livisay, MD
George Livornese, MD
Nicholas Livrieri, MD
Milagros C Liwanag, MD
Felix V Llamido, MD
Oscar Jaime Llugany, MD
Diane Lo Russo, MD
Kenneth Wm Loach, MD
Louis Anthony Lobes, MD
Jon Curtis Lochner, MD
Clyde Ronald Locke, MD
L Maxwell Lockie, MD
James Bernard Loftus, MD
Rodney Dennis Logan, MD
Luke Loh, MD
Peter C Lombardo, MD
Robert Peter Lombardo, MD
Francis J Loperfido, MD
John Jerome Lopes, MD
Jorge A Lopez-Ovejero, MD
John Marion Lore Jr., MD
Elbert Hall Loughran, MD
Herbert Lourie, MD
Harry H Love, MD
Milton Lowenthal, MD
Sung-Nien Lu, MD
Robert Eugene Lubanski, MD
Alfred Lubart, MD
Harry Robert Lubell, MD
John Peter Lubicky, MD
Virginia L Lubkin, MD
Basil Kassian Lucak, MD
Harold Ludman, MD
John Edward Lukaszewicz, MD
Maurice H Luntz, MD
Paul J Luparello, MD
Arnold David Lurie, MD
Heinz L Luschinsky, MD
Denis N Lusignan, MD
Ralph Lusskin, MD
John Pierpont Lyden, MD
Seamus Lynch, MD
Richard Marvin Lynn, MD
John L Lyon, MD
Norman C Lyster Jr., MD
Glenn John Mac Donald, MD
Mary Calder Mac Kay, MD
Paul Frederick Mac Leod, MD
Harry Alton Mac Millan, MD
William Addison Mac Vay, MD
Richard Martin Macarin, MD
Richard J Macchia, MD
James A Macdonald, MD
Sixto Ramos Maceda, MD
Richard J Mackool, MD
Vincent Ignatius Maddi, MD
Raymond A Maddocks, MD
Samuel H Madell, MD
Joseph Charles Maggio, MD
Louis J Maggio, MD
Albert Joseph Maggioli, MD
Rodolfo Ilao Magpantay, MD
Geronimo Sagullo Magsino, MD
Jesse Waring Mahoney, MD
Marvin Mailman, MD
Robert A Mainzer, MD
Monte Malach, MD
John H Malfetano, MD
Krishan Malik, MD
Barry T Malin, MD
Leonard Irving Malis, MD
Robert David Malkin, MD
Giuseppe M Maltese, MD
Harold L Mamelok, MD
Erich R Mamlok, MD
Hugo Walter N Mancini, MD
Jack Marshall Mancus, MD
Stanley M Mandell, MD
Edgar Oliver Mandeville, MD
Zoltan Mandl, MD
Otto Mandler, MD
W Anthony Mandour, MD
William Muir Manger, MD
Thomas Mangiaracina, MD
Delia Mary Manjoney, MD
Seth Oliver Mankes, MD
Robert Stephen Manogue, MD
Sheldon Manspeizer, MD
Augustus Gerard Mantia, MD
Philip Anthony Mantia, MD
Donald Nelson Mantle, MD
Teofilo M Manzanero, MD
Thomas Joseph Manzo, MD
Seymour Manzon, MD
Karl James Marchenese, MD
Robert Alexander Marciniak, MD
Harold Marcus, MD
Robert L Marcus, MD
Tarek Mardam-Bey, MD
Jonathan Mardirossian, MD
Edward Joseph Marine, MD
A W Martin Marino Jr., MD
Joseph Marino, MD
Joseph M Marino, MD
Leonard Joseph Marino, MD
Wm Duncan Marino, MD
Gary Richard Markoff, MD
Alfred M Markowitz, MD
Benjamin Frank Markowitz, MD
Robert Jay Markowitz, MD
Alfred W Marks, MD
Ansel R Marks, MD
Fausto E Marmolejos, MD
Gordon F Marquissee, MD
Vincent Marrone, MD
Richard Steven Marrus, MD
Arthur Charles Marsh, MD
Francis Joseph Martell, MD
Frederick Wm Martens, MD
George T Martin, MD
Josephine Diaz Martin, MD
Rudolph Joseph Martin, MD
Sidney Arthur Martin, MD
Lawrence R Marwill, MD
Gertie F Marx, MD
Robert John Marzziotti, MD
Anthony Robt Mascia, MD
Armond V Mascia, MD
Anthony Joseph Mastellone, MD
Milton Leon Masur, MD
Armando Bernabe Mata, MD
Martin Matalon, MD
John A Matheson, MD
Edwin Matlin, MD
Kenneth Francis Mattucci, MD
Gerald Cyril Matura, MD
Irving Mauer, MD
George G Mauler, MD
Carmine J Mauro, MD
Donald I Mauser, MD
Sanford L Max, MD
Allyn George May, MD
Charles E May, MD
Elliot Mayefsky, MD
Victor Mayer, MD
William D Mayer, MD
Robert A Mayers, MD
Denis G Mazeika, MD
John S Mazella, MD
Alan A Mazurek, MD
Michael Angelo Mazza, MD
Lawrence Mazzarella Jr., MD
Aldo F Mazzarino, MD
Margaret Mc Aloon, MD
Joseph F Mc Caffrey, MD
Philip E Mc Caffrey, MD
Brian K Mc Candless, MD
James Patrick Mc Carron Jr., MD
George Gordon Mc Cauley, MD
Joseph Paul Mc Cormick, MD
Guy C Mc Coy, MD
George Mc Donald, MD
Jack White Mc Elwain, MD
Frederick C Mc Fadden Jr., MD
Gary Jackson Mc Fadden, MD
M Donald Mc Goldrick, MD
John H Mc Govern, MD
James J Mc Guire, MD
John A Mc Gurty, MD
William B Mc Hugh, MD

Stephen J Mc Ilveen, MD
Douglas Denton Mc Kane, MD
Leon Russell Mc Kinney, MD
Sydney L Mc Louth, MD
George Alfred Mc Millan, MD
Gerard M Mc Phee, MD
Robert C Mc Veigh, MD
Allen W Mead, MD
Rosalinda S Medalla, MD
Parmeela Mediratta, MD
Henry Mednick, MD
Magued Ramzi Megalli, MD
Navin C Mehta, MD
Mark Joseph Meiler, MD
William F Meinhardt, MD
Harold Meiselas, MD
Philippine Meister, MD, P.C.
Mario Hugo Melamedoff, MD
Robert B Mellins, MD
Martin Kenneth Melman, MD
Charles Melone Jr., MD
Steven M Meltzer, MD
David Lewis Menchell, MD
Burton L Mendelsohn, MD
David Cyril Mendelson, MD
Myer Deutsch Mendelson, MD
Arthur William Menken, MD
Albert Lewis Menner, MD, P.C.
Mohandas Krishna Menon, MD
Nobert Menuhin, MD
Sheldon Gary Menzin, MD
Leon Merkin, MD
John Carlson Merriam, MD
Bentley Donald Merrim, MD
William Jay Mesibov, MD
Alexander Earl Messer, MD
Giacomo A Messina, MD
John William Messineo, MD
Ascher L Mestel, MD
Alfred Metellus, MD
Albert Meyer, MD
Jeffrey Meyer, MD
Robert Meyer, MD
Bernard Lawrence Meyers, MD
Paraskevas Michaelides, MD
Gerard Michaud, MD
Christopher Bruce Michelsen, MD
Julian Mierlak, MD
Maria Angelica Mieza, MD
Walter I Migdal, MD
Richard Thomas Milazzo Jr., MD
Mario Milazzo, MD
William Joseph Miles, MD
Joseph E Milgram, MD
Ade Thomas Milhorat, MD
Jerold B Millendorf, MD
Alan Barry Miller, MD
Brian Miller, MD
David Eugene Miller, MD
Frank Miller, MD
Jacob J Miller, MD
Leo Richard Miller, MD
Nelson L Miller, MD
Paul J Miller, MD
Richard L Miller, MD
Robert Eugene Miller, MD
Ronald A Miller, MD
Ruth Avers Miller, MD
Seymour A Miller, MD
Sherwood P Miller, MD
Angel Bueno Millora, MD
Roswell Sherman Mills, MD
Robert Vincent Milora, MD
Amelia M Milukas, MD
Joseph Minasi, MD
Eugene R Mindell, MD
Lawrence J Minei, MD
Amos John Minkel Jr., MD
Arthur Minsky, MD
Bernard J Mintz, MD
Steven A Mintzer, MD
Sidney Mishkin, MD
Yousri Youssef Mishriki, MD
Ashalatha Mital, MD
Dean Mitchell, MD
Harilal Mithalal, MD
Hal J Mitnick, MD
Julie Mitnick, MD
Sushil Kumar Mitra, MD
Rainer N Mittl, MD
William Francis Mitty Jr., MD
William Avery Mix, MD
Sha Moallem, MD
Reza A Moazzenzadeh, MD
Douglas H Moffat, MD
Harriette E Mogul, MD
Laura Molho, MD
Seymour J Molinoff, MD
Robert H Molson, MD
Akram A H Momani, MD
Felix Anthony Monaco, MD
Stanley A Mondrow, MD
Julius Ernest Moneagle, MD
Laurence Stephen Monga, MD
Daniel Monroe, MD
Joseph Frank Monte, MD
Judith Monteferrante, MD
Alejandro M Monteiro, MD
Frank A Monteleone, MD
Virgilio A Monteleone, MD
Royal M Montgomery, MD
Shi Hyong Moon, MD
David Benson Moore, MD
Eric Davidson Moore, MD
Norman Slawson Moore, MD
Damyanti G Moorjani, MD
John R Mootz, MD
Francisco Morales, MD
Max Edward Moravec, MD
Marvin S Mordkoff, MD
A Stephen Morel, MD
Kenneth N Morese, MD
F Andrew Morfesis, MD
Zena Lucille Warren Morris, MD
Frederick S Mortati, MD, P.C.
Donald Robt Morton, MD
John Henderson Morton, MD
Giuseppe Moscatelli, MD
Willy G Moscovici, MD
Eugene Moskowitz, MD
Edward Moss, MD
Leo David Moss, MD
Richard Donald Moss, MD
Mostafa Mossafa, MD
Hamid Mouallem, MD
Robert Emmet Mountain, MD
Samuel Movsas, MD
Joseph G Mowrer, MD
Margaret Ann Moxness, MD
Elizabeth Mroziewicz, MD
Joseph C Mucci, MD
Joseph Mucelli, MD
Richard Stuart Muchnick, MD
Nirmala A Mudaliar, MD
Rochana Muenthongchin, MD
Rafeak Muhammad, MD
Somasekhara Rao Mukkamala, MD
Louisa J Mulcare, MD
Robert John Mulcare, MD
William Gordon Mullin, MD
Francis J Mulvihill, MD
William Lewis Munger, MD
Lucille M Munion, MD
Anna J Munster, MD
Joseph Patrick Murray, MD
Thomas Michael Murray, MD
George Charles Muscillo, MD
David Reuben Musher, MD, P.C.
Jules Musinger, MD
Arif Muslim, MD
Tamton Mustapha, MD
Martin B Myles, MD
Robert A Nabatoff, MD
Sayeed Nabi, MD
Henry Mordecai Nachamie, MD
Robert Ian Nacht, MD
Burton Nackenson, MD
Sigmond Harold Nadler, MD
Gurumurthaiah Nagabhushana, MD
Brian Robson Nagy, MD
Robert Morton Naiman, MD
Michael J Napoli, MD
Richard B Narins, MD
Donald W Nash Jr., MD
Antoine Abu Nassar, MD
Jerome D Nataro, MD
Howard Gordon Nathanson, MD
Wm Henry Nauen, MD
Jack Stanley Nauheim, MD
Jagadish D Navare, MD
Sidney Nearenberg, MD
Walter Neglia, MD
Theodore H Neidengard, MD
John Neilson, MD
Joseph B Neiman, MD
Kevin Gerard Nelsen, MD
James Willard Nelson, MD
Mary Van Slyke Nelson, MD
Rosa Lee Nemir, MD
Eduardo V Nepomuceno Jr., MD
Ronald Leon Neschis, MD
Richard J Neudorfer, MD
Jacob Neuman, MD
Joseph Jehuda Neuschatz, MD
Ira Neustadt, MD
Nicholas Neuwirth, MD
George Francis Nevin, MD
David Martin Nevins, MD
Stuart Tobias Nevins, MD
Joseph Newall, MD
Amy Elaine Newburger, MD
E Roger Newman, MD
Herbert Arthur Newman, MD
Joyce Anita Newman, MD
Wanda Newman, MD
Paula Neyman, MD, P.C.
Lou-Fu Ni, MD
Robt John Nicaise, MD
Jack A Nicholson, MD
John Dailey Nicholson, MD
S Harold Nickerson, MD
James N Nicklas, MD
Vincent M Nicolais, MD
Orville F Nielsen, MD
Marvin A Nierenberg, MD
Abdol Hamid Nikkah, MD
Gerald Turtletaub Niles, MD
Martin Boris Nimkoff, MD
John Charles Ninfo, MD
Sriram D Nirgudkar, MD
Wallace Bortle Nixdorf, MD
John Baptist Nobiletti, MD
William J Noble, MD
John A Nolan, MD
Claudio F Norambuena, MD
William Clay Normand, MD
Jay Edward Norris, MD
Herman Judson Norton, MD
James J Norton, MD
Marjorie Spencer Norton, MD
Robert Jacob Norton, MD
Salvatore Louis Noto, MD
Richard L Nottingham, MD, P.C.
Brian E Novick, MD
Nelson Lee Novick, MD
Theodore W Nowicki, MD
William Kirk Nowill, MD
Kenneth Gerald Nudo, MD
Prasuna Nukalapati, MD
Abraham Nussbaum, MD
Irwin Nydick, MD
Mg Nyein, MD
Joseph H O'Connell, MD
William A O'Connell, MD
John E O'Connor, MD
Paul M O'Connor, MD
Robert J O'Connor, MD
Timothy Patrick O'Connor, MD
Donna O'Hare, MD
Edward V O'Hare, MD
Daniel Francis O'Keeffe, MD
James Gregory O'Keeffe, MD
Kathryn T O'Keeffe, MD
Michael R O'Leary, MD
John Barry O'Meara, MD
James Francis X O'Rourke, MD
Stephen A Obstbaum, MD
Pacifico T Ocampo, MD
Benjamin Hong Oen Oey, MD
Edward B Ogden, MD
Roger Martin Olander, MD
Thomas M Older, MD
William Oliver, MD
Ronald Neil Ollstein, MD
Edmunds O Olowosuko, MD
Pedro D Ong, MD
Ruth C Onukwue, MD
David Scott Orentreich, MD
Norman Orentreich, MD
Lazarus A Orkin, MD
Arthur Edward Orlick, MD
Paul Norman Orloff, MD
Paul Ort, MD
Edward Seymour Orzac, MD
Bernard J Oseroff, MD
Paul R Osterdahl, MD
Mortimer Ostow, MD
Lester L Ostrove, MD
Ronald Edward Ostrove, MD
Paul Owusu-Baah, MD
Nicholas Anthony Pace, MD
William Pace, MD
Daniel Richard Pack, MD
Charles James Paddock, MD
Concetto Pafumi, MD
Alphonse F Pagano, MD
David Allison Page, MD
W Jeffery Page, MD
Michael A Paglia, MD
Rafael Lontoc Palacios, MD
Martin Saul Palmer, MD
Milton Alwood Palmer, MD
Paul Richards Palmer, MD
Thomas Palmeri, MD
Anthony J Palumbo, MD
Ellen Pan, MD
Toula G Panay, MD
Mahendra Kumar Pandya, MD
Kenneth Kyae-Won Pang, MD
Nicholas Panin, MD
Jordan Lee Pantel, MD
Martin Edward Panzer, MD, P.C.
Joseph Papa, MD
Emmanuel Pappous, MD
Laxman Kumar Parashar, MD
Barbara A Parey, MD
Barry Paul Pariser, MD
Antonino Parisi, MD
Ralph Felix Parisi, MD
Jaekil Park, MD
Albert Cushman Parker, MD
Barbara Ann Parker, MD
George Warren Parker, MD
Robt Jos Parkes, MD
Anthony P Parrinello, MD
Henry S Partridge, MD
Melissa Susan Pashcow, MD
Hemlata J Patel, MD
Jagdischandra G Patel, MD
Pankaj R Patel, MD
Rohit A Patel, MD
Venkatachala Pathy, MD
Veeresh M Patil, MD
Roger Patnode, MD
James Gildo Patrei, MD
Chester L Patrick Jr., MD
Myron C Patterson, MD
Franklin V Peale, MD
Marilyn Pearl, MD
Alexander W Pearlman, MD
Donald M Pearlman, MD
Hubert S Pearlman, MD
J Louis Pecora, MD
Joseph Samuel Pecora, MD
Samuel P Pejo, MD
Joseph Peluso, MD
Thomas Donald Pemrick, MD
Jose Antonio Pena, MD
Gary M Pepper, MD
Eli Perchuk, MD
Anna Ward Perkins, MD
Jeffrey Perkins, DO
Peter Thos Perkins, MD
Edward Peter Perley, MD
Isidore I Perlmutter, MD
Steven Perlmutter, MD
Victor Perlow, MD
Solomon Perlroth, MD
Abraham P Perlstein, MD
David Arnold Perry, MD
Hope Craig Perry, MD
Mark Stephen Persky, MD
Ergi J Pesiri, MD
Andrew Joseph Peters, MD
G Michael Peters, MD
Ralph E Peters, MD
Alfred E Peterson, MD
Panagiotis Petropoulos, MD
Daniel Starr Pettee, MD
Herbert S Peyser, MD, P.C.
Arnold Morris Phillips, MD
Cecil A Phillips, MD
Robert S Phillips, MD
Dennis J Picano, MD

Paul Rosario Piccione, MD
Allen Arnold Pierce, MD
Charles Worthen Pierce, MD
Richard N Pierson Jr., MD
Yale I Piker, MD
Samuel Pilnik, MD
Donald Jay Pinals, MD
Jerome Pincus, MD
Joseph Pinkas, MD
Richard Francis Pino, MD
Walter Pinsker, MD
Shlomo Piontkowski, MD
Frederic C Pisanelli, MD
Anthony J Pisciotta, MD
Ernest Pisk, MD
M Eugene Pittman, MD
Harry Plaskov, MD
Bruce Howard Platnik, MD
Robert Jonathan Pletman, MD
Lester N Ploss, MD
Nathaniel Plotkin, MD
Roger Peyton Plotkin, MD
Laurence Roger Plumb, MD
Robert Mann Podell, MD
Donald B Polan, MD
Bernard Polatsch, MD
Michael H Polcino, MD
Victor Allen Poleshuck, MD
Mitchell Coleman Pollack, MD
Samuel Pollack, MD
Stephen J Pollack, MD
Virgil Britton Polley, MD
Donald Owen Pollock, MD
James Allen Pollowitz, MD
Virginia E H Pomeranz, MD
Eduardo R Pons Jr., MD
Florian Popa, MD
Leon M Popoff, MD
Sharon D Popper, MD
Robert Frank Porges, MD
John A Porter, MD
Charles Leo Poskanzer, MD
Marvin Posner, MD
Kalmon D Post, MD
Robert C Post, MD
Robert Sorell Potash, MD
Harry Randolph Potter, MD
Gary Irwin Povill, MD
Meera Prabhat, MD
Kenneth Michael Prager, MD
Renato S Prati Jr., MD
Kenneth Lee Pratt, MD
Edward Joseph Prenner, MD
Max Harold Presberg, MD
Saul Lloyd Presberg, MD
Peter I Pressman, MD
Joseph Anthony Prezio, MD
David Alan Price, MD
Cedric Joseph Priebe Jr., MD
Jose Prieto, MD
Marshall Philip Primack, MD
John Curtis Prindle, MD, P.C.
Ronald Edon Prindle, MD, P.C.
John Thompson Prior, MD
Alan Floyd Pritchard, MD, P.C.
Michael R Privitera Jr., MD
Peter M Procario, MD
Waddic Ralph Procci, MD
Anthony F Provenzano, MD
Arnold Stephen Prywes, MD
Bernard S Puglisi, MD
Marie Pulini, MD
John Michael Purcell, MD
Peter Francis Purcell, MD
David M Puro, MD
Thomas Clark Putnam, MD
Stuart H Q Quan, MD
Jacques Marc Quen, MD
Donald O Quest, MD
Walter Paul Quigley, MD
Brian O Quinn, MD
Murray Quinn, MD
Nathan Benami Rabhan, MD
Martin Rabin, MD
Ronald Rabinowitz, MD
Alfio Raciti, MD
Herbert Brahm Radack, MD
Elizabeth Josette Radle, MD
Richard Julian Radna, MD
Rudolf Radna, MD
Franciska Radocsay, MD
Herzl D Ragins, MD
James Joseph Rahal Jr., MD
Anthony M Raia, MD
Deborah Faye Raiken, MD
Harry George Rainey, MD
Greta M Rainsford, MD
Gary Scott Raizes, MD
Daniel A Rakowski, MD
Concetto S Rametta, MD
Gumpeni Rammohan, MD
Fredrico Ramos-Yordan, MD
John Alan Ramsdell, MD
Lawrence M Rand, MD
Govind C K Rao, MD
Perala Sudhakar Rao, MD
Vavilikolanu Vadana Rao, MD
Bruce Gordon Raphael, MD
Irving G Raphael, MD
Sidney Alan Rapp, MD
Yehoshua Rapp, MD
Lawrence Jay Rappaport, MD
Bert W Rappole, MD
Giovanna Rasile, MD
Boonlua Ratanawongsa, MD
Margaret L Rathbun, MD
Harold Ratner, MD
Michael Harvey Ratner, MD
Firooz Ravangard, MD
Lawrence Ravich, MD
William Bryant Rawls, MD
John S Raymond, MD
Paul Melbourne Read, MD
George Gordon Reader, MD
J Thomas Reagan, MD
Paz Tan Recientes, MD
Janardhan B Reddy, MD
Patricia E Redmond, MD
S Frank Redo, MD
Richard Jack Redvanly, MD
Ronald Craig Reese, MD
Ellen Frances Regan, MD
Donald Paul Regula, MD
Jaana Rehnstrom-Blane, MD
Edward J Reich, MD
Stanley Reichman, MD
J Alan Reid, MD
Joseph Griffin Reidel, MD
Marcus Milton Reidenberg, MD
Irving Lester Reif, MD
Robert W Reifenstein, MD
Edward Thomas Reilly, MD
Henry G Reinhardt, MD
Bruce Matthew Reitberg, MD
Russell Elsworth Reitz, MD
Ilan Reizes, MD
Stanley Renner, MD
Elise L. Renning, MD
Rita Silverberg Reuben, MD
Bienvenido L Reyes, MD
Gloria Pangilinan Reyes, MD
Franklin Cooper Reyner, MD
Jonathan Carl Reynhout, MD
J Victor Reyniak, MD
Stephan Robt Rheingold, MD
Woong Jin Rho, MD
Jeffrey Allen Ribner, MD
Joseph J Ricca, MD
Mario Quintus Ricci, MD
Nicholas M Ricciardi, MD
Brian Thomas Rice, MD
Jenifer G C Rich, MD
John Coolidge Richards, MD
Dorothy J Richardson, MD
Benjamin Richman, MD
Howard Richman, MD
Robert M Richman, MD
John J Ricotta, MD
Richard Rieger, MD
Robert A Riehle Jr., MD
Thomas S Riles, MD
Robert P Ringler, MD
Seymour David Ritter, MD
George Leonard Rivara, MD
Melanie Rivenson, MD
Salomon Rivera, MD
John Richard Robb, MD
William F X Robbett, MD
Chester Lewis Robbins, MD
Edwin S Robbins, MD
Guy Frank Robbins, MD
William Clinton Robbins, MD
Joseph Youmans Roberts, MD
Perry Robins, MD
Denise G Robinson, MD
Kenneth Brian Robinson, MD
Thomas Anthony Rocco Jr., MD
Elton Maisel Rock, MD
William George Rocktaschel, MD
Bernard Rodier, MD
John S Rodman, MD
Jose V Rodriguez-Rib, MD
Philip Reuben Roen, MD
Robert Jay Roffman, MD
Hymen Rogachefsky, MD
Peter Rogatz, MD
Lemuel Arsea Rogers Jr., MD
Judah Roher, MD
Michael Rohman, MD
Pedro Rafael Rojas, MD
Renato Hugo Rojas, MD
Francis John Romano, MD
John Francis Romano, MD
Carlos A Romero, MD
Mauro C Romita, MD
John Albert Root, MD
Robert Joseph Rosado, MD
Vincent Sabto Rosati, MD
Gustav Rosbasch, MD
Martin Eugene Rose, MD
Wilbur Stewart Rose, MD
Jonathan B Rosefsky, MD
Susan Rosell, MD
David Lee Rosen, MD
Howard Rosen, MD
Melvin Rosen, MD
Jacob D Rosenbaum, MD
Kenneth Jay Rosenbaum, MD
Siegfried Rosenbaum, MD
Alan Stephen Rosenberg, MD
Coleman H Rosenberg, MD
Henry Rosenberg, MD
Irwin Joseph Rosenberg, MD
Peter Marc Rosenberg, MD
William Bennett Rosenblatt, MD
Charles I Rosenblum, MD
Harvey Sheldon Rosenblum, MD
Michael A Rosenbluth, MD
Hyman Rosenfeld, MD
Louis Judah Rosenfeld, MD
Majer Rosenfeld, MD
Nachman Rosenfeld, MD
Joshua Tripp Rosenfield, MD
Leon Rosenkranz, MD
Joseph Rosenstein, MD
Alan David Rosenthal, MD
Elizabeth Robbins Rosenthal, MD
Morton L Rosenthal, MD
Thomas Claude Rosenthal, MD
Melvin Paul Rosenwasser, MD
Leonard F Rosenzweig, MD
Henry Rosner, MD
Louis M Rosner, MD
Victor Rosner, MD
Avron Herbert Ross, MD
Josephine Rose Ross, MD
Nathaniel Ross, MD
Steven Ira Ross, MD
Kevin Peter Rosteing, MD
Bernard Roswit, MD
Francis B Roth, MD
Kenneth Roth, MD
Laurence George Roth, MD
Samuel J Roth, MD
Alan Edward Rothberg, MD
Melvin Jay Rothberger, MD
Jeffrey Stuart Rothburd, MD
Steven Randy Rothstein, MD
David M Rowland, MD
John W Rowlingson, MD
George Rozsa, MD
Jonathan Ruan, MD
Benj Isaac Rubin, MD
Julius Rubin, MD
Herbert S Rubinowitz, MD
Norman E Rubinstein, MD
Arthur Leon Ruby, MD
Lawrence Ruchman, MD
Gustave T Ruckert, MD
Pauline L Rudnick, MD
John Smith Ruef, MD
Mauro Ledesma Ruffy, MD
Santo Thos Ruggieri, MD
Deborah Geraldine Ruiz, MD
Peter John Runge, MD
Harold N Runsdorf, MD
John Paul Ruppe Sr., MD
John Leslie Rury, MD
Steven Rush, MD
Edwin Robt Ruskin, MD
Daniel Edward Russo, MD
J George Ralph Russo, MD
John Rusu, MD
Howard Irwin Rutman, MD
Norman Ruttner, MD
Charles A Ruvolo, MD
William Donald Ryan Jr., MD
Bernard Joseph Ryan, MD
Francis Paul Ryan, MD
Kyuong Hwan Ryu, MD
Joseph Saal, MD
Hemalatha Sabapathy, MD
Robert A Sable, MD
Ved Parkash Sachdev, MD
Steven H Sacks, MD
Neil Scott Sadick, MD
Saul Sadka, MD
John Stanley Sadowski, MD
Sidney Hugh Saffer, MD
Michael Safran, MD
Maurice J H Safrin, MD
Freny D Sagar, MD
Ronald Sagerman, MD
Emma Lou Sailors, MD
Nathan Emile Saint-Amand, MD
Ernst Saint-Leger, MD
Peter Michael Saitta, MD
Ernest Salanitre, MD
Hugo Salerno, MD
Robert A Salerno, MD
Nambi Salgunan, MD
Saul F Salinas-Izaguirre, MD
Robert S Salisbury, MD
Barry A Salky, MD
Burton F Salpeter, MD
Stephen David Salsburg, MD
Joseph Pepo Saltiel, MD
Maan Singh Saluja, MD
Paul D Salzberg, MD
Iqbal Abdus Samad, MD
Engracio C Samala, MD
Rafaelita B Samala, MD
David Leonard Samostie, MD
Israel Samuelly, MD
Michaela Samuelly, MD
Steven Samuels, MD
Paul Laurence Samuelson, MD
Gerardo Alfredo San Roman, MD
Antonio J Sancetta, MD
Pascual Sanchez-Munoz, MD
Verisimo F Sanchez, MD
William H Sanders, MD
Denise Sanfilippo, MD
Rashmikant P Sangani, MD
Jitendra Motilal Sanghvi, MD
Kamrooz Sanii, MD
Jesus S Santos, MD
Thomas Santucci, MD
William S Saperstein, MD
Joseph Saponaro, MD
M Bruce Sarlin, MD
Charles Alan Sarner, MD
John E Sarno Jr., MD
Irving Arthur Sarot, MD
Robert Sarreck, MD
Jonathan William Sastic, MD
Lois Morgan Sastic, MD
Richard Faust Satriale, MD
Panas Savasatit, MD
Jose Bory Savigne, MD
Jan Myron Savit, MD
Philip Sawyer, MD
Henri Sayegh, MD
Adelaide T Scanlon, MD
Fritz G Schaal, MD
Jerome Arthur Schack, MD
Henry Allen Schaeffer, MD
Lawrence S Schechter, MD
Jonah Walter Schein, MD
Lawrence S Schek, MD
Sanford C Scheman, MD
Howard I Schenker, MD
Samuel Labe Scher, MD
Jane Schick, MD

Alexander Schiff, MD
John Schifferdecker, MD
Nathan Schifrin, MD
Carl Schiller, MD
Edward B Schlesinger, MD
Oscar Schlesinger, MD
Paul J Schlessinger, MD, P.C.
William Shain Schley, MD
Paul Richard Schloerb, MD
Curt Arthur Schmeidler, MD
Frederick Schmerer, MD
Philip Schmidt, MD
A Bernard Schmierer, MD
Herbert Wm Schmugler, MD
Herman Schneck, MD
Max Schneck, MD
Charlotte B Schneider, MD, P.C.
Edward K Schneider, MD
Gerald Paul Schneider, MD
Louis Schneider, MD
Kenneth Schnide, MD
Barton H Schoenfeld, MD
Jack Chas Schoenholtz, MD
William C Schraft Jr., MD
George Schreiber, MD
Klaus Schreiber, MD
Sidney S Schreiber, MD
Robert Miles Schrier, MD
Robert D Schrock, MD, P.C.
Martin Lewis Schulman, MD
Robert H Schulman, MD
Donald H Schultz, MD
Frank Eliot Schumacher, MD
Stephen Samuel Schuster, MD
James Scott Schutz, MD
Marilyn Schwab, MD
Irwin Schwade, MD
Alan Gelber Schwartz, MD
George Schwartz, MD
I Richard Schwartz, MD
Paul C Schwartz, MD
Ralph Schwartz, MD
Ralph M Schwartz, MD
Robert E Schwartz, MD
Stephen Alan Schwartz, MD
Stuart Jay Schwartz, MD
Samuel P Schwartzfarb, MD
Joseph Schwartzman, MD
Karl Schweig, MD, P.C.
Philip Edward Schweitzer, MD
Olga Schweizer, MD
David Schwimmer, MD
Bruce Robert Sckolnick, MD
Aldo Carlo Scorza, MD
William Doughty Scott, MD
Pasquale John Scotti, MD
Philip T Scozzaro, MD
Kendrick A Sears, MD
Peter Dodge Sears, MD
Victoria Conason Sears, MD
Robert Ira Seaver, MD
Alexander Sandor Sebo, MD
Guillermo Masis Seco, MD
Karim M Seddiq, MD
Harry L Segal, MD
Pedro Armando Segarra, MD
Roy Edward Seibel, MD
Robert Stephen Seibt, MD
Noel H Seicol, MD
Morton D Seigel, MD
Jerome Saul Seiler, MD
Robert H Seinfeld, MD
David Haim Sekons, MD
Stuart Gerald Selkin, MD
Robert George Sellig, MD
Richard John Sempowich, MD
Eugene Ira Senal, MD
Howard L Ser, MD
Joseph C Serletti, MD
Homer Marcus Servoss, MD
Leonard V Settembrini, MD
Sally K Severino, MD
Olaf Juell Severud, MD
John Hooton Seward, MD
Shi-Shen Sha, MD
Eugene Shaevich, MD
Mark Steven Shaffer, MD
Bruce Berner Shafiroff, MD
Dhiraj Harilal Shah, MD, P.C.
Bhadra B Shah, MD
Bipin Kantilal Shah, MD
Vasant G Shah, MD
Albert H Shaheen, MD
David John Shaheen, MD
Joseph Ayoub Shahen, MD
Abdol-Hamid Shahinfar, MD
Goda Shri Shailam, MD, P.C.
Vilasini Shanbhag, MD
David Shapiro, MD
Irene Shapiro, MD
Jerome Barry Shapiro, MD
Robert S Shapiro, MD
Timothy Smith Sharlow, MD
Rosewell D Shaw, MD
Wallace M Shaw, MD
William W Shaw, MD
Melvin Dov Shay, MD
Carole Lynne Shear, MD
Richard Kevin Sheehan, MD
John P Sheehy, MD
Thomas Joseph Sheehy, MD
Byron E Sheesley, MD
Fawzia I Shehata, MD
Sara Yampolsky Sheiner, MD
Michael Carl Shende, MD
Sadashiv Sarvotham Shenoy, MD
James Renwick Shepard, MD
Lewis Gordon Shepler, MD
Daniel Allan Sherber, MD
Neal Sherman, DO
Ronald Sherman, MD
Rubin Sherman, MD
Spencer Erwin Sherman, MD
Mark Victor Sherrid, MD
Carl James Sheusi, MD
Melvin Robt Shevach, MD
Nirmala Kata Shevde, MD
J William Shields, MD
Harold Shifrin, MD
Nak Kwang Shim, MD
Young Hi Shin, MD
Arthur George Ship, MD
Charles W Shlimbaum, MD, P.C.
Elbis A Shoales, MD, P.C.
Bernice Gunsberg Shoobe, MD
John Young Short, MD
Walter Harvey Short, MD
Steven Mark Shoum, MD
Anna Eva Shreve-Gergely, MD
Howard Shulman, MD
Tor Adam Shwayder, MD
Benjamin Sia Kian Bing, MD
Edwina Sia-Kho, MD
Bernardo V Sibuma, MD
Arthur Sicular, MD
Sankara A Sidat-Singh, MD
Mark R Siegelheim, MD
Alvin Morton Siegler, MD
Michael Sierp, MD
John M Silberblatt, MD
Benjamin Silberg, MD
Hilton Silver, MD
Richard T Silver, MD
Doris H P Silverberg, MD
Joseph David Silverberg, MD
Barney Baer Silverman, MD
Frank Silverman, MD
Samuel Silverman, MD
Sidney H Silverman, MD
David Nathan Silvers, MD
William Philip Simmonds, MD
Irving Edmund Simmons, MD
John W Simon, MD
Maurice N Simon, MD
William Harris Simon, MD
Jerome Simson, MD
Thomas C Sinatra, MD
Adolf Singer, MD
Morton Singer, MD
Concepcion R Singson, MD
Lalendra Kumar Sinha, MD
Vinod K Sinha, MD
Manfred Sinnreich, MD
Francis Sisca, MD
Benjamin Sischy, MD
Hampson Albert Sisler, MD
Irving E Sisman, MD
Shetra Sivamurthy, MD
Demetrios C Skamas, MD
John Victor Skiff, MD
Herb Skogland Jr., MD
Robt Alex Skrokov, MD
Bertram A Slaff, MD
Edmund Lee Slakter, MD
William Paul Slimbaugh, MD
Michael Slippen, MD
Joseph Slovak, MD
Arthur M Small, MD
Harold Small, MD
Gerald Jay Smallberg, MD
Melvin Agris Smalley, MD
Robert Henry Smart, MD
Malcolm Goff Smilay, MD
Adolph Smith, MD
Arthur Duane Smith, MD
Asa J Smith, MD
Barry Frederick Smith, MD
Charles Aloysius Smith, MD
Clifford Smith, MD
Darline Donalda Smith, MD
David Israel Smith, MD
Hugh William Smith, MD
Jean C Bechtold Smith, MD
Jeanne Helena Smith, MD
Margery Weyrauch Smith, MD
Neal Todd Smith, MD
Philip Howard Smith, MD
Robert Henry Smith, MD
Vincent Edmund Smith, MD
William Henry Smith, MD
Leonard Paul Smolin, MD, P.C.
Alan Gayton Snart, MD
Jeffrey S Sneider, MD
Jon Robert Snyder, MD
Lawrence J Sobocinski, MD
Susan Soboroff, MD
Edward R Sodaro Jr., MD
Hamilton David Soden, MD
Abram Sohmer, MD
Ezri S Sokol, MD
Faustino T Solis, MD
Henry A Solomon, MD
Irving Solomon, MD
Samuel Solomon, MD
Romeo C Solon, MD
Moses Sommer, MD
David H Sonabend, MD
Anant B Soni, MD
Chandra Soni, MD
Harvey Irwin Sonnenblick, MD
Sushila Sood, MD
Michael Soojian, MD
Carlo Angelo Soranno, MD
Moshe Sorek, MD
Anthony Sossi, MD
Thomas I Soule, MD
David Michael Spain, MD
David Dean Speck, MD
David B Spector, MD
Henry Theodore Spector, MD
Louis Harold Spector, MD
Stanley Spellman, MD
Robert Jack Sperber, MD
Edward Sperling, MD
Ralph Charles Sperrazza, MD
Jerry Alvin Spiegel, MD
Anna R Spiegelman, MD
Morris Spielberg, MD
David Spielsinger, MD
Nathaniel Spier, MD
Harry Spiera, MD
Gary Spierer, MD
Maria Spinak, MD, P.C.
George Jerzy Spinak, MD
Clifford L Spingarn, MD
Milton Leon Spinner, MD, P.C.
Lyman J Spire, MD, P.C.
Joan Florence Spitaleri, MD
Jerome Joseph Spitzer, MD
Robert M Spitzer, MD
Samuel Richard Spitzer, MD
Shepard H Splain, DO
Irving Spodek, MD
John Joseph Squadrito, MD
Vincent D Squilla, MD
Seymour Robert Stall, MD
Charles F Stamm, MD
Alfred Lionel Standfast, MD
Sherry F Stanton, MD
David Berkeley Stark, MD
David Callum C Stark, MD
John D States, MD
Tom G Stauffer, MD
Hania W Stawowy, MD
E Thomas Steadman, MD
Maus W Stearns, MD
Sheldon Edward Stearns, MD
George H Stechel, MD
Richard P Stechel, MD
Benjamin Maxwell Stein, MD
Jeffrey A Stein, MD
Meyer Stein, MD
Norman L Stein, MD
Samuel Joseph Stein, MD
Myron E Steinberg, MD
Robert Mark Steinberger, MD
Alfred Steiner, MD
Roger W Steinhardt, MD
Jacob M Steinhart, MD
Robert E Steinkraus, MD
Mallory Stephens, MD
James Lloyd Sterling, MD
Eli Jos Stern, MD
Leon B Stern, MD
Richard Stern, MD
Robert Stern, MD
Norman David Stevens, MD
Dorothy Stevens Steward, MD, P.C.
Donald H Stewart Jr., MD
David Smithson Stewart, MD
Elwin L Stillman, MD
Gerald Francis Stiner, MD
Walter H Stingle, MD
Gerald Stinziano, MD
George Joseph Stivala, MD
Richard L Stivelman, MD
Julius Edward Stolfi, MD
Frederic A Stone, MD
Leo Stone, MD
Maurice Jules Stone, MD
Peter Wm Stone, MD
Samuel Stone, MD
James M Stormont, MD
Norman Lewis Straker, MD
Elton Strauss, MD
Richard Jay Strauss, MD
Samuel David Strauss, MD
Philip Strax, MD
Eugene Streim, MD
John Harold Streit, MD
George Morris Stroll, MD
Samuel Stromberg, MD
Elliot Wilson Strong, MD
James Mark Strosberg, MD
Rodney Warren Stuart, MD
Nancy June Stubbe, MD
Philip Ching-Chang Su, MD
Ester Santos Suarez, MD
Jagat Mohan Subudhi, MD
Rodolfo Hofer Suguitan, MD
Leon Sultan, MD
Philip Chas Suriano, MD
Suphi Surmeli, MD
Ahalya Susarla, MD
Louis Anthony Susca, MD
Benjamin Raymond Susman, MD
C Susskind-Rubin, MD
John David Sussman, MD
Robert B Sussman, MD
Maganlal K Sutaria, MD
Gerald Joseph Sutin, MD
Jodi J Sutton, MD
Joseph David Sutton, MD
Velimir Stanley Svesko, MD
Chavalit Svetvilas, MD
Donald P Swartz, MD
Thomas Roger Sweeney, MD
Frederick Harris Swerdlow, MD
Gary David Swergold, MD
Stanley F Swiatek, MD
Edward A Hafner Swift, MD
Lucy Houghton Swift, MD
Roy Clemmer Swingle, MD
Jogesh Syalee, MD
Emile E Sylvestre, MD
Francis C Symonds, MD
Steven Emil Szebenyi, MD
Alvin J Tabankin, MD
Simeon Taffet, MD
Syeda Tahera, MD
Naseer Ahmad Tahir, MD
Irving Adrian Tainsky, MD

Michael J Tamul, MD
Morris Tanenbaum, MD
Morris T Tanenhaus, MD
Alf Michael Tannenberg, MD
Edward C Tanner, MD
John Stetson Tanner, MD
Albert H Tannin, MD
Kareem E Tannous, MD
Albert Paul Tarasuk, MD
Pamela Tarkington, MD
Muammer Tasbas, MD
Fred Ulysses Tate, MD
John Arthur Taylor Jr., MD
Charles Neil Taylor, MD
L Holland Taylor, MD
Philip Grant Taylor, MD
Joseph C Tedesco, MD
Jamshid M Tehrany, MD
Hal David Teitelbaum, MD
Franklin F Teller, MD
Reno Tempesta, MD
Paul W Temple, MD
Richard Rollin Temple, MD
Wayne Carver Templer, MD
Marvin J Tenenbaum, MD
Jason A Tepper, MD
Nicholas P Teresi, MD
Byron Harvey Terk, MD
Felix Terner, MD
Andrew N Tershakovec, MD
Enrique J Teuscher, MD
Meyer Texon, MD
Merrell E Thallinger, MD
Valerie G Thomas, MD
Joseph Thomas Thornton, MD
H Gregory Thorsell, MD
Joel Alan Thurm, MD
Harold B Thurston, MD
James Gilmore Tifft, MD
Louis H Tiger, MD
Carlos V Timm, MD
Seymour Tindel, MD
Er Yi Ting, MD
Mary D Ridgway Tinker, MD
Kian Ling Tio, MD
Charles Salvatore Tirone, MD
Perry Tirschwell, MD
Ira B Toback, MD
Anthony J Tocco, MD
Victor J Tofany, MD
Allan Harvey Toffler, MD
Allen J Togut, MD
Myra Richman Togut, MD
Richard B Toll, MD
Matthew Donald Tomaiuoli, MD
Douglas P Torre, MD
Jose Francisco Torres, MD
Paul Francis Torrisi, MD
Nestor J Totero, MD
Elia Salem Toueg, MD
Jon Terence Toussaint, MD
Harold M M Tovell, MD
George Joseph Train, MD
Arnold Joseph Trattler, MD
Samuel Carlyle Trattler, MD
Raymond Coleman Traver, MD
Alexander Traykovski, MD
David Treatman, MD
Ruth Kaplan Treiber, MD
Walter A Trenkle, MD
Wolfgang Tretter, MD
Vicente Trezza, MD
Vincent Tricomi, MD
Ajitkumar T Trivikram, MD
Marco Troiano, MD
Julita Trojanas, MD
Stephen Lewis Trokel, MD
George C Trombetta, MD
Nino M Trunfio, MD
Christine Camire Tsakis, MD
Raymond Chi-Ta Tse, MD
Chang-Chyi Tseng, MD
Li Norman Tsui, MD
Paul Tucci, MD
David Theodore Tucker, MD
Stephen Howard Tucker, MD
Marin Tudor, MD
Theodore Tuller, MD
Raymond J Tuoti, MD
Stanley Turecki, MD
Richard D Turin, MD
Herman Turndorf, MD
Daniel Stanley Turner, MD
William J Turner, MD
Robert David Turoff, MD
Arnold Irvin Turtz, MD
Ingeborg Ahrens Tuttle, MD
Joseph C Tutton, MD
Lawrence Henry Tydings, MD
William S Tyler, MD
Cornelius J Tyson, MD
Sam Louis Unterricht, MD
William R Updegraff, MD
Jerome A Urban, MD
Leonel Urcuyo, MD
Luis Enrique Uribe, MD
N D Krishne Urs, MD
Gavril Ursu, MD
Camilo Uy, MD
Charles Joseph Vacanti, MD
Frank P Vaccarino, MD
Leon L Valdez, MD
Frank Natale Valente, MD
Sivachandra Mohan Vallury, MD
Joseph A Valvo, MD
Peter H Van Brunt, MD
Kenneth George Van Dine, MD
Joseph R Van Dyne, MD
Greta Herta Van Fenema, MD
Glenn D Van Gaasbeek, MD
William T Van Huysen, MD
Ian Gerald L Van Praagh, MD
Nicholas Varallo, MD
Manuel Vargas, MD
George Varughese, MD
John J Vasile, MD
Russell S Vaughan, MD
David Wilson Vaules, MD
Dariush Vaziri, MD
Marina Cadiz Vea, MD
David Thomas Vega, MD
Damir Velcek, MD
Konstantin P Velis, MD
Manuel Veloso, MD
Thomas Skelly Velz, MD
Kalpathy V Venkatesan, MD
Michele P Verga, MD
S Veronneau-Troutman, MD
George Emil Verrilli, MD
Frank A Veteran, MD
Charles P Vialotti, MD
Stanley M Vickers, MD
William H Vickers, MD
John O Vieta, MD
Janis W Vieze, MD
Ambrosio B Villacorta, MD
Bernardo Ramos Villajuan, MD
Juan N Villarama Jr., MD
Paul Vilmos, MD
Henry Vinicor, MD
Filippo Vita, MD
Joseph S Vitiello, MD
Dominick Vitolo, MD
Bobb Charney Vladeck, MD
Julius Voehl, MD
George Vogel, MD
Herbert Paul Vogel, MD
John D Voltmann, MD
Laura J Von Doenhoff, MD
William G Von Stein, MD
Yaadbhiroon Vongtama, MD
Charles H Voorhees, MD
John A Vosburgh, MD
Robert D Vosburgh, MD
George Abbott Voss, MD
Themistocles C Vrachnos, MD
Anton A Vreede, MD
Sirkka E Vuornos, MD
Arthur A Wachtel, MD
Blair Tolmie Wachter, MD
Ruth Waelsch, MD
Harold H Wagner, MD
Samuel Wagreich, MD
Henry A Wahn, MD
Charles Lee Wahnon, MD
Paul M Walczak, MD
Arnold Myron Wald, MD
Pamela C Walders, MD
Irma M Mazzarella Waldo, MD
Allan Russell Walker, MD
Richard F Walker, MD
James H Wall, MD
Stanley L Wallace, MD
Wayne Arnold Wallace, MD
John Felix Waller, MD
John Vogel Waller, MD
Arthur J Wallingford Jr., MD
Andrew Arthur Wallman, MD
Walter S Walls, MD
Kevin M Walsh, MD
Raymond Barry Walsh, MD
Robert J Walsh, MD
William J Walsh, MD
Guy M Walters, MD
Jules G Waltner, MD
Max Waltuch, MD
Livia Shang-Yu Wan, MD
Herbert Wanderman, MD
Stanley A Wanlass, MD
Franklin Stewart Ward, MD
George J Ward, MD
Lewis Burt Ward, MD
Richard R P Warner, MD
Joseph Warshaw, MD
Maksymilan Wasserman, MD
Orest M Wasyliw, MD
Jean D Watkeys, MD
Joseph B Watrous, MD
Donald James Watt, MD
Norman Eugene Watt, MD
Hyla S Watters, MD
Lloyd Alfred Watts, MD
Jack Steven Waxler, MD
Ursula Hilde Weber, MD
Joseph A Wechter, MD
G Roger Weeden, MD
John Carnes Weeks, MD
Manfred Weichsel, MD
Ruth Helen Weichsel, MD
Geraldine L Wein, MD
Sidney B Weinberg, MD
Zwi Weinberg, MD
Howard Alan Weiner, MD
Irwin Weiner, MD
David Jack Weinreich, MD
Alan Stephen Weinstein, MD
Gilbert Ernest Weinstein, MD
Joseph Weinstein, MD
Joshua W Weinstein, MD
Herman Louis Weisberg, MD
Eugene Edward Weise, MD
Elwood G Weisenburn, MD
Richard Jay Weishaar, MD
Ahinora P Weiss, MD
Bernard W Weiss, MD
Leonard S Weiss, MD
Paul Eric Weiss, MD
Jay Michael Weissbrot, MD
Ann L Weissman, MD
Charles H Weissman, MD
Harold Weissman, MD
David George Welch, MD
Francis Minot Weld, MD
Mark B Welles, MD
Bernard Wells, MD
Wayne Oliver Wells, MD
Milton Welt, MD
Johannes D Weltin, MD
Barry David Wenglin, MD
Janet Mary Werkmeister, MD
Martin G Wertkin, MD
Gerald Steven Wertlieb, MD
Coenraad Dirk Westermann, MD
Florent Franklin Westfall, MD
Doris L Wethers, MD
Robert F Wettingfeld, MD
Joseph R Whelan, MD
Robert J Whelpley, MD
Abraham G White, MD
James J White, MD
Philip L Whitelaw, MD
Wayne Graham Whitmore, MD
William A Whyland, MD
Robert D Wickham, MD
Walter W Wicks, MD
Philip D Wiedel, MD
George H Wiedeman, MD
Melvin Leo Wiedman, MD
Howard E Wiener, MD
Jeffery Wiersum, MD
Eva M Z Wiesner, MD
Norman S Wikler, MD
Jason La Rue Wiley Jr., MD
William David Wiley, MD
Herbert Jack Wilk, MD
Daniel Charles William, MD
Jesse S Williams, MD
John Joseph Williams, MD
John R Williams, MD
Stanley Willner, MD
Edwin F Wilson, MD
Frederick A Wilson, MD
Richard Marc Wind, MD
George L Wineburgh, MD
William Winner, MD
Jay J Winokur, MD
Abraham Tobias Winter, MD
Michele L Winter, MD
Arnold Wise, MD
Lawrence F Withington, MD
Sidney Wolan, MD
Ann C Cea Wolanske, MD
Herman Leonard Wolf, MD
Irving Wolf, MD
Leonard Maurice Wolf, MD
Morris J Wolf, MD
Paul J Wolfgruber, MD
Claude D Wolgel, MD
Milton I Wolgel, MD
John Archibald Wood, MD
Lawrence Warren Wood, MD
William Graves Woodin, MD
Robert W Woods Jr., MD
Kenneth Joonkyoon Wooh, MD
George Woolf, MD
Paul J Wopperer, MD
Kenneth Neil Wortman, MD
Naum Wortman, MD
Robert S Wren, MD
Ranvir S Yadav, MD
Arnold D Yagoda, MD
Sung H Yang, MD
Wen-Chang Yang, MD
Frank Vincent Yannelli, MD
Lawrence A Yannuzzi, MD
Allen S Yanoff, MD
Pei Sen Yao, MD
Ferdinand D Yates Jr., MD
Robert P Yatto, MD
Lewis D Yecies, MD
David Gwoon Yep, MD
Mehmet Yilmaz, MD
Kiyoon Yoon, MD
Wilfred Yoslow, MD
Henry Young, MD
Marianne Young, MD
Morris Nathan Young, MD
Nicholas Young, MD
Ruperto S Young, MD
Renato C Yu, MD
Charlotte D Yudell, MD
Shyh-Jong Yue, MD
Albert M Yunich, MD
Abraham Yurkofsky, MD
Edward Theodore Zabawa, MD
Paul M Zabetakis, MD
Steven B Zabin, MD
Les A Zakrzewski, MD
George J Zambetti Jr., MD
Alfred Victor Zamm, MD
Manuel D Zane, MD
Miltiades Zaphiropoulos, MD
Eva Zarrow, MD
Irma Zelig, MD
Ronald Lee Zerbe, MD
Sanford S Zevon, MD
Robert E Zickel, MD
Albert Zilkha, MD
Robert H Zimmer, MD
Frederick T Zimmerman, MD
Lester S Zimmerman, MD
Maurice Jacob Zimmerman, MD
Joseph George Zimring, MD
Ernest G Zingapan, MD
Allen Gerald Zippin, MD
Charlotte Marker Zitrin, MD
Joseph A Zizzi, MD
John G Zoll, MD
Lawrence A Zolnik, MD
Richard Mario Zoppa, MD
Ronald Zoppel, MD
Harvey S Zuckerman, MD
Herman Chas Zuckerman, MD
Murray Zung, MD
Jerome Zwanger, MD

BOOKS RECEIVED

ANESTHESIOLOGY

A Calculus of Suffering. Pain, Professionalism, and Anesthesia in Nineteenth-Century America. By Martin S. Pernick. 421 pp, illustrated. New York, NY, Columbia University Press, 1985. $35.00 (hardcover)

Labat's Regional Anesthesia: Techniques and Clinical Applications. Fourth Edition. By John Adriani, MD. 728 pp, illustrated. St. Louis, MO, Warren H. Green, Inc, 1985. $75.00 (hardcover)

CARDIOLOGY

Diagnostic Procedures in Cardiology: A Clinician's Guide. Edited by James V. Warren, MD, and Richard P. Lewis, MD. 494 pp, illustrated. Chicago, IL, Year Book Medical Publishers, 1985. $39.95 (hardcover)

EMERGENCY MEDICINE

Current Emergency Therapy '85. Edited by Richard F. Edlich, MD, and Daniel A. Spyker, MD. 1,150 pp, illustrated. Rockville, MD, Aspen Systems Corp, 1985. $85.00 (hardcover)

INTERNAL MEDICINE

Clinical Pharmacology of Drug Dependence. By Pier Francesco Mannaioni and William W. Clendenin, MD. 282 pp, illustrated. St. Louis, MO, Ishiyaku EuroAmerica, Inc Publishers, 1984. $23.50 (softcover)

Diseases of Blood Vessels. By Orville Horwitz, MD, Peter R. McCombs, MD, Brooke Roberts, MD. 429 pp, illustrated. Philadelphia, PA, Lea & Febiger, 1985. $52.00 (hardcover)

Oxford Handbook of Clinical Medicine. By R. A. Hope and J. M. Longmore. 724 pp, illustrated. New York, NY, Oxford University Press, 1985. $14.95 (paperback)

Update VI. Harrison's Principles of Internal Medicine. New Treatment Modalities. By Robert G. Petersdorf, MD, Raymond D. Adams, MD, Eugene Braunwald, MD, Kurt J. Isselbacher, MD, Joseph B. Martin, MD, Jean D. Wilson, MD. 268 pp, illustrated. New York, NY, McGraw-Hill Book Co. 1985. $35.00 (hardcover)

INTERNATIONAL HEALTH

Canadian Medicine. A Study in Restricted Entry. By Ronald Hamowy. 417 pp. Vancouver, British Columbia, Canada, The Fraser Institute, 1984. $14.95 (paperback)

MISCELLANEOUS

Appleton's Review For FLEX. 1,000 test questions, answers, and explanations! 13 patient management problems! Each and every question conforms to Subject, Content, and Format of the actual examinations! Computerized Practice Test included! By Ira Jeffrey Berg, MD. 408 pp, illustrated. Norwalk, CT, Appleton-Century-Crofts, 1985. $29.95 (paperback)

The Book of Floating: Exploring the Private Sea. By Michael Hutchison. 255 pp, illustrated. New York, NY, William Morrow & Co, 1984. $7.95 (paperback)

Christiaan Barnard's Program For Living With Arthritis. Including the Seven Steps to Pain Management. By Christiaan Barnard, MD. 224 pp, New York, NY, Bantam Books, 1985. $3.95 (paperback)

Frontiers of Research For Human Biologists Next Hundred Years. By Asok Kumar Mukhopadhyay, MD. 22 pp. New Delhi, Duggal Printers, 1985.

Managing Incontinence. Travel Anywhere. Socialize Again. Avoid Accidents. Enjoy Confidence. By Cheryle B. Gartley. 150 pp, illustrated. Ottawa, IL, Jameson Books, 1985. $12.95 (clothcover)

The Parent's Pediatric Companion. By Gilbert Simon, MD, Marcia Cohen. 378 pp, New York, NY, William Morrow & Co, 1985. $17.95 (hardcover)

Remodeling Your Professional Office. By Murray Schwartz, DDS. 220 pp, illustrated. Oradell, NJ, Medical Economics Books, 1985. $26.95 (hardcover)

Respiratory Problems. Volume 5 of the RN Nursing Assessment Series. 216 pp, illustrated. Oradell, NJ, Medical Economics Books, 1985. $12.95 (paperback)

The Rights of Crime Victims. Comprehensive and Up-To-Date. A Basic Guide to Victims' Rights Under Today's Laws. By James Stark and Howard W. Goldstein. 448 pp, New York, NY, Bantam Books, 1985. $4.95 (paperback)

The Rights of Single People. Comprehensive and Up-To-Date. A Basic Guide to the Legal Rights of Unmarried People Under Current Law. By Mitchell Bernard, Ellen Levine, Stefan Presser, and Marianne Stecich. 218 pp, New York, NY, Bantam Books, 1985. $4.95 (paperback)

The Rights of Young People. Completely Revised and Up-To-Date. A Comprehensive Guide to Young People and the Law. By Martin Guggenheim and Alan Sussman. 352 pp. New York, NY, Bantam Books, 1985. $4.95 (paperback)

RX for Addiction. The Dramatic Story of a Doctor's Battle to Save His Practice, His Marriage, His Life. By W. Robert Gehring, MD. 250 pp. Grand Rapids, MI, Zondervan Publishing House, 1985. $12.95 (hardcover)

The Stroke Fact Book. By Conn Foley, MD, and H. F. Pizer. 193 pp, illustrated. New York, NY, Bantam Books, 1985. $3.95 (paperback)

Thirty-Five Years in the Tower. By Solomon Papper, MD. 155 pp. Boston, MA, Little, Brown & Co, 1985. $19.95 (hardcover)

Witness to War. By Charles Clements, MD. 304 pp, illustrated. New York, NY, Bantam Books, 1985. $3.95 (paperback)

Your Right To Government Information. Comprehensive and Up-To-Date. A Basic Guide to Exercising Your Right to Government Information Under Today's Laws. By Christine M. Marwick. 288 pp, New York, NY, Bantam Books, 1985. $4.95 (paperback)

NEPHROLOGY

Renal Pathophysiology. Third Edition. By Alexander Leaf, MD, and Ramzi S. Cotran, MD. 418 pp, illustrated. New York, NY, Oxford University Press, 1985. $18.95 (paperback)

OBSTETRICS AND GYNECOLOGY

Infertility. A Practical Guide for the Physician. Second Edition. Edited by Mary G. Hammond, MD, Luther M. Talbert, MD. 208 pp, illustrated. Oradell, NJ, Medical Economics Co, 1985. $17.50 (paperback)

Management of High-Risk Pregnancy. Edited by John T. Queenan, MD. Second Edition. 752 pp, illustrated. Oradell, NJ, Medical Economics Co, 1985. $49.95 (hardcover)

Te Linde's Operative Gynecology. Sixth Edition. By Richard E. Mattingly, MD, and John D. Thompson, MD. 938 pp, illustrated. Philadelphia, PA, Lippincott Co, 1985 $95.00 (hardcover)

PHARMACOLOGY

Clinical and Social Pharmacology Postmarketing Period. By Jose L. Alloza. 175 pp, illustrated. Aulendorf, FR Germany, Editio Cantor, 1985. $21.75 (softcover)

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Michael M. Abbene, MD, Bethpage. Died April 7, 1985; age 68. He was a 1943 graduate of the State University of New York Downstate Medical Center. Dr Abbene was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the New York State Surgical Society, the American Geriatrics Society, the American Society of Abdominal Surgeons, the American Academy of Occupational Medicine, the Nassau County Medical Society and the Medical Society of the State of New York.

Stanley Earl Alderson, MD, New York. Died March 28, 1985; age 90. He was a 1917 graduate of Albany Medical College of Union University. Dr Alderson was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the Medical Society of the County of Albany and the Medical Society of the State of New York.

G. Stanley Baker, MD, Warsaw. Died March 31, 1985; age 79. Dr Baker was a 1932 alumnus of Harvard Medical School. He was a member of the Medical Society of the County of Wyoming and the Medical Society of the State of New York.

Alfred Kelley Bates, MD, Bradenton, Florida. Died April 6, 1985; age 97. Dr Bates was a 1914 alumnus of Cornell University Medical College. He was a member of the Medical Society of the County of Cayuga and the Medical Society of the State of New York.

Alexei Nicolai Berk, MD, New York. Died March 27, 1985; age 75. He was a 1937 graduate of Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin. Dr Berk was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Obstetrics and Gynecology, and a member of the New York County Medical Society and the Medical Society of the State of New York.

Alice R. Bernheim, MD, Longboat Key, Florida. Died April 19, 1985; age 102. Dr Bernheim was a 1922 alumna of Columbia University College of Physicians and Surgeons. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Maurice Bornstein, MD, Brooklyn. Died November 21, 1984; age 88. He was a 1918 graduate of New York University College of Medicine. Dr Bornstein was a Fellow of the American College of Cardiology, and a member of the New York Cardiological Society, the New York State Society of Internal Medicine, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Ubaldo M. Cennerazzo, MD, New York. Died April 16, 1985; age 71. He was a 1939 graduate of Università degli Studi di Siena, Facoltà di Medicina e Chirurgia. Dr Cennerazzo was a Qualified Fellow of the International College of Surgeons, and a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Jacob Yonny Cohen, MD, New York. Died January 26, 1985; age 79. Dr Cohen was a 1929 alumnus of the State University of New York Downstate Medical Center. He was a Fellow of the American Academy of Family Physicians and a Diplomate of the American Board of Family Practice, and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

John Philip Crosby, MD, Lockport. Died March 23, 1985; age 75. Dr Crosby was a 1936 graduate of the University of Buffalo School of Medicine. He was a Fellow of the American College of Anesthesiologists, and a member of the Academy of Medicine, the Medical Society of the County of Niagara and the Medical Society of the State of New York.

Thomas Patrick Gaines, MD, Bronx. Died March 27, 1985; age 76. He was a 1935 alumnus of the State University of New York Downstate Medical Center. Dr Gaines was a Qualified Fellow of the International College of Surgeons, and a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Gilbert Edward Ganong, MD, Saranac. Died March 15, 1985; age 77. He was a 1932 graduate of New York University College of Medicine. Dr Ganong was a Fellow of the American Academy of Family Physicians, and a member of the Medical Society of the County of Clinton and the Medical Society of the State of New York.

Jerome Glaser, MD, Scottsdale, Arizona. Died April 4, 1985; age 87. He was a 1923 alumnus of Cornell University Medical College. Dr Glaser was a Fellow of the American College of Allergists and a Diplomate of the American Board of Pediatrics. His memberships included the Academy of Medicine, the American Academy of Pediatrics, the American Academy of Allergy, the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Arthur L. Goodrich, MD, Geneva. Died March 20, 1985; age 61. Dr Goodrich was a 1951 alumnus of the University of Rochester School of Medicine and Dentistry. He was a member of the Academy of Medicine, the Ontario County Medical Society and the Medical Society of the State of New York.

In Hwan Kim, MD, Englewood Cliffs, New Jersey. Died April 2, 1985; age 59. Dr Kim was a 1953 alumnus of Seoul National University College of Medicine, Seoul. He was a Diplomate of the American Board of Radiology, and a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

George G. Krauss, MD, Elmhurst. Died March 31, 1985; age 78. Dr Krauss was a 1932 graduate of Medizinische Fakultät der Universität, Wien. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

John J. Lamberti, MD, Bronxville. Died March 11, 1985; age 73. He was a 1938 graduate of St. Louis University School of Medicine. Dr Lamberti was a member of the Academy of Medicine, the American Academy of Family Physicians, the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Martin Langsam, MD, Hallandale, Florida. Died November 10, 1984; age 73. He was a 1935 alumnus of New York University College of Medicine. Dr Langsam was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Arnold A. Migliaccio, MD, Yonkers, Died October 21, 1984; age 48. Dr Migliaccio was a 1963 alumnus of Università degli Studi di Bologna, Facoltà di Medicina e Chirurgia. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Ernst Neumann, MD, Forest Hills. Died May 31, 1984; age 87. Dr Neumann was a 1923 graduate of Medizinische Fakultät der Universität, Wien. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Kings and the Medical Society of the State of New York.

William Alexander Newlands, MD, North Tarrytown. Died April 2, 1985; age 85. He was a 1926 graduate of Queens University Faculty of Medicine. Dr Newlands was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Otolaryngology. He was a member of the Academy of Medicine, the American Academy of Otolaryngology, the American Laryngological, Rhinological and Otological Society, the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Allen Laurence Parlow, MD, Rochester. Died March 20, 1985; age 89. He was a 1923 alumnus of McGill University Faculty of Medicine, Montreal. Dr Parlow was a Diplomate of the American Board of Urology, and a member of the Academy of Medicine, the American Urological Association, the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Howard A. Patterson, MD, Bronxville. Died March 27, 1985; age 83. He was a 1925 graduate of Harvard Medical School. Dr Patterson was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the Academy of Medicine, the New York State Surgical Society, the American Surgical Association, the New York County Medical Society and the Medical Society of the State of New York.

Louis Perrotta, MD, Bronx. Died March 29, 1985; age 86. He was a 1927 alumnus of New York University College of Medicine. Dr Perrotta was a Qualified Fellow of the International College of Surgeons. His memberships included the American Gastro-enterological Association, the International Academy of Proctology, the American Pediatric Society, the Bronx County Medical Society and the Medical Society of the State of New York.

Francis M.J. Pschierer, MD, Kenmore. Died March 20, 1985; age 70. Dr Pschierer was a 1938 alumnus of the University of Buffalo School of Medicine. He was a member of the New York State Dermatological Society, the Medical Society of the County of Erie and the Medical Society of the State of New York.

Abraham Ravich, MD, New York. Died December 1, 1983; age 95. Dr Ravich was a 1912 alumnus of Columbia University College of Physicians and Surgeons. He was a Fellow of the American College of Surgeons, and a member of the American Urological Association, the Medical Society of the County of Kings and the Medical Society of the State of New York.

George Richman, MD, New Hyde Park. Died April 7, 1985; age 69. He was a 1949 alumnus of Université de Paris Faculté de Médecine. Dr Richman was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Hyman Rivkin, MD, Hollywood, Florida. Died December, 1983; age 85. He was a 1921 graduate of the State University of New York Downstate Medical Center. Dr Rivkin was a Diplomate of the American Board of Radiology, and a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

S. Sack, Sr, MD, West Palm Beach, Florida. Died April 15, 1985; age 82. Dr Sack was a 1929 graduate of the State University of New York Downstate Medical Center. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Edward Santora, MD, Miller Place. Died April 8, 1985; age 79. Dr Santora was a 1931 graduate of New York University College of Medicine. He was a Fellow of the American College of Pathologists, a Diplomate of the American Board of Pathology, and a member of the New York State Society of Pathologists, the American Society of Clinical Pathologists, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Milton J. Schwartz, MD, Douglaston. Died March 29, 1985; age 66. He was a 1943 alumnus of the University of Louisville School of Medicine. Dr Schwartz was a Fellow of the American College of Angiology, and a member of the New York Cardiological Society, the American Academy of Allergy, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Paul Skok, MD, Bronxville. Died April 5, 1985; age 54. He was a 1957 graduate of Albany Medical College of Union University. Dr Skok was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Neurological Surgery. His memberships included the Academy of Medicine, the New York Neurological Society, the New York Society of Neurosurgery, the American Association of Neurological Surgeons, the Medical

Society of the County of Westchester and the Medical Society of the State of New York.

Ralph Slater, MD, Bronxville. Died April 17, 1985; age 71. He was a 1938 graduate of New York University College of Medicine. Dr Slater was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the American Psychiatric Association, the Academy of Psychoanalysis, the New York County Medical Society and the Medical Society of the State of New York.

Raymond Dimmick Snyder, MD, Corinth. Died March 24, 1985; age 82. Dr Snyder was a 1927 graduate of Cornell University Medical College. He was a member of the Medical Society of the County of Saratoga and the Medical Society of the State of New York.

Patricia Gill Squillace, MD, Rockville Centre. Died April 17, 1985; age 60. Dr Squillace was a 1951 alumna of the State University of New York Downstate Medical Center. She was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Nicholas Aladar B. Szabo, MD, Huntington. Died April 8, 1985; age 48. Dr Szabo was a 1966 alumnus of the University of Florida College of Medicine. He was a Fellow of the American College of Surgeons, a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Neurological Surgery. His memberships included the New York Neurological Society, the American Neurological Association, the American Association of Neurological Surgeons, the Suffolk County Medical Society and the Medical Society of the State of New York.

Albert Vander Veer, MD, Delmar. Died April 1, 1985; age 83. Dr Vander Veer was a 1929 alumnus of Cornell University Medical College. He was a Fellow of the American College of Surgeons, and a member of the Medical Society of the County of Albany and the Medical Society of the State of New York.

Samuel A. Vogel, MD, Buffalo. Died March 28, 1985; age 85. Dr Vogel was a 1924 graduate of the University of Illinois College of Medicine. He was a Fellow of the American College of Physicians and the American College of Chest Physicians, and a Diplomate of the American Board of Internal Medicine. He was a member of the Academy of Medicine, the Medical Society of the County of Erie and the Medical Society of the State of New York.

Norman L. Woodford, MD, Union Springs. Died April 2, 1985; age 87. He was a 1921 graduate of the State University of New York Upstate Medical Center. Dr Woodford was a member of the Medical Society of the County of Cayuga and the Medical Society of the State of New York.

Herbert Earl Walker, MD, New York. Died March 29, 1985; age 50. He was a 1961 graduate of the Medical College of Alabama. Dr Walker was a Diplomate of the American Board of Psychiatry and Neurology, and a member of the American Public Health Association, the American Society of Clinical Hypnosis, the American Psychiatric Association, the American Fertility Society, the New York County Medical Society and the Medical Society of the State of New York.

Robert West, MD, Chappaqua. Died March 5, 1985; age 68. Dr West was a 1943 graduate of Columbia University College of Physicians and Surgeons. He was a Diplomate of the American Board of Pediatrics, and a member of the American Academy of Pediatrics, the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Felix Widder, MD, North Bellmore. Died April 8, 1985; age 86. He was a 1923 alumnus of Medizinische Fakultät der Universität, Wien. Dr Widder was a member of the New York County Medical Society and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089–1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), an abbreviated title of no greater than 40 spaces for use as a running foot, and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear on the text. When there are six or fewer authors, all should be listed; where there are seven or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. McGuigan JE. Peptic ulcer. In: Petersdorf RG, Adams RD, Braunwald E, Isselbacher KJ, Martin JB, Wilson JD, eds. *Harrison's Principles of Internal Medicine.* New York, McGraw-Hill, 1983, pp 1697–1712.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Alan Blum, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

Classified Advertising

New York State Journal of Medicine

GENERAL INFORMATION

All classified advertisements are payable in advance, except for government agencies and advertising agencies; a 15% commission to recognized advertising agencies.

Frequency: Monthly
Mailing date: 15th of the month
Closing date: 30 days before mailing date

Circulation: 27,000 (mainly physicians & medical libraries)

DISPLAY ADVERTISING

	1 time	*6 time*	*12 time*	*Width × Depth*
1/4-page	$450	$400	$350	3-1/2″ × 4-7/8″
1/6-page	210	190	170	2-1/8″ × 4-7/8″
1/8-page	190	170	150	3-1/2″ × 2-3/8″
1/16-page	95	85	75	3-1/2″ × 1-1/8″
1/18-page	70	60	50	2-1/8″ × 1-1/2″

PARAGRAPH ADVERTISING

The first several words are set in bold face caps.

Rates: $25.00 per insertion for 50 words or less; additional words are 30 cents each. On request, Dept. numbers will be assigned by the Journal for replies to be forwarded to the advertiser. Dept. numbers are 50 cents per insertion. There is no frequency discount for paragraph ads.

Counting Words: Two initials, each abbreviation, figures consisting of 5 numerals or less, are counted as separate words. For replies, your name and address, or telephone number should appear at the end of the ad and be counted in the total number of words.

For further information, please call the Advertising Department at 516-488-6100.

CLASSIFIED ADVERTISING ORDER FORM

New York State Journal of Medicine
Attn: Classified Advertising
420 Lakeville Rd., P.O. Box 5404
Lake Success, N.Y. 11042

Date________________

Name______________________________

Address______________________________

City/State/Zip________________________

Payment enclosed: $__________

Number of Insertions: __________

Assign Dept. Number: __________

Please make check payable to New York State Journal of Medicine.

BACK COPIES NEW YORK STATE JOURNAL OF MEDICINE

For a limited time, back copies may be purchased at the following rates plus postage.

1971-1980 ... $2.00
1981-1983 ... 3.00

Exception: for the special issue of December 1983 which costs $5.00.

Send your order to:
Circulation Dept., New York State Journal of Medicine, 420 Lakeville Road, P.O. Box 5404, Lake Success, NY 11042.

Inquiries: (516) 488-6100, Circulation Dept.

REAL ESTATE FOR SALE OR RENT—CONT'D

MEDICAL OFFICE AVAILABLE in village of Syosset on the north shore of Long Island. 1800 sq. ft., prime location on first floor, for rent with option to buy in co-op ownership. Call (516) 921-6020.

EQUIPMENT

EKG Bendix, portable. Cost $1,500, sell $700. Write William H. Brockman, M.D., 49 Center Street, Ellenville, NY 12428 or call (914) 647-3518.

MISCELLANEOUS

PROFESSIONAL MISCONDUCT ATTORNEYS. WILLIAM L. WOOD, JR., formerly Executive Director of the New York State Office of Professional Discipline and ANTHONY Z. SCHER, formerly Director of Prosecutions, announce the opening of their law office, WOOD & SCHER. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct proceedings, malpractice, licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. WOOD & SCHER, Attorneys at Law, One Chase Road, Scarsdale, New York 10583, Telephone 914-723-3500.

MISCELLANEOUS—CONT'D

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuariat certification and employee statements of participation—partnership agreements and professional incorporation—No insurance required—references upon request—Jack Wachstock, PC, 3000 Marcus Ave., Lake Success, N.Y. 11530 (516) 437-4848.

MEDICAL EDITOR/WRITER: Seeks freelance assignments. Experienced in clinical/socioeconomic subjects, MD manuscripts preparation, article research, presentations, graphics, copyediting, proofreading. Call (212) 861-7280.

ARCHITECTURAL DESIGN AND CONSTRUCTION: Designers, builders of custom professional offices. Consulting services available. Whether you're opening a new office, relocating, remodeling or expanding an existing office in New York City area and adjacent New Jersey counties. Donald Young, Inc. Tel: (718) 948-5191.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate, with no points, fees or charges of any kind. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

DOCTOR, YOU CAN'T BEAT the quality or the price! Holter Monitor Scanning Service. Physician owned, trained and supervised. No contracts to sign. We can arrange for lease/purchase of Holter equipment. New dual channel recorders with test cables $1,550. Now using UP Service for faster turn around time. DCG Interpretation (313) 879-8860.

ST. LUKE'S-ROOSEVELT HOSPITAL CENTER: CONTINUING EDUCATION INSTITUTE. Series of weekly seminars in general medicine for the general medical practitioner. Approved for Category 1 credit and submitted for AAFP approval. Total hours approximately 48–50. Beginning Thursday, September 26, 1985. Complimentary parking available. For information contact: Dr. Kenneth Weinstein, 262 Central Park West, New York, NY 10025. (212) 799-2627.

SEMINARS

Most major ski areas, Club Med. Disney World and other resorts.

Topic: Medical/Legal and Financial Management

Accredited *Fee $175*

Current Concept Seminars, Inc. *(since 1980)*
3301 Johnson St., Hollywood, FL 33021
(800) 428-6069

Safe Deposit.

Most people look for a safe place to deposit their money. A place where they can keep an eye on it.

Well, buying U.S. Savings Bonds is a safe and easy way to a safe deposit. Because Bonds can be replaced if lost, stolen, or destroyed.

Plus, Bonds offer guarantees: a guaranteed way to save, through the Payroll Savings Plan; a guaranteed interest rate; and guaranteed tax benefits. All guaranteed by America.

Add it all up. U.S. Savings Bonds are the safest deposit.

Keep your eye on them *and* watch them grow.

Take stock in America.

When you put part of your savings into U.S. Savings Bonds you're helping to build a brighter future for your country and for yourself.

A public service of this publication and The Advertising Council.

1986 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
New York Hilton, New York City

Maintaining the Quality of Health Care into the 21st Century

FRIDAY, MARCH 7

10:00 a.m. - 1:00 p.m.	Cardiovascular Diseases Gastroenterology/Alcoholism Obstetrics/Gynecology Neurosurgery Physical Medicine & Rehabilitation
2:00 p.m. - 5:00 p.m.	Neurology Pediatrics/Alcoholism Chest Diseases Medical Legal & Workers Comp./Occupational Medicine Medical Liability Mutual Insurance Co. Realistic Uses of Computers in Medical Practice

SATURDAY, MARCH 8

10:00 a.m. - 1:00 p.m.	Ophthalmology (All Day) Internal Medicine (All Day) Hand Surgery/Anesthesiology (All Day) Urology/Dermatology Psychiatry (All Day) Realistic Uses of Computers in Medical Practice
2:00 p.m. - 5:00 p.m.	Ophthalmology Internal Medicine Otolaryngology Hand Surgery/Anesthesiology Psychiatry

SUNDAY, MARCH 9

10:00 a.m. - 1:00 p.m.	Allergy & Immunology General Surgery Women's Medical Society of N.Y./Family Practice Pathology

◆

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis.

◆

Technical Exhibits

Essential reading for every physician

NEW YORK STATE JOURNAL OF MEDICINE

JULY 1985 • Volume 85, Number 7

Table of Contents, page 279

THE WORLD CIGARETTE PANDEMIC

Part II

PRIMUM NON NOCERE?

The July issue of the **New York State Journal of Medicine** is the most comprehensive and disturbing ever published by a medical journal on the subject of smoking.

SEND A COPY TO COLLEAGUES AND MEDICAL STUDENTS in the US and abroad who might not normally receive the Journal. Or order an extra copy for your patients or yourself.

NOTE: the extra press run of The World Cigarette Pandemic Part 1 (December 1983 issue) sold out quickly. The issue has been updated and republished as *The Cigarette Underworld* ($9.95 plus $1.50 postage). Copies may be ordered from Lyle Stuart, Inc., 120 Enterprise Avenue, Secaucus, NJ 07094.

An extra press run has enabled the NEW YORK STATE JOURNAL OF MEDICINE to offer single or bulk copies of its recent issue on the world cigarette pandemic. As the official publication of the Medical Society of the State of New York since 1900, the *Journal* is one of the foremost independent peer-reviewed general medical journals in the country. Providing timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news, the *Journal* makes important reading—and a welcome gift—to physicians and students in the United States or abroad. A free copy of the July issue will be included with each subscription.

Quantity	Per Copy*
1	$7.50
2-9	$7.00
10-24	$6.50
25-49	$6.00
50-99	$5.50
100-149	$5.00
150-500	$4.00

ORDER FORM

Name________________________

Address________________________

City/State/Zip________________________

Please send________copies of July 1985 issue of the Journal.

Enter my subscription to the Journal. ☐ $25.00 U.S. & Canada; ☐ $30.00 Foreign

Total amount enclosed $________ ***Note:** Domestic postage included. Please add New York state & local sales tax where applicable, or sales tax exemption certificate. Foreign postage additional.

Mail to: Circulation Department, **New York State Journal of Medicine**
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

FOR PHYSICIANS AND SURGEONS WITH A PART-TIME EXPOSURE ONLY

Now offering Medical Malpractice Insurance on an ***occurrence basis*** at drastically reduced rates thru Frontier Insurance Co., Monticello, NY.

With the tail on claims made policies, you might lose your shirt.

Call or write for further information and application.

MEDICAL
MQ
QUADRANGLE

Medical Quadrangle, Inc.
P. O. Box 456
318 Bedford Road
Bedford Hills, New York 10507
914 666-0170
212 829-3654
"Specializing in Medical Liability Insurance"

Need A Temporary Physician?
During: vacations, CME's, illness, recruiting.
Want Free Time While You Practice Medicine?
Join CompHealth's Locum Tenens Physician Group.
For further information, call 412-741-3310.

CompHealth A Physician Group
WILSON ROSS, Regional Administrator
114 Centennial Ave., Sewickley, PA 15143

NATIONAL EMERGENCY SERVICES, INC. (N.E.S.)

Directorships, full- and part-time emergency department opportunities are available throughout New York State and the country for primary care physicians with ER experience. Enjoy a variety of practice settings, competitive remuneration, paid malpractice insurance, and flexible scheduling. For information and application materials contact the recruitment department at (516) 365-4010, (Outside N.Y. State 1-800-645-4848) or send C.V. to N.E.S., One Hollow Lane, Suite 304, Lake Success, N.Y. 11042.

Index to Advertisers

Ayerst Laboratories 4A,5A,
Classified Advertising 7A,15A,18A,2
CyCare 1
Future Information Systems Computers . . . .
Healthways 24A,2
Eli Lilly and Company . . . . 2nd Co
Marion Laboratories 9A,1
McGraw Hill Publications 2
Medical Quadrangle 1
Medical World News 1
N.Y.S. Department of Health 2
A. H. Robins 8A,2
Roche Laboratories . . . . 3rd & 4th Co
Stuart Pharmaceuticals 19A,2
Upjohn Company 2
U.S. Army Reserve . . . .
Winthrop Breon 13A,1

Angina comes in many forms...

So does SORBITRATE®

(ISOSORBIDE DINITRATE)

Unsurpassed flexibility in nitrate therapy.

2.5 mg 5 mg 10 mg
Sublingual Tablets

5 mg 10 mg
Chewable Tablets

5 mg 10 mg 20 mg 30 mg 40 mg
Oral "Swallow" Tablets

40 mg
Sustained Action
"Swallow" Tablets

© 1985 ICI AMERICAS INC

See following page for brief summary of prescribing information.

SORBITRATE®
(ISOSORBIDE DINITRATE)

Please consult full prescribing information before use. A summary follows:

INDICATIONS AND USAGE: SORBITRATE (isosorbide dinitrate) is indicated for the treatment and prevention of angina pectoris. All dosage forms of isosorbide dinitrate may be used prophylactically to decrease frequency and severity of anginal attacks and can be expected to decrease the need for sublingual nitroglycerin.

The sublingual and chewable forms of the drug are indicated for acute prophylaxis of angina pectoris when taken a few minutes before situations likely to provoke anginal attacks. Because of a slower onset of effect, the oral forms of isosorbide dinitrate are not indicated for acute prophylaxis

CONTRAINDICATIONS: SORBITRATE is contraindicated in patients who have shown purported hypersensitivity or idiosyncrasy to it or other nitrates or nitrites. Epinephrine and related compounds are ineffective in reversing the severe hypotensive events associated with overdose and are contraindicated in this situation.

WARNINGS: The benefits of SORBITRATE during the early days of an acute myocardial infarction have not been established If one elects to use organic nitrates in early infarction, hemodynamic monitoring and frequent clinical assessment should be used because of the potential deleterious effects of hypotension.

PRECAUTIONS: General: Severe hypotensive response, particularly with upright posture, may occur with even small doses of SORBITRATE The drug should therefore be used with caution in subjects who may have blood volume depletion from diuretic therapy or in subjects who have low systolic blood pressure (eg, below 90 mmHg) Paradoxical bradycardia and increased angina pectoris may accompany nitrate-induced hypotension. Nitrate therapy may aggravate the angina caused by hypertrophic cardiomyopathy.

Marked symptomatic, orthostatic hypotension has been reported when calcium channel blockers and organic nitrates were used in combination. Dose adjustment of either class of agents may be necessary.

Tolerance to this drug and cross-tolerance to other nitrates and nitrites may occur. Tolerance to the vascular and antianginal effects of isosorbide dinitrate or nitroglycerin has been demonstrated in clinical trials, experience through occupational exposure, and in isolated tissue experiments in the laboratory The importance of tolerance to the appropriate use of isosorbide dinitrate in the management of patients with angina pectoris has not been determined However, one clinical trial using treadmill exercise tolerance (as an end point) found an 8-hour duration of action of oral isosorbide dinitrate following the first dose (after a 2-week placebo washout) and only a 2-hour duration of effect of the same dose after 1 week of repetitive dosing at conventional dosing intervals On the other hand, several trials have been able to differentiate isosorbide dinitrate from placebo after 4 weeks of therapy and, in open trials, an effect seems detectable for as long as several months.

Tolerance clearly occurs in industrial workers continuously exposed to nitroglycerin. Moreover, physical dependence also occurs since chest pain, acute myocardial infarction, and even sudden death have occurred during temporary withdrawal of nitroglycerin from the workers In clinical trials in angina patients, there are reports of anginal attacks being more easily provoked and of rebound in the hemodynamic effects soon after nitrate withdrawal The relative importance of these observations to the routine, clinical use of isosorbide dinitrate is not known. However, it seems prudent to gradually withdraw patients from isosorbide dinitrate when the therapy is being terminated, rather than stopping the drug abruptly.

Information for Patients: Headache may occur during initial therapy with SORBITRATE. Headache is usually relieved by the use of standard headache remedies or by lowering the dose and tends to disappear after the first week or two of use.

Drug Interactions: Alcohol may enhance any marked sensitivity to the hypotensive effect of nitrates.

Isosorbide dinitrate acts directly on vascular smooth muscle; therefore, any other agent that depends on vascular smooth muscle as the final common path can be expected to have decreased or increased effect depending on the agent

Carcinogenesis, Mutagenesis, Impairment of Fertility: No long-term studies in animals have been performed to evaluate the carcinogenic potential of this drug. A modified two-litter reproduction study in rats fed isosorbide dinitrate at 25 or 100 mg/kg/day did not reveal any effects on fertility or gestation or any remarkable gross pathology in any parent or offspring fed isosorbide dinitrate as compared with rats fed a basal-controlled diet.

Pregnancy Category C: Isosorbide dinitrate has been shown to cause a dose-related increase in embryotoxicity (increase in mummified pups) in rabbits at oral doses 35 and 150 times the maximum recommended human daily dose There are no adequate and well-controlled studies in pregnant women. SORBITRATE should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when SORBITRATE is administered to a nursing woman.

Pediatric Use: The safety and effectiveness of SORBITRATE in children has not been established

ADVERSE REACTIONS: Adverse reactions, particularly headache and hypotension, are dose-related In clinical trials at various doses, the following have been observed.

Headache is the most common (reported incidence varies widely, apparently being dose-related, with an average occurrence of about 25%) adverse reaction and may be severe and persistent Cutaneous vasodilation with flushing may occur. Transient episodes of dizziness and weakness, as well as other signs of cerebral ischemia associated with postural hypotension, may occasionally develop (the incidence of reported symptomatic hypotension ranges from 2% to 36%). An occasional individual will exhibit marked sensitivity to the hypotensive effects of nitrates and severe responses (nausea, vomiting, weakness, restlessness, pallor, perspiration, and collapse) may occur even with the usual therapeutic dose Drug rash and/or exfoliative dermatitis may occasionally occur Nausea and vomiting appear to be uncommon. Case reports of clinically significant methemoglobinemia are rare at conventional doses of organic nitrates The formation of methemoglobin is dose-related and, in the case of genetic abnormalities of hemoglobin that favor methemoglobin formation, even conventional doses of organic nitrate could produce harmful concentrations of methemoglobin.

DOSAGE AND ADMINISTRATION: For the treatment of angina pectoris, the usual starting dose for sublingual SORBITRATE is 2.5 to 5 mg, for chewable tablets, 5 mg; for oral (swallowed) tablets, 5 to 20 mg, and for controlled-release forms, 40 mg

SORBITRATE should be titrated upward until angina is relieved or side effects limit the dose. In ambulatory patients, the magnitude of the incremental dose increase should be guided by measurements of standing blood pressure

The initial dosage of sublingual or chewable SORBITRATE for prophylactic therapy in angina pectoris patients is generally 5 or 10 mg every 2 to 3 hours. Adequate controlled clinical studies demonstrating the effectiveness of chronic maintenance therapy with these dosage forms have not been reported

SORBITRATE in oral doses of 10 to 40 mg given every 6 hours or in oral controlled-release doses of 40 to 80 mg given every 8 to 12 hours is generally recommended. The extent to which development of tolerance should modify the dosage program has not been defined *The oral controlled-release forms of isosorbide dinitrate should not be chewed*

DOSAGE FORMS AVAILABLE: Sublingual Tablets (2.5, 5, 10 mg), Chewable Tablets (5, 10 mg); Oral Tablets (5, 10, 20, 30, 40 mg); Sustained Action Tablets (40 mg)

STUART PHARMACEUTICALS
Division of ICI Americas Inc.
Wilmington, DE 19897

STR-2282

Oct 31–Nov 1. **Genetics in Clinical Oncology.** 20½ Cat 1 credits. Info: R. S. K. Chaganti, PhD, Laboratory of Genetics, Memorial Hospital for Cancer and Allied Diseases, 1275 York Ave, New York, NY 10021. Tel: (212) 794-6754.

BROOKLYN

Oct 15. **Stress and the Geriatric Patient: Assessment and Therapeutic Intervention.** 7 Cat 1 credits. Info: Morris Kleinfeld, MD, Director of the Department of Long-Term Care Medicine, 585 Schenectady Ave, Brooklyn, NY 11203. Tel: (718) 604-5297.

Oct 8. **Toxicity of Local Anesthetic Agents.** *Oct 9.* **Regional Anesthesia for Out-patient Surgery.** Info: Department of Anesthesiology, CME, SUNY Downstate Medical Center, Box 6, 450 Clarkson Ave, Brooklyn, NY 11203. Tel: (718) 270-1934.

Oct 15. **Neurology Update.** 8 Cat 1 credits. Info: Dr. Walter R. Slade, Jr. Chief, Neurology Service, VA Medical Center, 800 Poly Place, Brooklyn, NY 11209. Tel: (718) 836-6600, ext 562.

NASSAU

Oct 30. **A Clinical Day in Ophthalmology: Pediatric Topics.** 7 Cat 1 credits. Garden City Hotel. Info: Ann J. Boehme, CME, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

ALBANY

Oct 10–13. **Sixth Annual Critical Care Symposium.** 22½ Cat 1 credits. Info: Ellis Hospital Critical Care Center, 1101 Nott St, Schenectady, NY 12308. Tel: (518) 382-4135.

MONROE

Oct 3–5. **Psychosocial, Psychiatric, and Behavioral Strategies in Health Care: An Interdisciplinary Conference.** Rochester. Info: Karen Sanders, University of Rochester Medical Center, 300 Crittenden Blvd, Rochester, NY 14642. Tel: (716) 275-3617.

ERIE

Oct 11–12. **Pathophysiology and Treatment of Cystic Fibrosis.** Niagara Falls. Info: Rayna Saville, Pediatric CME, Children's Hospital of Buffalo, 219 Bryant St, Buffalo, NY 14222. Tel: (716) 878-7630.

WESTCHESTER

Oct 16. **Ninth Annual Day in Cardiology.** 6 Cat 1 credits. Info: Toni Calabrese Boelsen, White Plains Hospital Medical Center, Davis Avenue, White Plains, NY 10601. Tel: (914) 681-1118.

(*Continued on p 21A*)

AROUND THE NATION

CALIFORNIA

Oct 4–5. **Legal Issues in Medical Practice, 1985.** 9 Cat 1 credits. Info: Extended Programs in Medical Education, University of California School of Medicine, Room 569-U, San Francisco, CA 94143. Tel: (415) 666-4251.

Oct 10–12. **Clinical Reconstructive Upper Extremity Microsurgery.** San Francisco. 16½ Cat 1 credits. Info: American Society for Surgery of the Hand, 3025 South Parker Road, Suite 65, Aurora, CO 80014. Tel: (303) 755-4588.

Oct 19–20. **Head and Neck Radiology: Advanced Techniques in Diagnostic Imaging.** Hyatt Regency Hotel. *Oct. 19–20.* **Magnetic Resonance Imaging for Technologists.** Hyatt Regency Hotel. *Oct 21–25.* **Clinical Magnetic Resonance Imaging.** Hyatt Regency Hotel. Info: Department of Radiology, University of California School of Medicine, San Francisco, CA 94143.

FLORIDA

Oct 14–18. **Eleventh Annual PanAmerican Seminar**—totally in Spanish for Spanish-speaking physicians. 29 Cat 1 credits. *Oct 31–Nov 1.* **Clinical Applications for Pulsed, Continuous and Color Flow Doppler Echocardiography.** 5 Cat 1 credits. Info: Mount Sinai Medical Center, 4300 Alton Rd, Miami Beach, FL 33140. Tel: (305) 674-2311.

ILLINOIS

Oct 13–18. **American College of Surgeons 71st Annual Clinical Congress.** Info: American College of Surgeons, 55 East Erie St, Chicago, IL 60611. Tel: (312) 664-4050.

Oct 24–26. **Stress, Impairment, and the Resident.** Lake Bluff. 8 Cat 1 credits. Info: The University of Illinois at Chicago, Conference and Institutes, 912 South Wood St, 2 North, Chicago, IL 60612. Tel: (312) 996-8025.

INDIANA

Oct 17–20. **Fourth National Seminar on Community Cancer Care.** Indianapolis. Info: Office of CME, Methodist Hospital of Indiana, Inc, 1604 North Capitol Ave, Indianapolis, IN 46202. Tel: (317) 929-3733.

KANSAS

Oct 9–12. **Group Psychotherapy.** 21 Cat 1 credits. Info: Brenda Vink, CME, The Menninger Foundation, Box 829, Topeka, KS 66601. Tel: (913) 273-7500, ext 5992.

MARYLAND

Oct 3–5. **Eleventh Annual Topics in Gastroenterology and Liver Disease.** Baltimore. Info: Jeanne Ryan, Office of Continuing Education, The Johns Hopkins University School of Medicine, 720 Rutland Ave, Baltimore, MD 21205.

MASSACHUSETTS

Oct 17–18. **Medical Emergencies.** 12½ Cat 1 credits. Cambridge. Info: Department of Continuing Medical Education, Boston University School of Medicine, 80 E. Concord St, Boston, MA 02118. Tel: (617) 424-5429.

Oct 21–25. **Fundamentals of Industrial Hygiene.** Oct 27–Nov 8. Program for Health Systems Management. Info: Office of Continuing Education, Harvard School of Public Health, 677 Huntington Ave, Boston, MA 02115. Tel: (617) 732-1171.

Oct 28–Nov 1. **Thirteenth Annual Postgraduate Course in Clinical Pharmacology, Drug Development, and Regulation.** Info: Kristine Niven, Center for the Study of Drug Development, Tufts University, 136 Harrison Ave, Boston, MA 02111. Tel: (617) 956-0070.

TEXAS

Oct 25–27. **Diagnostic Radiology Update.** 18 Cat 1 credits. Dallas. Info: Dolly Christensen, University of Texas Health Science Center, 5323 Harry Hines Blvd, Dallas, TX 75235. Tel: (214) 688-2502.

VIRGINIA

Oct 25–27. **First Annual Review Course in Reproductive Endocrinology and Infertility.** 20 Cat 1 credits. Williamsburg Hilton. Info: Eastern Virginia Medical School, CME, PO Box 1980, Norfolk, Va 23501.

AROUND THE WORLD

ISRAEL

Oct 17–27. **Hematology-Oncology for the Practicing Physician.** Tel Aviv and Jerusalem. Info: Ann J. Boehme, CME, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

Associate Commissioner for New York City Affairs

The New York Department of Health has an excellent opportunity in its New York City Regional Office, 8 East 40th Street, for a MD licensed to practice in New York State to act as representative of the Commissioner of Health on all matters affecting public health in New York City.

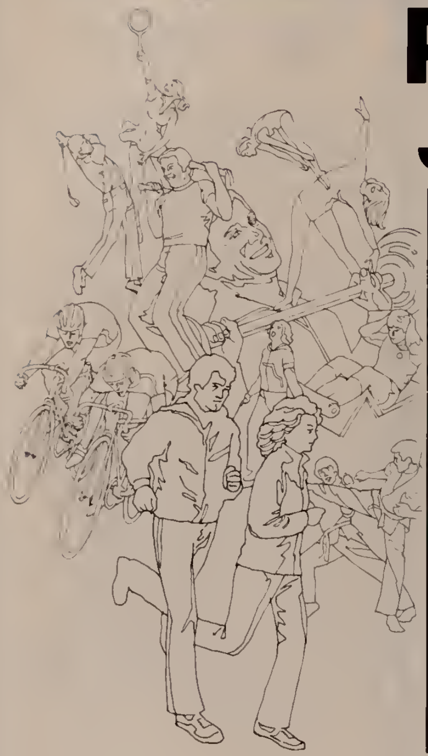
Under the supervision of the Director of the Office of Public Health, you will maintain effective relationships with governmental and health officials in New York City, as well as assist them in the identification and resolution of public health problems. You will also direct the administration of all public health programs carried out by the Department of Health in New York City.

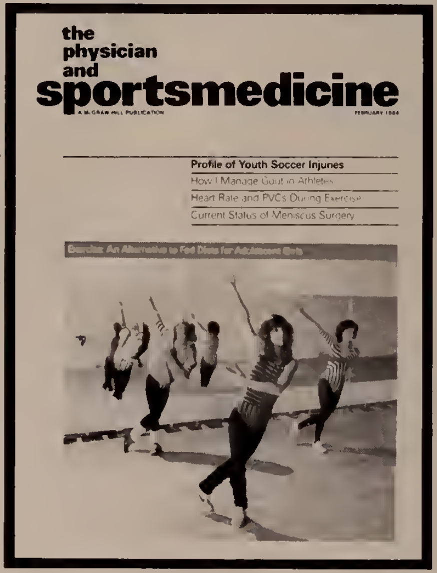
Board eligibility or certification in a medical specialty is required. We would prefer to hear from candidates with experience in any of the following areas: management of health delivery and monitoring systems; communications and consultation with local health officials on a broad range of public health issues; interpretation and enforcement of federal and/or state health policies and requirements at a regional or local level. A Master's degree in Public Health is desirable.

This permanent appointment in the exempt class offers an annual salary of $62,336 per year. Interested and qualified candidates should send their resume by October 25, 1985 to: Human Resources Management Group, ACNYC/RJA1, Room 632, Corning Tower Building, Empire State Plaza, Albany, NY 12237. An equal opportunity/affirmative action employer.

N.Y.S. Department of Health

OVER 66,000 FAMILY PHYSICIANS READ THIS JOURNAL




the physician and sportsmedicine

A McGRAW-HILL PUBLICATION

FEBRUARY 1984

Profile of Youth Soccer Injuries

How I Manage Gout in Athletes

Heart Rate and PVCs During Exercise

Current Status of Meniscus Surgery

Practical information on the medical aspects of fitness and exercise.

Tennis elbow: Joint resolution by conservative treatment.

Hypertrophic cardiomyopathy and the athlete.

Effects of sunscreen use during exercise in the heat.

Overuse injuries to the knee in runners.

How I manage ingrown toenails.

McGraw-Hill

Medical Society of the State of New York

ANNUAL CME ASSEMBLY

MARCH 7-9, 1986

The New York Hilton, New York City

Combined With The Annual Meeting of

The House of Delegates

MARCH 6-9

A limited number of Freestanding Ambulatory Surgical Centers will now be established under the New York State Certificate of Need regulations. If you are planning to license your Surgical Center, get ready for months of applications and headaches.

Maybe we can help. We are a consulting firm that specializes in Certificate of Need applications and capital financing for health care projects.

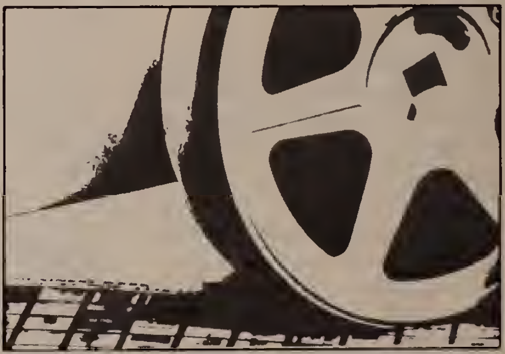
We are licensed mortgage brokers and experienced developers. In the last ten years we have built many projects and cured many a headache.

ART GLAUDE BRIAN O'CONNOR

NORTH ATLANTIC CAPITAL CORPORATION

NEW YORK — ALBANY (212) 864-4300

This Publication is available in Microform.



University Microfilms International

Please send additional information

for ____________________ (name of publication)

Name____________________

Institution____________________

Street____________________

City____________________

State__________ Zip__________

300 North Zeeb Road, Dept. P.R., Ann Arbor, Mi. 48106

FOR SEVERE COUGHS, CAN YOU REMEMBER THESE LETTERS?

You know Robitussin®, and the "DAC" formula gives you just what you need to manage those severe coughs complicated with nasal congestion . . . Decongestant And Codeine!

Robitussin®-DAC Ⓒ

Each 5 ml (1 teaspoonful) contains:

Guaifenesin, USP	100 mg
Pseudoephedrine Hydrochloride, USP	30 mg
Codeine Phosphate, USP	10 mg

(Warning: May be habit forming)
Alcohol 1.4 percent

℞ ONLY

A-H-ROBINS
Pharmaceutical Division
Richmond, Virginia 23261-6609

WHY PARTICIPATE IN HEALTHWAYS

HealthWays

THE WAY TO BETTER HEALTH CARE

WHAT IS HEALTHWAYS?

A private practice IPA/HMO (Individual Practice Association/Health Maintenance Organization).

AREN'T ALL HMO'S CLINICS?

No. There are two basic types of HMO's:

1. The IPA
2. The clinic or center model

WHAT IS AN IPA?

An IPA is a corporation comprised of physicians that contracts with an HMO to provide physician services.

ARE ALL IPA/HMO'S ALIKE?

No. Some HMO's contract directly with individual physicians. Others, like HealthWays, contract with a physician corporation.

WHICH TYPE IS BETTER?

We strongly believe the corporation IPA far better serves the physicians and patients.

WHY?

Because the physician corporation is your voice assuring high quality standards, fair payment and an effective mechanism to protect your best interests.

WHY SHOULD I PARTICIPATE IN HEALTHWAYS?

For the following reasons:

- reimbursement policy
- maintenance of private practice
- quality of care
- physician-set standards of medical care
- broad scope of benefits for your patients
- payment procedure

WHAT IS HEALTHWAYS REIMBURSEMENT POLICY?

All HealthWays participating physicians are paid on a fee-for-service basis.

HOW DOES HEALTHWAYS HELP ME MAINTAIN MY PRIVATE PRACTICE?

Through patient retention and new patient growth.

WHAT DO YOU MEAN BY QUALITY OF CARE AND STANDARDS SET BY PHYSICIANS?

HealthWays' philosophy is that physicians—and only physicians—set medical policies and guidelines.

HOW DOES HEALTHWAYS BENEFIT MY PATIENTS?

The benefit package available to HealthWays members is far more comprehensive than traditional health insurance today—and that includes coverage for well care.

WHAT IS HEALTHWAYS PAYMENT PROCEDURE?

HealthWays pays physicians directly—not the patient—for all covered services rendered. This eliminates bad debt.

HOW CAN I BE SURE I'LL BE HAPPY WITH HEALTHWAYS?

In four years of operations, HealthWays physician roster has grown to over 1400. Of that number, only four have left.

HOW DO I PARTICIPATE?

Contact your local IPA for full information. Or if you want more information about HealthWays, call 1-800-624-0720.

HealthWays, Inc. • Parkway Towers • 485A U.S. Route 1 • Iselin, NJ 08830

New
Motrin® 800 TABLETS mg
ibuprofen

Extra strength
Convenience
Economy

Upjohn

The Upjohn Company
Kalamazoo, Michigan 49001 USA

© 1985 The Upjohn Company

J-5491 June 1985

EXCERPTS FROM A SYMPOSIUM
"THE TREATMENT OF SLEEP DISORDERS"[8]

> ". . . highly effective for both sleep induction and sleep maintenance"

Sleep Laboratory Investigator
Pennsylvania

> ". . . onset of action is rapid. . .provides sleep with no rebound effect to agitate the patient the following day"

Psychiatrist
California

> ". . . appears to have the best safety record of any of the benzodiazepines"

Psychiatrist
California

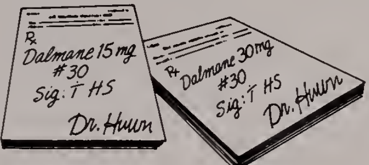
After 15 years, the experts still concur about the continuing value of Dalmane (flurazepam HCl/Roche). It provides sleep that satisfies patients. . . and the wide margin of safety that satisfies you.

The recommended dose in elderly or debilitated patients is 15 mg. Contraindicated in pregnancy.

DALMANE®
flurazepam HCl/Roche Ⓒ
sleep that satisfies

15-mg/30-mg
capsules

References: 1. Kales J, *et al: Clin Pharmacol Ther 12*:691-697, Jul-Aug 1971. **2.** Kales A, *et al: Clin Pharmacol Ther 18*:356-363, Sep 1975. **3.** Kales A, *et al: Clin Pharmacol Ther 19*:576-583, May 1976. **4.** Kales A, *et al: Clin Pharmacol Ther 32*:781-788, Dec 1982. **5.** Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc 27*:541-546, Dec 1979. **6.** Dement WC, *et al: Behav Med*, pp. 25-31, Oct 1978. **7.** Kales A, Kales JD: *J Clin Psychopharmacol 3*:140-150, Apr 1983. **8.** Tennant FS, *et al:* Symposium on the Treatment of Sleep Disorders, Teleconference, Oct 16, 1984. **9.** Greenblatt DJ, Allen MD, Shader RI: *Clin Pharmacol Ther 21*:355-361, Mar 1977.



DALMANE®
flurazepam HCl/Roche Ⓒ

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended. Repeated therapy should only be undertaken with appropriate patient evaluation.

Contraindications: Known hypersensitivity to flurazepam HCl; pregnancy. Benzodiazepines may cause fetal damage when administered during pregnancy. Several studies suggest an increased risk of congenital malformations associated with benzodiazepine use during the first trimester. Warn patients of the potential risks to the fetus should the possibility of becoming pregnant exist while receiving flurazepam. Instruct patients to discontinue drug prior to becoming pregnant. Consider the possibility of pregnancy prior to instituting therapy.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Withdrawal symptoms rarely reported; abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase, and paradoxical reactions, *e.g.* excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg recommended initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

ROCHE®
Roche Products Inc.
Manati, Puerto Rico 00701

#1 IN EXPERIENCE

Worldwide, it's a known quantity... known for sleep that satisfies.

You know it helps patients fall asleep quickly and stay asleep till morning.[1-8] You know its exceptionally wide margin of safety.[7-9] You know it better than any other hypnotic. The *only* benzodiazepine hypnotic with more than 15 years of continuing satisfactory performance. As always, caution patients about driving or drinking alcohol.

Please see references and summary of product information on reverse side

DALMANE®
flurazepam HCl/Roche Ⓒ
sleep that satisfies

ROCHE

Copyright © 1985 by Roche Products Inc. All rights reserved.

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

OCTOBER 1985 • Volume 85, Number 10

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

Contents

COMMENTARIES

Membranoproliferative glomerulonephritis 571
RODRIGO E. URIZAR, MD

Massive hemoptysis 573
JOHN A CROCCO, MD

The charitable obligations of the physician 574
DANIEL M. MUSHER, MD

The strengths of organized medicine 576
GEORGE J. LAWRENCE, JR., MD

RESEARCH PAPERS

Quantitative Doppler spectrum analysis of extracranial carotid artery stenosis 577
MALCOLM O. PERRY, MD, MICHAEL F. SILANE, MD, DAVID CALCAGNO, MD, SUSAN SPRADLIN

Use of gonadotropins in ovulation induction: Analysis of treatment protocols and pregnancy outcome 580
PAUL R. GINDOFF, MD, RAPHAEL JEWELEWICZ, MD

THE CPC

A 28-year-old man with a left-sided palatal mass 585

MEDICAL EDUCATION

Institutional ethics and graduate medical education 590
ALFRED GELLHORN, MD

CASE REPORTS

Epidermoid cysts of the cranium 596
V. JAGADHA, MD, [illegible]

Primary osteogenic sarcoma of the skull: Five-year disease-free survival following surgery and high-dose methotrexate therapy 598
NARAYAN SUNDARESAN, MD, JOSEPH A. GALICICH, MD, GERALD ROSEN, MD, ANDREW G. HUVOS, MD

Silicone granulomas of the breast 599
GLADYS CRUZ, MD, JOHN F. GILLOOLEY, MD, MARIAN WAXMAN, MD

Erythroleukemia in the adjuvant treatment of breast cancer 601
STUART M. LICHTMAN, MD, PHILIP SCHULMAN, MD, DANIEL R. BUDMAN, MD

Massive lower gastrointestinal hemorrhage secondary to colonic varices 604
CHARLES C. CANVER, MD, JAMES F. UPSON, MD

Massive hemoptysis due to retained rib fragment 40 years after thoracotomy 605
FREDERICK A. BEVELAQUA, MD, ROSEMARY WIECZOREK, MD, ARTHUR D. BOYD, MD

LETTERS TO THE EDITOR

Porcelain gallbladder in a 24-year-old woman 609
STEPHEN R. BAKER, MD
SHALOM BUCHBINDER, MD

Thrombophlebitis: an unusual cause of chest pain 609
DANIEL FAIERMAN, MD, JOE H. DWEK, MD

A large swallowed foreign body in the esophagus 610
V. K. KAPOOR, MS, A. K. KRIPLANI, MS, HEMANT SINGHAL, MBBS, M. C. MISHRA, MS, T. K. CHATTOPADHYAY, MS, L. K. SHARMA, MS, FICS

Glomus tumor of the clitoris 611
V. JAGADHA, MD, K. SRINIVASAN, MD, P. PANCHACHARAM, MD

BOOK REVIEWS 612

OBITUARIES 615

GUIDELINES TO AUTHORS 618

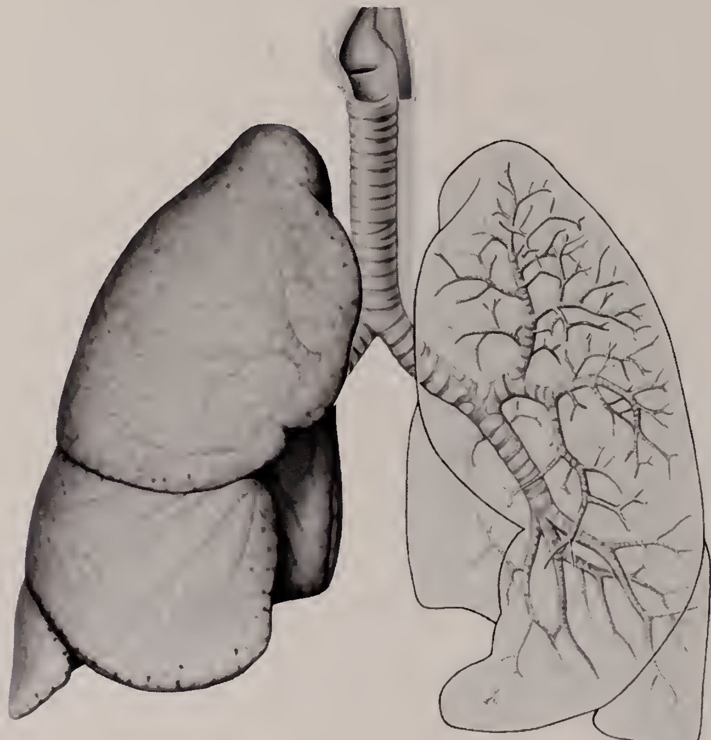
MEDICAL MEETINGS AND LECTURES 5A

March 7–9, 1986, New York Hilton, New York City
cember issue for Preliminary Program)

Library of the College Physicians
of Philadelphia Dec. 85
19 S. 22nd St.
Phila, PA 19103

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA
NOV 14 1985

Consider the causative organisms...



Ceclor®
cefaclor

250-mg Pulvules® t.i.d.

offers effectiveness against the major causes of bacterial bronchitis

H. influenzae, *H. influenzae*, *S. pneumoniae*, *S. pyogenes*
(ampicillin-susceptible) (ampicillin-resistant)

Brief Summary Consult the package literature for prescribing information

Indications and Usage Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms.

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae)*, *Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci).

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out.

Precautions *General Precautions* – If an allergic reaction to Ceclor® (cefaclor, Lilly) occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy – Pregnancy Category B – Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor® (cefaclor, Lilly). There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers – Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor is administered to a nursing woman.

Usage in Children – Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below.

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain – Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic – Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic – Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal – Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

[061782R]

Note Ceclor® (cefaclor, Lilly) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

© 1984, ELI LILLY AND COMPANY

Lilly

Additional information available to the profession on request from
Eli Lilly and Company,
Indianapolis, Indiana 46285

Eli Lilly Industries, Inc
Carolina, Puerto Rico 00630

400441

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD
FRANK C. MAXON, JR, MD
ELIZA H. CALDWELL, MD
RICHARD B. NOLAN, MD
URSULA A. MATULONIS (Medical Student)
JOHN T. PRIOR, MD

Editor	ALAN BLUM, MD
Deputy Editor and Book Review Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Production Manager	KATHLEEN WALSH TULLEY
Librarian and Administrative Assistant	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS
Editorial Assistant	MILDRED ARFMANN
Secretary	JOSEPHINE KREMPASKY
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1985

RANDALL BLOOMFIELD, MD
ROBERT D. BRANDSTETTER, MD
JOHN S. DAVIS, MD
MARK C. HOMONOFF, MD
ALFRED P. INGEGNO, MD
FLORENCE KAVALER, MD
ROBERT LANDESMAN, MD
JAMES M. MORRISSEY, ESQ
STEPHEN NORDLICHT, MD
JOSEPH SCHLUGER, MD
BJORN THORBJARNARSON, MD
RODRIGO E. URIZAR, MD
NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. Synergistic Media Sales, 50 East 42nd St., Suite 504, New York, N.Y. 10017. Telephone: (212) 682-2200.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

DANIEL F. O'KEEFFE, MD, *President*
ALLISON B. LANDOLT, MD, *Past-President*
JOHN A. FINKBEINER, MD, *President-Elect*
SAMUEL M. GELFAND, MD, *Vice-President*
JOHN H. CARTER, MD, *Secretary*
GEORGE LIM, MD, *Assistant Secretary*
CHARLES D. SHERMAN, JR, , MD, *Treasurer*
MORTON KURTZ, MD, *Assistant Treasurer*
CHARLES N. ASWAD, MD, *Speaker*
SEYMOUR R. STALL, MD, *Vice-Speaker*

Councilors

Term Expires 1986
ROBERT A. MAYERS, MD, Westchester
THOMAS D. PEMRICK, MD, Rensselaer
RALPH E. SCHLOSSMAN, MD, Queens
STANLEY A. STECKLER, MD, Suffolk
ROBERT J. NEWBORN, Albany
(Councilor representing the medical student membership)

Term Expires 1987
DAVID M. BENFORD, MD, New York
DUANE M. CADY, MD, Onondaga
JAMES M. FLANAGAN, MD, Wayne
M. THEODORE TANENHAUS, MD, Brooklyn

Term Expires 1988
JAMES H. COSGRIFF, JR, MD, Erie
RICHARD A. HUGHES, MD, Warren
ANTONIO F. LASORTE, MD, Broome
SIDNEY MISHKIN, MD, Nassau

Trustees

G. REHMI DENTON, MD, Albany, *Chairman*
RALPH M. SCHWARTZ, MD, New York
EDGAR P. BERRY, MD, New York
RICHARD D. EBERLE, MD, Onondaga
JOSEPH F. SHANAPHY, MD, Richmond
VICTOR J. TOFANY, MD, Monroe
BERNARD J. PISANI, MD, New York

Executive Vice-President	GEORGE J. LAWRENCE, JR, MD
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Associate	IRMA A. ERICKSON
Director, Division of Scientific Publications	ALAN BLUM, MD
Director, Division of Management Services	GEORGE FORREST, JR
Assistant Director, Division of Governmental Affairs	JERRY S. HOFFMAN, ESQ
Director, Division of Communications	EDWARD HYNES
Director, Division of Insurance and Membership Benefits	BERNARD M. JACKSON
Director, Division of Membership	EUNICE SKELLY
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Scientific Activities	PETER B. FARNSWORTH, MD
Director, Division of Advertising-Sales and Meeting Services	ROBERT MILLER
General Counsel	DONALD R. MOY, JD
Director, Division of Computer Services	IVAN H. NAJMAN
Acting Director, Division of Medical Services	MARIO MENGHINI
Director, Division of Long-Range Planning	DEBORAH R. SIEGEL
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. Editorial material published in the *Journal* is covered by copyright. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

Angina Protection with Benefits for a Lifetime

ONCE-DAILY CONTROL WITH HEART-SAVING BENEFITS

By reducing heart rate and cardiac contractility, INDERAL LA helps protect the heart from the potentially serious and debilitating consequences of ischemia. A highly effective antianginal agent for around-the-clock control of symptoms, INDERAL LA also provides cardiovascular protection for a sense of security in the years ahead.

PROTECTION AND EXPERIENCE NO CALCIUM BLOCKER CAN MATCH

Unlike calcium blockers, INDERAL LA—either alone or with a nitrate—is recommended for early treatment of angina in the majority of patients. Equally important, INDERAL LA delivers the proven performance and safety profile of INDERAL tablets—confirmed by millions of patients during 16 years of clinical use.

START WITH 80 MG ONCE DAILY

Dosage may be increased to 160 mg once daily, as needed, to achieve optimal control. INDERAL LA should not be used in congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma. Please see next page for further details and brief summary of prescribing information.

ONCE-DAILY **INDERAL LA**

(PROPRANOLOL HCl) *LONG ACTING CAPSULES*

Ayerst.

The appearance of INDERAL LA capsules is a registered trademark of Ayerst Laboratories

JUST ONCE EACH DAY FOR SIMPLIFIED CORE THERAPY IN ANGINA

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES

Just once each day...

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

INDERAL* LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established. Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain. Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use. Effects on plasma volume appear to be minor and somewhat variable. INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients.

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction. Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential The significance of the membrane action in the treatment of arrhythmias is uncertain.

The mechanism of the antimigraine effect of propranolol has not been established. Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction. Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

Propranolol is not significantly dialyzable.

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock; 2) sinus bradycardia and greater than first degree block; 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY. The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e.g., dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes In these patients, it may be more difficult to adjust the dosage of insulin.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General:* Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL is not indicated for the treatment of hypertensive emergencies

Beta adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure.

Clinical Laboratory Tests. Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy: Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: bradycardia; congestive heart failure; intensification of AV block, hypotension; paresthesia of hands; thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type.

Central Nervous System. lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia; visual disturbances; hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics.

Gastrointestinal: nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic: pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

Respiratory: bronchospasm.

Hematologic: agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous: alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained. INDERAL LA should not be considered a simple mg for mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS).

MIGRAINE—*Dosage must be individualized.* The initial oral dose is 80 mg INDERAL LA once daily. The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis It a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

*The appearance of INDERAL LA capsules is a registered trademark of Ayerst Laboratories

8833/384

Ayerst.

AYERST LABORATORIES.
New York, N.Y. 10017

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meetings and lectures unless the information is submitted at least three months prior to the event.*

NOVEMBER 1985

AROUND THE STATE

MANHATTAN

Nov 6. **Suicide: Epidemic of the 80s.** 1 Cat 1 credit. Info: The Regent Hospital, 425 East 61st St, New York, NY 10021. Tel: (212) 935-3400.

Nov 12–13. **Reducing Legal Risks and Liabilities in the Management and Delivery of Health Care.** Novatel New York. *Nov. 20–21.* **Cost Effective Management Strategies for the Long-Term Care Setting.** Doral Inn. Info: The Health and Education Council, 7201 Rossville Blvd, Baltimore, MD 21237. Tel: (301) 686-3610.

Nov 13. **Important Concepts in Nephrology for Pediatricians.** 6½ Cat 1 credits. Info: Beth Israel Medical Center, First Avenue at 16th Street, New York, NY 10003. Tel: (212) 420-2849.

Nov 16–17. **The Treatment of Eating Disorders: What's Working and Why.** Omni Park Central Hotel. Info: Center for the Study of Anorexia and Bulimia, 1 West 91 St, New York, NY 10024. Tel: (212) 595-3449.

Nov 20. **Psychosis and Epilepsy.** New York University Medical Center. Info: F. Frank LeFever, PhD, Neuropsychology Lab, Helen Hayes Hospital, West Haverstraw, NY 10993. Tel: (914) 947-3000.

Nov 21. **Thrombolysis in Acute Myocardial Infarction: Is It Worth It?** Harley Hotel. 2 Cat 1 credits. Info: The New York Cardiological Society, Inc., 84 Grove St, Suite 2, New York, NY 10014. Tel: (212) 924-5346.

Nov 22. **Suicide and Adolescents.** Doral Inn. Info: The Health and Education Council, 7201 Rossville Blvd, Baltimore, MD 21237. Tel: (301) 686-3610.

(*Continued on p 16A*)

The appearance of these tablets is a registered trademark of Wyeth Laboratories

®

DISTINCTIVE DESIGN 0.5, 1.0 and 2.0 mg

Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Transient amnesia or memory impairment has been reported in association with the use of benzodiazepines

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levarterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

Ativan® Ⓒ IV for (lorazepam) Anxiety

DOSAGE: Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.

HOW SUPPLIED: 0.5, 1.0 and 2.0mg tablets.

Wyeth Laboratories
Philadelphia, PA 19101

Only Ativan®
(lorazepam) ©IV

ffers all these enefits in ddition to rapid elief of anxiety:

elief of anxiety ssociated with epressive symptoms

learance not ignificantly delayed y age, liver or idney dysfunction

umulative sedative ffects seldom problem

ttle likelihood of rug interaction
ll benzodiazepines produce additive ffects when taken with alcohol or ther CNS depressants)

o significant changes vital signs in ardiovascular patients*

hort duration of action, imple metabolism

enzodiazepines have not been shown to e of benefit in treating the cardiovascular omponent.

ee important information
n preceding page
19[illegible] Wyeth Laboratories

Vyeth Laboratories

Classified Advertising

PHYSICIANS WANTED

EXCELLENT OPPORTUNITY FOR READY MADE PRACTICE for General Practice Specialist or Internist willing to do a little general practice, available immediately, in an attractive Medical Building with in-house lab and x-ray facilities. Permanent relationship very desirable. Beautiful countryside-Cortland, NY. Phone collect (607) 756-9941 or submit c.v. with references to: Donald R. Gibbs, M.D., 110 North Main Street, Cortland, NY 13045.

NEW YORK, NEW PALTZ—Seeking full-time experience or residency trained physicians; ACLS certified; for state of the art FEC in beautifull Mid-Hudson Valley, 80 miles from NYC. Start date is July 15, 1985. Package includes Health, Life, Disability and Malpractice Insurance. Excellent salary, attractive scheduling and potential profit sharing. Send CV/Inquiries to Gary W. Greer, M.D., RD 3 Box 44A, Cogan Station, PA., 17728: phone 1-717-494-0420.

ARIZONA BASED PHYSICIAN RECRUITMENT firm has opportunites Coast to Coast. "Professionals working with Professionals". Over 13 years experience. Call (602) 795-7474; or send CV to: Mitchell & Associates, Inc., 2761 N. Country Club Rd., Suite 202, Tucson, AZ 85716.

DESPERATELY SEEKING ASSOCIATE to share some expenses and gradually (3–5 yrs.) take over active internal medicine practice in prime location, White Plains, NY. Prefer creative, industrious, entrepreneurial individual with some capital. Send cv to Dept. 393 c/o NYSJM.

INTERNAL MEDICINE, large practice outside Buffalo, New York for sale. For more information send C.V. to: Health Care Personnel Consulting, Inc., 400 GSB Building, One Belmont Avenue, Bala Cynwyd, PA 19004 (215) 667-8630.

SURGERY: GENERAL PRACTICE AVAILABLE—this GP/GS combination practice in upstate New York is a unique opportunity for the physician interested in a broad practice. Send C.V. to: Health Care Personnel Consulting, Inc., 400 GSB Building, One Belmont Avenue, Bala Cynwyd, PA 19004 (215) 667-8630.

EASTERN LONG ISLAND, NEW YORK. Internist wanted to form a partnership in a busy primary care practice. Position immediately available. Reply Dept. 396 c/o NYSJM.

PHYSICIAN WANTED for urgent care office in Rochester, New York. Full time, competitive salary, flexible hours. Apply to: Promptcare Management Corp., 2024 W. Henrietta Road, Rochester, New York 14623, Attn: V.F. Distefano.

PHYSICIANS WANTED—CONT'D

FAMILY PRACTITIONER—Established medical group needs a family practitioner. Located in the sun-belt and no state tax. Guaranteed salary and early partnership for the conscientious person. Contact clinic administrator 10737 Gateway West, El Paso, Texas 79935 or call (915) 592-0201.

PHYSICIAN—NEW YORK CITY—MANHATTAN. Must be licensed to practice in NY State. Good salary, many fringe benefits including profit sharing. Patient population intelligent and cooperative. No medicaid. Good working conditions. Apply to G. H. Ebreo, 133 East 58th Street, Suite 502, New York City, NY 10022.

GASTROENTEROLOGIST: Two man gastroenterology group in Central New York near Syracuse looking for third M.D. One hundred percent gastroenterology practice. Excellent opportunity. Many recreational facilities. Must be proficient in all phases of invasive gastroenterology, endoscopy, and nutrition. Please send curriculum vitae with three references to Dept. 397 c/o NYSJM.

WANTED TO LOCATE IN THE SUNBELT: all specialties. To communicate your availability, we'll condense your CV and circulate the condensed CV to over 2300 hospitals, clinics, group practices, and labs in 12 southern states. No cost to you. Send CV to Trent Associates, 2421 Shades Crest Road, Birmingham, AL 35216.

FAMILY PRACTITIONER—FOR FAMILY HEALTH CENTER Operated by 350 bed community hospital in Eastern, L.I. affiliated w/Medical School SUNY at Stony Brook. Outstanding local recreational & cultural activities; 60 miles from NYC. Opportunity for resident teaching & faculty appointments. Send C.V. to: Dorothy S. Lane, M.D., Brookhaven Memorial Hospital Medical Center, 101 Hospital Road, Patchogue, L.I.N.Y. 11772 (516) 654-7795.

SURGERY-VASCULAR SURGEON—Full time, academic position in a major teaching medical center in New York area. Must be BE/BC in Vascular Surgery. Guaranteed income plus incentives. Benefits negotiable. Send complete CV to Dept. 398 c/o NYSJM. All inquiries confidential.

PRACTICES AVAILABLE

SELLING PRACTICES IS OUR ONLY BUSINESS—Want to maximize the return on your investment? Before you buy or sell, call for a consultation to assure the best financial and transfer terms. We guide you through the entire sales process from initial meeting to closing. Serving 100 mile radius of New York City. Countrywide Business Brokerage, Inc., 319 East 24th Street, Suite 23-G, New York, NY 10010 (212) 686-7902.

MEDICAL PRACTICE SALES and Appraisals. We specialize in the valuation and selling of medical practices. If interested in buying or selling a medical practice, contact our Brokerage Division at: The Health Care Group, 400 GSB Building, Bala Cynwyd, PA 19004. (215) 667-8630.

FIGHT LUNG DISEASE WITH CHRISTMAS SEALS.®

TAKE CARE OF YOUR LUNGS. THEY'RE ONLY HUMAN.

AMERICAN LUNG ASSOCIATION
The Christmas Seal People ®

Space contributed by the publisher as a public service.

PRACTICES AVAILABLE—CONT'D

APPRAISALS-SALES-RELOCATION
Medical Practices—All Specialties

MEDICAL PRACTICES TRANSFER
Frederick Katz, Ph.D. & Assoc.

42-27 Union Street, Flushing, N.Y. 11355
Tel. (718) 445-9797 (any time)

INTERNAL MEDICINE—Active, long-established solo practice for sale. Nassau county; home-office fully equipped for 1 or 2 physicians, within minutes of several hospitals. Phone (516) 747-3072.

PRACTICES WANTED

OPHTHALMOLOGIST, BOARD CERTIFIED desires to purchase practice in New York City or suburban area. Confidence maintained. Please contact Dept. 168, c/o NYSJM.

FOR SEVERE COUGHS, CAN YOU REMEMBER THESE LETTERS?

You know Robitussin®, and the "DAC" formula gives you just what you need to manage those severe coughs complicated with nasal congestion . . . Decongestant And Codeine!

Robitussin®-DAC ⓥ

Each 5 ml (1 teaspoonful) contains:
Guaifenesin, USP .... 100 mg
Pseudoephedrine Hydrochloride, USP .... 30 mg
Codeine Phosphate, USP .... 10 mg
(Warning: May be habit forming)
Alcohol 1.4 percent

℞ ONLY

A·H·ROBINS
Pharmaceutical Division
Richmond, Virginia 23261-6609

BALANCED CALCIUM CHANNEL BLOCKADE!

Low incidence of side effects
CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

Reduces angina attack frequency*
42% to 46% decrease reported in multicenter study.[1]

Increases exercise tolerance*
In Bruce exercise test,[2] control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes ($P<.005$).

CARDIZEM®
(diltiazem HCl)

THE BALANCED
CALCIUM CHANNEL BLOCKER

Please see full prescribing information on following page. 2/84

---

*Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

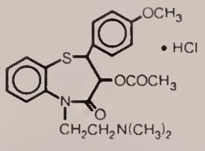
References:
1. Strauss WE, McIntyre KM, Parisi AF, et al: Safety and efficacy of diltiazem hydrochloride for the treatment of stable angina pectoris: Report of a cooperative clinical trial. Am J Cardiol 49:560-566, 1982.
2. Pool PE, Seagren SC, Bonanno JA, et al: The treatment of exercise-inducible chronic stable angina with diltiazem: Effect on treadmill exercise. Chest 78 (July suppl):234-238, 1980.

PROFESSIONAL USE INFORMATION

cardizem®
(diltiazem HCl)
30 mg and 60 mg tablets

DESCRIPTION

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one,3-(acetyloxy) -5-[2-(dimethylamino)ethyl]-2,3-dihydro-2-(4-methoxyphenyl)-, monohydrochloride,(+) -cis-. The chemical structure is:



Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform. It has a molecular weight of 450.98. Each tablet of CARDIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration.

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth muscle.

Mechanisms of Action. Although precise mechanisms of its antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways:

1. Angina Due to Coronary Artery Spasm: CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial and subendocardial. Spontaneous and ergonovine-induced coronary artery spasm are inhibited by CARDIZEM.
2. Exertional Angina: CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal and maximal exercise work loads.

In animal models, diltiazem interferes with the slow inward (depolarizing) current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Diltiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance.

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricular conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load. Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect; cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of diltiazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by diltiazem.

Intravenous diltiazem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Diltiazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, diltiazem significantly prolongs sinus cycle length (up to 50% in some cases).

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Diltiazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40%. CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 60 mg are given; a 120-mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem.

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm.** CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina).** CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia.
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing.
Cardiac Failure	Administer inotropic agents (isoproterenol, dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

The oral/LD_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort acute anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy**—CARDIZEM may be safely coadministered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

Cardizem 30-mg tablets are supplied in bottles of 100 (NDC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1771-49). Each green tablet is engraved with MARION on one side and 1771 engraved on the other. CARDIZEM 60-mg scored tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1772-49). Each yellow tablet is engraved with MARION on one side and 1772 on the other.

Issued 4/1/84

Another patient benefit product from

PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MISSOURI 64137

HMO FACTS OF LIFE

By now, you've undoubtedly heard about HMOs, IPAs, et al. They're complicated, confusing and causing physicians considerable concern in terms of their present and future practices.

The following clearly defines what each is. After reviewing these various types of pre-paid health care, we believe you will have the basic information necessary to choose the best for you.

HEALTH MAINTENANCE ORGANIZATIONS (HMOs)

CENTER OR CLINIC

- Central facility or facilities
- Staff physicians
- Patient selects a primary care physician (Internist, GP, FP, and Pediatrician) from the staff
- May frequently have physician turnover resulting in short term doctor-patient relationship and/or the need to re-select primary physician

DIRECT PHYSICIAN CONTRACTING

- No central facility
- Private practicing physicians
- Patient selects a primary care physician (Internist, GP, FP, Pediatrician)
- Obstetrician-Gynecologist is not a primary, but a specialist. The patient is seen by the Obstetrician-Gynecologist only by referral from the primary care physician.
- Primary physicians are capitated. That means they are paid a fixed amount annually to treat a specific patient whether they see that patient once—or a hundred times.
- No group voice because there is no physician group
- Specialists are reimbursed a percentage of their fee
- Patients can see physicians when they want

IPA CORPORATIONS

- No central facility
- Private practicing physicians
- Patient selects 1 or 2 primary physicians: Female and male adults select an Internist, GP or FP; females additionally select an Obstetrician-Gynecologist; parents select a Pediatrician, GP or FP for their child(ren).

  Obstetrician-gynecologists are primary care physicians, and can be seen directly by the member for both routine and specialized care with no referral necessary.
- A corporate body which is organized by physicians. The IPA then contracts with an HMO to provide services to the HMO members. *The physician does not join the HMO, but simply participates.*
- The IPA is the physicians' voice to the HMO which represents the physician re: fee reimbursement, quality of care, peer review, etc.
- Traditional fee-for-service payment
- Patients can see physicians when needed. Each visit is paid for by the HMO on a fee-for-service basis

EXAMPLES

HIP	HMO-NJ	HealthWays, Inc.
Kaiser Permanente	HMO of Greater NY	

If you have further questions regarding any of the above, call HealthWays' IPA Department at 1-800-624-0720 in New Jersey or 201-636-6200 out-of-state.

HealthWays

THE WAY TO BETTER HEALTH CARE

© 1985 HealthWays, Inc.

On nitrates,
but angina still
strikes...

After a nitrate, add ISOPTIN®

(verapamil HCl/Knoll)

To protect your patients, as well as their quality of life, add Isoptin instead of a beta blocker.

First, Isoptin not only reduces myocardial oxygen demand by reducing peripheral resistance, but also increases coronary perfusion by preventing coronary vasospasm and dilating coronary arteries — both normal and stenotic. These are antianginal actions that no beta blocker can provide.

Second, Isoptin spares patients the beta-blocker side effects that may compromise the quality of life.

With Isoptin, fatigue, bradycardia and mental depression are rare. Unlike beta blockers, Isoptin can safely be given to patients with asthma, COPD, diabetes or peripheral vascular disease. Serious adverse reactions with Isoptin are rare at recommended doses; the single most common side effect is constipation (6.3%).

Cardiovascular contraindications to the use of Isoptin are similar to those of beta blockers: severe left ventricular dysfunction, hypotension (systolic pressure <90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no artificial pacemaker is present) and second- or third-degree AV block.

So, the next time a nitrate is not enough, add Isoptin... for more comprehensive antianginal protection without side effects which may cramp an active life style.

ISOPTIN. Added antianginal protection without beta-blocker side effects.

Please see brief summary on following page.

ISOPTIN®

(verapamil HCl/Knoll)

80 mg and 120 mg scored, film-coated tablets

Contraindications: Severe left ventricular dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (except in patients with a functioning artificial ventricular pacemaker), 2nd- or 3rd-degree AV block. **Warnings:** ISOPTIN should be avoided in patients with severe left ventricular dysfunction (e.g., ejection fraction < 30% or moderate to severe symptoms of cardiac failure) and in patients with any degree of ventricular dysfunction if they are receiving a beta blocker. (See *Precautions.*) Patients with milder ventricular dysfunction should, if possible, be controlled with optimum doses of digitalis and/or diuretics before ISOPTIN is used. (Note interactions with digoxin under *Precautions.*) ISOPTIN may occasionally produce hypotension (usually asymptomatic, orthostatic, mild and controlled by decrease in ISOPTIN dose). Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported. Such elevations may disappear even with continued treatment; however, four cases of hepatocellular injury by verapamil have been proven by rechallenge. Periodic monitoring of liver function is prudent during verapamil therapy. Patients with atrial flutter or fibrillation and an accessory AV pathway (e.g. W-P-W or L-G-L syndromes) may develop increased antegrade conduction across the aberrant pathway bypassing the AV node, producing a very rapid ventricular response after receiving ISOPTIN (or digitalis). Treatment is usually D.C.-cardioversion, which has been used safely and effectively after ISOPTIN. Because of verapamil's effect on AV conduction and the SA node, 1° AV block and transient bradycardia may occur. High grade block, however, has been infrequently observed. Marked 1° or progressive 2° or 3° AV block requires a dosage reduction or, rarely, discontinuation and institution of appropriate therapy depending upon the clinical situation. Patients with hypertrophic cardiomyopathy (IHSS) received verapamil in doses up to 720 mg/day. It must be appreciated that this group of patients had a serious disease with a high mortality rate and that most were refractory or intolerant to propranolol. A variety of serious adverse effects were seen in this group of patients including sinus bradycardia, 2° AV block, sinus arrest, pulmonary edema and/or severe hypotension. Most adverse effects responded well to dose reduction and only rarely was verapamil discontinued. **Precautions:** ISOPTIN should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacologic effects. Studies in a small number of patients suggest that concomitant use of ISOPTIN and beta blockers may be beneficial in patients with chronic stable angina. Combined therapy can also have adverse effects on cardiac function. Therefore, until further studies are completed, ISOPTIN should be used alone, if possible. If combined therapy is used, close surveillance of vital signs and clinical status should be carried out. Combined therapy with ISOPTIN and propranolol should usually be avoided in patients with AV conduction abnormalities and/or depressed left ventricular function. Chronic ISOPTIN treatment increases serum digoxin levels by 50% to 70% during the first week of therapy, which can result in digitalis toxicity. The digoxin dose should be reduced when ISOPTIN is given, and the patients should be carefully monitored to avoid over- or under-digitalization. ISOPTIN may have an additive effect on lowering blood pressure in patients receiving oral antihypertensive agents. Disopyramide should not be given within 48 hours before or 24 hours after ISOPTIN administration. Until further data are obtained, combined ISOPTIN and quinidine therapy in patients with hypertrophic cardiomyopathy should probably be avoided, since significant hypotension may result. Clinical experience with the concomitant use of ISOPTIN and short- and long-acting nitrates suggest beneficial interaction without undesirable drug interactions. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. *Pregnancy Category C*: There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor and delivery only if clearly needed. It is not known whether verapamil is excreted in breast milk; therefore, nursing should be discontinued during ISOPTIN use. **Adverse Reactions:** Hypotension (2.9%), peripheral edema (1.7%), AV block: 3rd degree (0.8%), bradycardia: HR < 50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%), elevations of liver enzymes have been reported. (See *Warnings.*) The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain: ecchymosis, bruising, gynecomastia, psychotic symptoms, confusion, paresthesia, insomnia, somnolence, equilibrium disorder, blurred vision, syncope, muscle cramp, shakiness, claudication, hair loss, macules, spotty menstruation. **How Supplied:** ISOPTIN (verapamil HCl) is supplied in round, scored, film-coated tablets containing either 80 mg or 120 mg of verapamil hydrochloride and embossed with "ISOPTIN 80" or "ISOPTIN 120" on one side and with "KNOLL" on the reverse side. Revised August, 1984. 2385

knoll **KNOLL PHARMACEUTICAL COMPANY**
30 NORTH JEFFERSON ROAD, WHIPPANY, NEW JERSEY 07981

2406

ORGANIZED 1807

PHYSICIANS IN RECOVERY

HELP!

The MSSNY wants the help of physicians who are recovering from chemical dependence (alcohol and other drugs) or mental illness. Other physicians attempting recovery need your guidance and support. The Committee for Physicians' Health (Physicians' Committee) can put you in touch. Please call or write.

We'll keep it confidential.

Barton A. Harris, M.D.
Medical Director
Committee for Physicians' Health
Medical Society of the
State of New York
P.O. Box 5404
Lake Sucess, NY 11042

516-488-7777

ALZHEIMER'S DEMENTIA

Cure of the disease is still out of reach. In as devastating a condition as this, even the most modest relief of symptoms—or for that matter keeping them from getting worse or merely slowing their intensification—is a great contribution to patient and family.

HYDERGINE® LC (ergoloid mesylates) is indicated for patients over age sixty who manifest signs and symptoms of idiopathic mental decline. It appears that individuals who respond to HYDERGINE LC therapy are those who would be considered to suffer from some ill-defined process related to aging or to suffer from some underlying condition such as Alzheimer's dementia.

Before prescribing HYDERGINE therapy, the possibility that the patient's signs and symptoms arise from a potentially reversible and treatable condition should be excluded. In addition, because the presenting clinical picture may evolve to suggest an alternative treatment, the decision to use HYDERGINE therapy should be continually reviewed.

HYDERGINE® LC
(ergoloid mesylates)
liquid capsules, 1 mg

THE ONLY PRODUCT INDICATED FOR ALZHEIMER'S DEMENTIA.

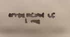
© 1985 Sandoz, Inc.

HYD-1085-13

For Brief Summary, please see following page.

HYDERGINE® LC

(ergoloid mesylates) liquid capsules 1 mg



Indications: Symptomatic relief of signs and symptoms of idiopathic decline in mental capacity (i.e., cognitive and interpersonal skills, mood, self-care, apparent motivation) in patients over sixty. It appears that individuals who respond to HYDERGINE therapy are those who would be considered clinically to suffer from some ill-defined process related to aging or to have some underlying dementing condition, such as primary progressive dementia, Alzheimer's dementia, senile onset, or multi-infarct dementia. Before prescribing HYDERGINE® (ergoloid mesylates), the physician should exclude the possibility that signs and symptoms arise from a potentially reversible and treatable condition, particularly delirium and dementiform illness secondary to systemic disease, primary neurological disease, or primary disturbance of mood. Not indicated for acute or chronic psychosis regardless of etiology (see Contraindications).

Use of HYDERGINE therapy should be continually reviewed, since presenting clinical picture may evolve to allow specific diagnosis and specific alternative treatment, and to determine whether any initial benefit persists. Modest but statistically significant changes observed at the end of twelve weeks of therapy include: mental alertness, confusion, recent memory, orientation, emotional lability, self-care, depression, anxiety/fears, cooperation, sociability, appetite, dizziness, fatigue, bothersome(ness), and overall impression of clinical status.

Contraindications: Hypersensitivity to the drug; psychosis, acute or chronic, regardless of etiology.

Precautions: *Because the target symptoms are of unknown etiology, careful diagnosis should be attempted before prescribing HYDERGINE (ergoloid mesylates) preparations.*

Adverse Reactions: Serious side effects have not been found. Some transient nausea and gastric disturbances have been reported, and sublingual irritation with the sublingual tablets.

Dosage and Administration: 1 mg three times daily. Alleviation of symptoms is usually gradual and results may not be observed for 3-4 weeks.

How Supplied: HYDERGINE LC (liquid capsules): 1 mg, oblong, off-white, branded "HYDERGINE LC 1 mg" on one side, "△" other side. Packages of 100 and 500. (Encapsulated by R. P. Scherer, N.A., Clearwater, Florida 33518).

HYDERGINE (ergoloid mesylates) tablets (for oral use): 1 mg, round, white, embossed "HYDERGINE 1" on one side, "△" other side. Packages of 100 and 500.

Each liquid capsule or tablet contains ergoloid mesylates USP as follows: dihydroergocornine mesylate 0.333 mg, dihydroergocristine mesylate 0.333 mg, and dihydroergocryptine (dihydro-alpha-ergocryptine and dihydro-beta-ergocryptine in the proportion of 2:1) mesylate 0.333 mg, representing a total of 1 mg.

Also available: HYDERGINE sublingual tablets; 1 mg, oval, white, embossed "HYDERGINE" on one side, "78-77" other side. Packages of 100 and 1000. 0.5 mg, round, white, embossed "HYDERGINE 0.5" on one side, "△" other side. Packages of 100 and 1000.

HYDERGINE liquid; 1 mg/ml. Bottles of 100 mg with an accompanying dropper graduated to deliver 1 mg. [HYD—ZZ24—6 15 84]

Before prescribing, see package circular for full product information. HYD-1085-13

DORSEY PHARMACEUTICALS
Division of Sandoz, Inc. • East Hanover, NJ 07936
A SANDOZ COMPANY

QUEENS

Nov 8. **Immune System Disorders: Diagnosis, Prevention, and Management.** 6 Cat 1 credits. Sheraton Inn at La Guardia. Info: Barbara Lewis, Immune System Disorders Symposium, c/o 2966 Jerusalem Ave, Wantagh, NY 11793. Tel: (516) 785-4951.

Nov 5. **Pathophysiology and Treatment of Septic Arthritis.** *Nov 7.* **Neonatal Seizures: Update.** *Nov 12.* **Pharmacotherapeutics of Shock.** *Nov 14.* **Hemophilus Influenza: Disease and Vaccine.** *Nov 19.* **Symposium: Clinical Applications and Ethical Implications of HTLV-III Antibody.** *Nov 26.* **Symposium: Early Diagnosis and Management of Breast Carcinoma.** Info: Ms. Clo Vezza, Booth Memorial Medical Center, 56-45 Main St, Flushing, NY 11355. Tel: (718) 670-1419.

NASSAU

Nov 1. **Investigation and Treatment of Adrenal Tumors in Adults.** *Nov 15.* **Introduction to Nuclear Medicine.** Info: Medical Staff Office, Long Beach Memorial Hospital, 455 East Bay Dr, Long Beach, NY 11561. Tel: (516) 432-8000, ext 2275.

Nov 6. **Small Airway Dysfunction.** *Nov 20.* **Management of Thoracic and Abdominal Trauma.** Info: Medical Education Department, Mercy Hospital, 1000 North Village Ave, Rockville Centre, NY 11570. Tel: (516) 225-2260.

Nov 7. **A Breath in Time.** Info: Franklin General Hospital, 1072 Franklin Ave, Valley Stream, NY 11580. Tel: (516) 794-1160.

WESTCHESTER

Nov 1–2. **The Role of the Burn Team in the Future.** 12 Cat 1 credits. Info: Deborah Creedy, RN, New York Group for Plastic Surgery and Rehabilitation, Macy Pavilion, Westchester County Medical Center, Valhalla, NY 10595. Tel: (914) 347-4900.

Nov 15. **Sleep Disorders.** 3 Cat 1 credits. Info: Beth Davies, RN, Harlem Valley Psychiatric Center, Wingdale, NY 12594. Tel: (914) 832-6611, ext 655.

ALBANY

Nov 9. **Second Annual Anesthesiology Teaching Day.** 6 Cat 1 credits. *Nov 13.* **Hearing and Learning: An Ageless Problem.** 6 Cat 1 credits. *Nov 14.* **Flexible Sigmoidoscopy for the Family Practitioner.** 7 Cat 1 credits. *Nov 21.* **Sports Medicine: Musculo-skeletal Injuries, Cardiac Evaluation, and Conditioning.** 4 Cat 1 credits. Info: Office of Continuing Medical Education, Albany Medical College, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

ERIE

Nov 7. **Help for Today, Hope for Tomorrow: Diabetes Mellitus Management and Research Update.** 6 Cat 1 credits. Info: American Diabetes Assocation, P.O. Box 1037, Syracuse, NY 13201. Tel: (315) 472-9111.

AROUND THE NATION

CALIFORNIA

Nov 8–9. **Epilepsy in Childhood: Current Concepts.** The Stanford Court, Nob Hill. 7½ Cat 1 credits. Info: Extended Programs in Medical Education, Room 569-U, University of California, San Francisco, CA 94143. Tel: (415) 475-4251.

LOUISIANA

Nov 18–21. **Primary Care Update.** Marriott Hotel, New Orleans. 4 Cat 1 credits. Info: Interstate Postgraduate Medical Association, P.O. Box 1109, Madison, WI 53701. Tel: (608) 257-6781.

Nov 22. **Clinical Care and Cost Constraints in Geriatric Medicine.** New Orleans Marriott. 6¾ Cat 1 credit. Info: American Geriatrics Society, 10 Columbus Circle, Suite 1470, New York, NY 10019.

MASSACHUSETTS

Nov 22–24. **Group Psychotherapy.** 22 Cat 1 credits. Info: Harvard Medical School, Department of Continuing Medical Education, Boston, MA 02115. Tel: (617) 732-1525.

POSITIONS WANTED

PULMONARY INTERNIST, 30, ABIM certified, completing fellowship June 1986. Fully trained in all aspects of pulmonary medicine. Seeks hospital-based or subspecialty group practice affiliated with a teaching institution. Reply Harlan Weinberg, M.D., 337 N. Palm Drive, Beverly Hills, CA. 90210, Apt. 8.

PHYSICIAN COUPLE SEEKING STAFF POSITION OR PRACTICE. Husband, BC Pediatrician with private practice and emergency room experience of five years. Wife, BE in Physical Medicine and Rehabilation with extensive EMG training. Both university trained, FLEX, age 35. Available immediately. Call (201) 667-6534 after 7 P.M.

REAL ESTATE FOR SALE OR RENT

CENTRAL PARK WEST MEDICAL at 86th St. & CPW. PHYSICIANS—Time-sharing (staff, equipment & services provided) or your own exclusive office built to suit. PSYCHIATRIST—Beautiful ground floor soundproof office—exclusive or shared. N.Y.'s most elegant rental, beautiful lobby, security. Prime location on busy block near all transportation. (718) 229-8440.

PROFESSIONAL SPACE AVAILABLE: Prestigious Bay Ridge location. Walk-down office. Will alter to suit. Long term lease. Call (718) 996-0166.

REAL ESTATE FOR SALE OR RENT—CONT'D

BAY SHORE, Leasing new luxurious medical building. Located near Southside Hospital. Minimum suite 740 sq. ft. Large parking area, handicapped designed. Individual gas heat, a/c-electric. (516) 665-4064, or 665-4482.

WANTED: MEDICAL OFFICE BUILDINGS. As principals we are seeking "existing buildings"—fully tennanted by physicians, dentists and health care services. Contact; Paul Gellert, Gelco Associates, 155 West 68th St., N.Y., N.Y. 10023; (212) 724-7900.

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

MANHATTAN WESTEND AVENUE AT 93rd STREET. Professional co-op apartment for sale with private entrance on Westend Avenue. Avenue views. Newly gut renovated, new kitchen, bathroom, windows, wiring and floors. Closets galore. Perfect for live/work combination. Maintenance $642.00; Price $260,000. Call Irving Kahn at (212) 223-2935.

REAL ESTATE FOR SALE OR RENT—CONT'D

MEDICAL OFFICE AVAILABLE in village of Syosset on the north shore of Long Island. 1800 sq. ft., prime location on first floor, for rent with option to buy in co-op ownership. Call (516) 921-6020.

FOR SALE Beautiful Doctor's home and office in Great Neck, corner property in Kensington. 3 examining rooms and lab, 4 blocks to stores and station. 4 bedrooms, 2 servants rooms, 4½ baths double sinks. Front and back stairways. 2 car garage (overhead doors) electric openers. $535,000. Telephone (516) 487-3474 or 751-8880.

Manheim Realty, Inc.
Commercial, Industrial & Business Brokerage

MANHEIM. . .the professional name in private practice sales. Let the professional division of MANHEIM REALTY INC. help you in buying or selling your practice, or locating professional space. We can insure that you will receive personal service and that your business will be held in the strictest confidence. Please contact:

Ruth Karp, R.N., EAB Plaza, Uniondale, NY 11555
(516) 794-6600

Oceanside Living At Its Finest

Seabrook Island is resort living at its best on South Carolina's coastline.

Located off the coast of historic Charleston, this private residential resort features 2 championship golf courses, Har-Tru tennis, miles of beach, excellent stables and sports programs.

You'll discover homes and homesites situated on the fairways and tennis courts, the sea marsh and ocean, all in a splendidly private natural setting.

So come for a week and stay for a lifetime. For information or reservations, call toll free 800-845-2475.

Seabrook Island
Ocean Club
Box 32099, Charleston, South Carolina 29417
800-845-2475

Send for our free Guide to Resort Living

☐ Send your free Guide to Resort Living.
☐ Send vacation information

Name ____
Address ____
Phone ____
NYJM 1085

THE ADVANCED **HAIR REPLACEMENT** SYMPOSIUM
SPONSORED BY
AMERICAN ACADEMY
OF FACIAL PLASTIC AND RECONSTRUCTIVE SURGERY
FEBRUARY 6-8, 1986
in
HOT SPRINGS, ARKANSAS

This course is designed to teach the principles and operative techniques i.e., punch autograft refinements, scalp reductions, hair-bearing flaps by utilizing an outstanding multidiscipline faculty selected from dermatologists, otolaryngologists and plastic surgeons. Formal presentations and open discussions are featured.

COURSE DIRECTORS: D. BLUFORD STOUGH, III, M.D. and RICHARD WEBSTER, M.D., P.S. For more information contact: The Stough Dermatology & Cutaneous Surgery Clinic, P.A., Doctors Park, Hot Springs, AR 71901, 501/624-0673.

FACULTY

T. H. Alt, M.D.
E. C. Gay, Jr., M.D.
L. A. Lewis, M.D.
G. D. Monheit, M.D.
O. T. Norwood, M.D.
D. Orentreich, M.D.
D. B. Stough, III, M.D.
W. P. Unger, M.D.
R. C. Webster, M.D.
M. R. Wright, PhD
W. K. Wright, M.D.

Emphasis will be placed on a live surgical work shop for participants with minimal experience.

EQUIPMENT

FOR SALE: Irex M-Mode Echocardiogram Machine with phonocardiogram, apex and carotid pulse tracing capabilities. Supplies, recording paper and transducers included. For further information call: Ralph J. D'Angelo, M.D. (914) 793-1606.

MISCELLANEOUS

1986 CME CRUISE/CONFERENCES on Selected Medical Topics—Caribbean, Mexican, Hawaiian, Alaskan, Mediterranean. 7-12 days year-round. Approved for 20-24 CME Cat. 1 credits (AMA/PRA) & AAFP prescribed credit. Distinguished professors. Fly Roundtrip Free on Caribbean, Mexican & Alaskan Cruises. Excellent group fares on finest ships. Registration Limited. Pre-scheduled in compliance with present IRS requirements. Information: International Conferences, 189 Lodge Ave., Huntington Station, NY 11746. (516) 549-0869.

TIMESHARING MEDICAL OFFICES. Start a new practice, expand an established practice, retire and practice part-time, without the hassle and expense of establishing or managing an office. Dr. Share, a medical timesharing network, now has three new fully equiped prestigous Manhattan locations as part of our network. Please, write or call for more information, Dr. Share, P.O. Box 8051, Garden City, NY (516) 486-8400.

U S Postal Service

STATEMENT OF OWNERSHIP, MANAGEMENT AND CIRCULATION
Required by 39 U S C 3685)

1A TITLE OF PUBLICATION NEW YORK STATE JOURNAL OF MEDICINE	1B PUBLICATION NO 0 0 2 8 7 6 2 8	2 DATE OF FILING 9/16/85
3 FREQUENCY OF ISSUE Monthly	3A NO OF ISSUES PUBLISHED ANNUALLY 12	3B ANNUAL SUBSCRIPTION PRICE $25.00

4 COMPLETE MAILING ADDRESS OF KNOWN OFFICE OF PUBLICATION *(Street, City, County, State and ZIP+4 Code) (Not printers)*
420 Lakeville Road, Post Office Box 5404, Lake Success, New York 11042

5. COMPLETE MAILING ADDRESS OF THE HEADQUARTERS OF GENERAL BUSINESS OFFICES OF THE PUBLISHER *(Not printer)*
420 Lakeville Road, Lake Success, New York 11042

6 FULL NAMES AND COMPLETE MAILING ADDRESS OF PUBLISHER, EDITOR, AND MANAGING EDITOR *(This item MUST NOT be blank)*

PUBLISHER *(Name and Complete Mailing Address)*
Medical Society of the State of New York 420 Lakeville Road, Lake Success, New York 11042

EDITOR *(Name and Complete Mailing Address)*
Alan Blum, M.D. Medical Society of the State of New York, 420 Lakeville Road-Lake Success, New York 11042

MANAGING EDITOR *(Name and Complete Mailing Address)*
None

7 OWNER *(If owned by a corporation, its name and address must be stated and also immediately thereunder the names and addresses of stockholders owning or holding 1 percent or more of total amount of stock. If not owned by a corporation, the names and addresses of the individual owners must be given. If owned by a partnership or other unincorporated firm, its name and address as well as that of each individual must be given. If the publication is published by a nonprofit organization, its name and address must be stated.) (Item must be completed.)*

FULL NAME	COMPLETE MAILING ADDRESS
Medical Society of the State of New York	420 Lakeville Road, Post Office Box 5404
	Lake Success, New York 11042
A non-profit membership corporation. No	
stockholders.	

8 KNOWN BONDHOLDERS, MORTGAGEES, AND OTHER SECURITY HOLDERS OWNING OR HOLDING 1 PERCENT OR MORE OF TOTAL AMOUNT OF BONDS, MORTGAGES OR OTHER SECURITIES *(If there are none, so state)*

FULL NAME	COMPLETE MAILING ADDRESS
NONE	

9 FOR COMPLETION BY NONPROFIT ORGANIZATIONS AUTHORIZED TO MAIL AT SPECIAL RATES *(Section 423 12 DMM only)*
The purpose, function, and nonprofit status of this organization and the exempt status for Federal income tax purposes *(Check one)*

(1) ☐ HAS NOT CHANGED DURING PRECEDING 12 MONTHS
(2) ☐ HAS CHANGED DURING PRECEDING 12 MONTHS
(If changed, publisher must submit explanation of change with this statement.)

10 EXTENT AND NATURE OF CIRCULATION *(See instructions on reverse side)*	AVERAGE NO. COPIES EACH ISSUE DURING PRECEDING 12 MONTHS	ACTUAL NO. COPIES OF SINGLE ISSUE PUBLISHED NEAREST TO FILING DATE
A TOTAL NO. COPIES *(Net Press Run)*	27,767	28,630
B PAID AND/OR REQUESTED CIRCULATION 1 Sales through dealers and carriers, street vendors and counter sales	-0-	-0-
2 Mail Subscription *(Paid and/or requested)*	22,410	22,471
C TOTAL PAID AND/OR REQUESTED CIRCULATION *(Sum of 10B1 and 10B2)*	22,410	22,471
D FREE DISTRIBUTION BY MAIL, CARRIER OR OTHER MEANS SAMPLES, COMPLIMENTARY, AND OTHER FREE COPIES	5,155	5,474
E TOTAL DISTRIBUTION *(Sum of C and D)*	27,565	27,945
F COPIES NOT DISTRIBUTED 1. Office use, left over, unaccounted, spoiled after printing	202	685
2 Return from News Agents	-0-	-0-
G TOTAL *(Sum of E, F1 and 2—should equal net press run shown in A)*	27,767	28,630

11 I certify that the statements made by me above are correct and complete

SIGNATURE AND TITLE OF EDITOR, PUBLISHER, BUSINESS MANAGER, OR OWNER
Philip G Keating

PS Form 3526, July 1984 *(See instruction on reverse)*

NEW YORK STATE
JOURNAL OF MEDICINE

October 1985 • Volume 85, Number 10

COMMENTARIES

Membranoproliferative glomerulonephritis

Progressive idiopathic glomerulonephritis encompasses a variety of poorly defined glomerulopathies, some of which lead to end stage renal disease (ESRD).[1] Gotoff,[2] West,[3] and their collaborators[4] distinguished idiopathic membranoproliferative glomerulonephritis (MPGN) from this amorphous group of chronic nephritides, and demonstrated persistent hypocomplementemia and ultrastructural abnormalities that allowed the further differentiation of two varieties. Subendothelial and mesangial electron dense deposits are characteristic of type I,[4–7] whereas partial or complete replacement of the lamina densa of the glomerular basement membrane by a ribbon-like, dense deposit defines MPGN type II, or dense deposit disease.[4–9] Chemically, this material is slightly different from the normal lamina densa, and does not react with antihuman globulin C_3 or other antisera in the immunohistopathologic setting.[10] In addition, approximately 90% of patients with MPGN type II have a circulating C_3 nephritic factor (C_3NeF).[4] This immunoglobulin autoantibody possesses a larger heavy chain than normal IgG and blocks the C_3 convertase-inactivator of the alternative pathway of complement activation.[11]

A third type of MPGN (MPGN type III) is characterized by the juxtaposition of epimembranous and subendothelial deposits and lamination of the glomerular basement membrane.[4] However, these changes have been thought to occur often enough in MPGN type I to consider type III a part of the spectrum of type I. Clinical presentation and evolution of MPGN types I and II are indistinguishable.[4,7] Although more children than adults develop MPGN type II, persistent proteinuria, recurrent hematuria, and acute poststreptococcal glomerulonephritis-like onset occur more often at a younger age regardless of the type of MPGN.[7] Occasional direct and indirect evidence of recent streptococcal infection may be present and cause the clinician to diagnose incorrectly the postinfectious disease. Persistent hematuria, proteinuria, and urinary sediment abnormalities associated with hypocomplementemia, beyond the time the poststreptococcal glomerulopathy should have abated, strongly suggest MPGN. Histologic evidence should be sought to confirm the diagnosis since older children and adults frequently develop hypertension, progressive renal failure, and the nephrotic syndrome, and both groups of patients may show a prolonged silent period.[4]

The etiology and pathogenesis of MPGN remain unclear; it is presumed that circulating, or in situ, immune complexes[12] and complement, prostaglandin, and platelet abnormalities play a key role in the progression of MPGN.[1,13,14] The association of MPGN type I with alpha-1 antitrypsin deficiency[15] and MPGN types I and II with partial lipodystrophy[16] is unique and makes the understanding of the pathogenesis of this entity even more complex.

A thrombocytopathic mechanism is considered essential for the development and progression of inflammation and glomerulosclerosis in MPGN. Decreased platelet survival, platelet antigen deposition in glomerular capillaries, and platelet release of myointimal, histiocyte, and mesangial cell-growth factors have been identified.[1,13,14,17] Platelets in patients with MPGN are destroyed selectively without concurrent changes in fibrinogen and plasminogen survival.[13,14,17] In addition, injection of adenosine monophosphate in rabbits has been shown to induce platelet aggregation and release reaction, the formation of abundant platelet microemboli, and glomerulonephritic-sclerotic lesions associated with arterial hypertension.[18] Thus, it appears established at this time that platelet-related acute and chronic renal inflammatory processes and progressive glomerulosclerosis are responsible in part for the severe clinical impact of MPGN (ie, greater than 50% ESRD by five to ten years after diagnosis).[5]

Morbidity and/or mortality of this degree supported several uncontrolled and controlled therapeutic trials applied to MPGN in the past.[5,19-21] Most studies utilized immunosuppressive, antiinflammatory (prednisone and other derivatives), and anticoagulant drugs, alone or in combination, with claims of up to 70% to 76% improvement of the five year survival. A dilemma arises, however, when one attempts to analyze a disparate array of data obtained with multiple medications, schedules, dosages, clinical follow-up, and histologic studies. Since 1964 West[3] and McEnery[4] have consistently administered oral prednisone to MPGN patients in a dosage of 2 mg/kg of body weight every other day early in the course of the disease. In 1984, 48 of 66 patients undergoing this regimen had been followed from two to 20 years; the five and ten year survival rates were greater than 95% and greater than 80%, respectively. Five patients (10.4%) developed ESRD; normal complement levels were present in 93% of patients whereas 91% had low levels at onset. Serum creatinine levels were less than 3.0 mg/dL in over 90% and less than 1.6 mg/dL in 86% of patients (normal, 0.7–1.5 mg/dL). A randomized, double blind, placebo-controlled trial of aspirin and dipyridamole in patients with MPGN type I showed the treated group to have better stabilization of the glomerular filtration rate.[17] Progression to ESRD was less frequent and occurred over longer intervals, and platelet survival improved. The overall success of the trial was 18 of 21 (86%, $P < 0.05$).

The salutary effect of antiplatelet aggregating medications is due to the inhibition of two platelet enzymes: cyclooxygenase (aspirin) and phosphodiesterase (dipyridamole).[1,7] This combined action blocks the platelet aggregation-release reaction, decreasing the synthesis of thromboxane (aggregating and vasoconstrictor agent) with the predominance of prostacyclin (PGI_2) effects. Thus, the potent antiinflammatory action of prednisone, which improves platelet survival,[22] and modulating the thromboxane/prostacyclin balance are highly desirable for the treatment of patients with MPGN; it may also be useful in patients with other progressive and sclerosing glomerulopathies.

A correlation of clinical tests (complement, C_3NeF and creatinine levels, glomerular filtration rate), disappearance of the nephrotic syndrome, normalization of urinary sediment and platelet function, and immunohistopathologic criteria provided by serial kidney biopsies yields information critical to the assessment of therapeutic efficacy and patient status. Immunohistopathology may become a controversial issue because of interpretative ambiguities. Development of glomerular and tubulo-interstitial fibrosis may be prominent. McEnery[4,24] and West[3,23] indicated that although histologic studies in MPGN, particularly type I, revealed prompt and quantitatively significant reduction of inflammation, hypercellularity, and mesangial interposition, long-term evaluation demonstrated either increased segmental scarring or glomerular obsolescence. Such abnormalities are offset, however, by the persistence of viable and functional renal tissue without progressive inflammatory changes that may indicate treatment-induced healing. After prolonged follow-up, a number of patients showed sustained improvement of the parameters mentioned earlier, thus enforcing the contention that the patient's condition is the result of therapy. Thus, these data emphasize the hypothesis that early and long-term use of prednisone, aspirin, and dipyridamole control the progression of, but may not necessarily cure, MPGN.

Analysis of this information can hardly be ignored; undoubtedly it reinforces that active intervention with the combined use of antiinflammatory and antiplatelet aggregating agents is the primary treatment modality for MPGN. The experience at the Division of Pediatric Nephrology of the Albany Medical College with early treatment of this condition is limited. Since 1969, however, patients with MPGN type I and those with type II who had no glomerular filtration rate impairment, who were started on alternate day prednisone therapy and more recently on a combination of prednisone every other day and aspirin and dipyridamole daily within six months of diagnosis, and maintained on it for at least three years, have fared remarkably well. Those whose clinical and laboratory test results remained stable after three years were able to taper and eventually discontinue their medications. Retreatment of those who escape control is always feasible and may be successful again. Thus, the results alluded to above[3,4,17] clearly reconcile the rationale for the use of these medications with the clinical improvement of renal function and patient survival with minimal or no untoward side effects. These substances provide the physician with the unique opportunity to treat a progressive glomerulopathy of both children and adults. Therefore, further combined (prednisone-aspirin-dipyridamole) controlled trials are warranted in the treatment of patients with both types of MPGN.

RODRIGO E. URIZAR, MD
Research Physician
Wadsworth Center for Laboratories
and Research
New York State Department of
Health
Professor of Pediatric Nephrology
Albany Medical College
Albany, NY 12208.

1. Cameron JS: Glomerulonephritis: current problems and understanding. *J Lab Clin Med* 1982; 99:755–787.
2. Gotoff SP, Fellers FX, Vawter GF, Janeway CA, Rosen FS: The beta-1C globulin in childhood nephrotic syndrome: laboratory diagnosis of progressive glomerulonephritis. *N Engl J Med* 1965; 273;524–529.
3. West CD, McAdams AJ, McConville JM, et al: Hypocomplementemic and normocomplementemic persistent (chronic) glomerulonephritis; clinical and pathologic characteristics. *J Pediat* 1965; 67:1089–1112.
4. McEnery PT, Strife CF: Chronic glomerulonephritis in children, in Tine BM, Mendoza SA, Brenner MB, Stein JH (eds): *Pediatric Nephrology*. New York, Churchill Livingstone, 1984, pp 231–264.
5. Habib R, Levy M: Clinicopathologic correlations in 128 cases of membranoproliferative glomerulonephritis. *Proc 6th Int Congr Nephrol* 1976, pp 462–467.
6. Kim Y,Michael AF: Idiopathic membranoproliferative glomlerulonephritis. *Annu Rev Med* 1980; 31:273–288.
7. Cameron JS, Turner DR, Heaton J, et al: Idiopathic mesangiocapillary glomerulonephritis. Comparison of types I and II in children and adults and long-term prognosis. *Am J Med* 1983; 74:175–192.
8. Habib R, Gubler MC, Loirat C, Ben-Maiz H, Levy M: Dense deposit disease: a variant of membranoproliferative glomerulonephritis. *Kidney Int* 1975; 7:204–215.
9. Sibley RK, Kim Y: Dense intramembranous deposit disease: new pathologic features. *Kidney Int* 1984; 25:660–670.
10. Kim Y, Vernier RL, Fish AJ, Michael AF: Immunofluorescence studies of dense deposit disease. The presence of railroad tracks and mesangial rings. *Lab*

Invest 1979; 40:474-480.

11. Schifferli JA: Complement in Nephritis, in Bertani J, Remuzzi G (eds): *Glomerular Injury 300 Years after Morgani*. Milan, Wichtig Editore, 1983, pp 57-74.

12. Urizar RE, Gilboa N, Largent JA: Immunologic mechanisms of childhood renal diseases. *Paediatrician* 1981; 10:254-313.

13. Cameron JS: Platelets in glomerular disease. *Annu Rev Med* 1984; 35:175-180.

14. Hayslett JP: Role of platelets in glomerulonephritis [editorial]. *N Engl J Med* 1984; 310:1457-1458.

15. Strife CF, Hug G, Chuck G, McAdams AJ, Davis CA, Kline JJ: Membranoproliferative glomerulonephritis and alpha 1-antitrypsin deficiency in children. *Pediatrics* 1983; 71:88-92.

16. Sissons JG, West RJ, Fallows J, et al: The complement abnormalities of lipodystrophy. *N Engl J Med* 1976; 294:461-465.

17. Donadio JV Jr, Anderson CF, Mitchell JC III, et al: Membranoproliferative glomerulonephritis. A prospective clinical trial of platelet-inhibitor therapy. *N Engl J Med* 1984; 310:1421-1426.

18. Jørgensen L, Glynn MF, Hovig T, Murphy EA, Buchanan MR, Mustard JF: Renal lesions and raise in blood pressure caused by adnenosine diphosphate-induced platelet aggregation in rabbits. *Lab Invest* 1970; 23:347-357.

19. International Study of Kidney Diseases in Children. Alternate-day steroid therapy in membranoproliferative glomerulonephritis: A randomized control clinical trial (abstr). *Kidney Int* 1982; 21:150.

20. Cattran DC, Cardella CJ, Roscoe CM, et al: Results of a controlled drug trial in membranoproliferative glomerulonephritis. *Kidney Int* 1985; 27:436-441.

21. Zimmerman SW, Moorthy AV, Dreher WH, Friedman A, Varanasi U: Prospective trial of warfarin and dipyridamole in patients with membranoproliferive glomerulonephritis. *Am J Med* 1983; 75:920-927.

22. Harker LA, Slichter SJ: Platelet and fibrinogen consumption in man. *N Engl J Med* 1972; 287:999-1005.

23. West CD: Membranoproliferative glomerulonephritis: classification and treatment, in Gruskin AB, Norman ME (eds): *Pediatric Nephrology. Proceedings of the Fifth International Pediatric Nephrology Symposium*. Boston, Martinus Nijhoff, 1981, p 108.

24. McEnery PT, McAdams AJ, West CD: Membranoproliferative glomerulonephritis: improved survival with alternate day prednisone therapy. *Clin Nephrol* 1980; 13:117-124.

Massive hemoptysis

Hemoptysis has been defined as coughing up blood, whether it be gross blood, a mixture of blood and sputum, or blood-streaked sputum. The blood may arise from anywhere in the air passages or from the pulmonary parenchyma itself, as well as from other areas including the nose, throat, mouth, and larynx. In determining the source of the blood, these areas can be excluded via careful examination and laryngoscopy. The most common cause of hemoptysis is bronchitis, and it is usually characterized by coughing, particularly during a respiratory infection.

Hematemesis may be mistaken for hemoptysis, but in the former the blood is vomited and is rarely frothy, usually dark red in color, acidic, and often mixed with food particles.

Massive hemoptysis has many definitions, but the most useful one is pulmonary hemorrhage of more than 600 mL of blood in 48 hours.[1] Causes of massive hemoptysis include tuberculosis (active or inactive, as well as that associated with aspergilloma in a remaining cavity), bronchiectasis, lung abscess, and lung cancer. Other less common causes are pulmonary arteriovenous fistula, mitral stenosis, and broncholithiasis.

The problems of massive hemoptysis have been reviewed by Bevelaqua et al in this issue of the *Journal*.[2] Although the cause of massive hemoptysis in this case report is rare, the method of diagnosis and management is well presented.

The mortality rates cited for severe pulmonary bleeding range from 50% to 100%.[3-6] Therefore, vigorous and proper management must be instituted in this dangerous situation. The modes of therapy that have been proposed range from sedation to watchful waiting to emergency surgery. Drug therapy using conjugated estrogenic substances,[7-9] vasopressin injections,[10,11] and phytonadione oxide[12-14] have been used, but there is no physiologic evidence to support their efficacy.[15] Another form of therapy in severe pulmonary hemorrhage has been occlusion of the pulmonary[15] and, even more commonly, the bronchial[16] arteries. Flexible fiberoptic bronchoscopy with endobronchial tamponade has also been used as a rapid, simple, and effective technique for controlling massive hemoptysis in patients who are not able to undergo resection due to poor pulmonary reserve or coexisting medical problems.[17]

Surgery is the most definitive form of treatment in massive hemoptysis for it removes the site of hemorrhage, thereby controlling bleeding or preventing recurrence.[1] The lethal element in massive hemoptysis appears to be the rate of bleeding. The greater the rate of bleeding, the higher the mortality.[1] Rapid flooding of the tracheobronchial tree causes the patient to drown in his own blood regardless of the patient's age or the extent of underlying pulmonary disease. In patients who are bleeding more slowly, surgery is performed to avert future hemorrhage rather than to control bleeding. Since 87% of patients with massive hemoptysis have experienced previous pulmonary bleeding and more than half of these patients die without surgery, prophylactic resection seems worthwhile.[1]

The difference in mortality rates between patients who undergo surgery and those patients who could undergo surgery but do not suggests that surgery offers a better prognosis than conservative management, especially when the rate of bleeding is greater than 600 mL in 16 hours.[1]

Successful surgical resection for uncontrolled pulmonary hemorrhage has been reported with increasing frequency.[1,3-6,18-20] Crocco et al[1] studied 67 patients with pulmonary hemorrhage of more than 600 mL within 48 hours. Mortality was primarily related to the rate of bleeding and exceeded 75% when the patient bled more than 600 mL within 16 hours. Surgical intervention in patients' bleeding at this rate was associated with a 23% mortality rate. Also, in lung abscess complicated by massive hemoptysis, prompt resection carried a better prognosis than conservative therapy.[1,15,20]

In one study,[1] 16 patients with active tuberculosis underwent surgery prior to receiving complete medical therapy. There were no postoperative complications. The tuberculosis was well controlled with chemotherapy, and it resolved after surgery. These results lend support to the contention that lung resection is practical even in the presence of active tuberculosis or incompletely treated lung abscess.

In the management of massive hemoptysis, the following program is utilized. The physician evaluates the condition of the patient, with emphasis on maintaining an adequate airway. Tracheostomy is performed when necessary to maintain adequate clearance of blood and secretions from the tracheobronchial tree as well as adequate ventilation. (Indeed, asphyxiation is the major cause of death in massive hemoptysis.) Vital signs are monitored and frequent determinations of hemoglobin or hematocrit values, or both, are made. Six units of whole blood should be available so that transfusion is not unduly delayed, and any deficiency of clotting factors should be corrected. An emergency chest roentgenogram is obtained to localize the site of bleeding and to evaluate the underlying disease.

The patient is positioned to prevent the gravitational spread of blood into the opposite lung. Reassurance by the physician and 30 mg to 60 mg of phenobarbital sodium every six hours are beneficial in allaying anxiety. Codeine sulfate is contraindicated because it depresses coughing and enhances aspiration.

A thoracic surgeon should be consulted in order that the surgeon may see the patient at the onset of treatment. Bronchoscopy is performed in all patients with hemoptysis, however slight, to determine the site of bleeding. The optimal time for bronchoscopy is during the bleeding episode. During severe hemorrhage, the bleeding site may not be visualized because of rapid flooding of the tracheobronchial tree by fresh blood. However, it is preferable to accept the risk of an unrewarding bronchoscopic examination rather than to await a reduction in bleeding and fail to discover the source. The site of bleeding was localized in 65 of the 67 patients in one series.[1]

In addition to the clinical evaluation, ventilatory function is determined in all patients. A single breath forced vital capacity determination can be obtained at the bedside by means of a portable spirometer. This simple test does not aggravate the bleeding and permits an estimation of the extent of both restrictive and obstructive pulmonary disease. The validity of ventilatory measurements in patients with blood in their tracheobronchial tree is subject to question. However, if the result of such a test is above the limits set to recommend against surgery, then the test has further helped to indicate the suitability of these patients for resection without danger of residual pulmonary insufficiency.

The wealth of evidence today suggests that the high mortality rate in massive hemoptysis is not related to the age of the patient or the extent of the underlying disease, but rather to the rate of bleeding. The success of prompt surgical intervention in reducing the mortality rate suggests that resection is preferable to conservative management.

JOHN A. CROCCO, MD
Associate Director of Medicine
Chief, Pulmonary Section
St. Vincent's Hospital
and Medical Center of New York
153 W 11th St
New York, NY 10011

1. Crocco JA, Rooney JJ, Fankushen DS, et al: Massive hemoptysis. *Arch Intern Med* 1968; 121:495–498.
2. Bevelaqua FA, Wieczorek R, Boyd AD: Massive hemoptysis due to a retained rib fragment 40 years after thoracotomy. *NY State J Med* 1985; 85:605–608.
3. Ehrenhaft JL, Taber RE: Management of massive hemoptysis not due to pulmonary tuberculosis or neoplasm. *J Thorac Surg* 1955; 30:275–287.
4. Ford JM: The surgical treatment of pulmonary hemorrhage in tuberculosis. *J Thorac Cardiovasc Surg* 1958; 35:341–346.
5. Laforet EJ, Strieder JW: Subcostal extraperiosteal plombage for massive tuberculous hemoptysis. *Amer Rev Resp Dis* 1960; 81:397–402.
6. Linberg EJ: Emergency operation in patients with massive hemoptysis. *Amer Surg* 1964; 30:158–159.
7. Stefanini M, Dameshek W: *The Hemorrhagic Disorders*, ed 2. New York, Grune and Stratton, Inc, 1962, p 143.
8. Johnson JF: Changes in plasma prothrombin, Ac-globulin and antithrombin concentration following intravenous administration of estrogens. *Proc Soc Exp Biol Med* 1957; 94:92–94.
9. Popper J: The use of premarin IV in hemoptysis. *Dis Chest* 1960; 37:659–660.
10. Tsakiris A, Haemmerli UP, Bühlmann A: Reduction of portal venous pressure in cirrhotic patients with bleeding from esophageal varicies, by administration of a vasopressin derivative, phenylalanine-2-lysine-3 vasopressin. *Am J Med* 1964; 36:825–839.
11. Trimble HG, Wood JP: Pulmonary hemorrhage: its control by use of intravenous pituitrin. *Dis Chest* 1950; 18:345–351.
12. Levy S: Vitamin K in tuberculosis with special reference to pulmonary hemorrhage. *Am Rev Tuberc* 1942; 45:377–389.
13. Farber JE, Miller DK: Nutritional studies in tuberculosis. *Am Rev Tuberc* 1943; 48:406–411.
14. Sheely RF: Prothrombin deficiency in pulmonary tuberculosis, clinical relation and significance in hemoptysis. *JAMA* 1941; 117:1603–1606.
15. Bredin CP, Richardson PR, King TK, Sniderman KW, Sos TA, Smith JP: Treatment of massive hemoptysis by combined occlusion of pulmonary and bronchial arteries. *Am Rev Respir Dis* 1978; 117:969–973.
16. Tafreshi M, Sobo S, Adler L, Liss A, Pillari G: Treatment of hemoptysis via bronchial artery embolization. *NY State J Med* 1985; 85:38–40.
17. Saw EC, Gottlieb LS, Yokoyama T, et al: Flexible fiberoptic bronchoscopy and endobronchial tamponade in the management of massive hemoptysis. *Chest* 1976; 70:589–591.
18. Fisher AM, Shelley WM: Major hemoptysis. A presenting symptom in two interesting cases. *Arch Intern Med* 1966; 117:412–416.
19. Garzon AA, Cerruti M, Gourin A, et al: Pulmonary resection for massive hemoptysis. *Surgery* 1970; 67:633–638.
20. Mattox KL, Guinn GA: Emergency resection for massive hemoptysis. *Ann Thorac Surg* 1974; 17:377–383.

See also pp 605–608.

The charitable obligations of the physician

In my experience soliciting contributions for a variety of secular and religious charities, I have found physicians to be singularly uncharitable. For example, the median yearly gift by doctors who contribute to the United Way, the nondenominational umbrella charity that serves the greater Houston area, ranges between $100 and $250 at several Houston hospitals. The median gift among all doctors is actually $0, since the majority make no contribution at all. This level of giving is not appreciably greater than that of secretaries, technicians, or ancillary personnel. Some might argue that the United Way and other unified fund drives are not representative because physicians are more likely to contribute to individual organizations. However, with the exception of a few individuals, this same low level of participation can be seen in an array of communal, religious, and cultural charities. When doctors' names do appear on lists of contributors, they tend to gravitate toward the lowest categories of donors. Most practicing physicians in the United States run small but highly profitable private corporations, yet the medical profession is not associated with corporate giving or sponsorship of communal activities that characterize other corporations and small businesses. In an informal poll of nine accountants in three cities, I found that most doctors contribute less than 1% of their income to charitable causes, the amount generally falling between 0.1% and 1%.

Physicians utilize a complex set of rationalizations to justify their low level of financial commitment to philanthropic causes. The most often cited, by far, is that the work of a physician serves mankind, and no further contribution is warranted. I consider this response problematic. Teachers, firemen, farmers, and shopkeepers also serve mankind, as, in the broadest sense, do industrialists who, together, with their managerial staff, accountants, and lawyers, make jobs available. Furthermore, monetary contribution to charitable causes is a separate issue. In determining remuneration for services, society has placed physicians near the top of the wage scale; I regard as unfair a decision by the beneficiaries of that system not to contribute financially to it.

The second rationalization is that of self-sacrifice. In general, physicians believe that they have undergone a longer training without salary than others in society and have completed the training period greatly in debt. Earlier in this century, attending college was the exception, and the eight-year difference between graduating high school and graduating medical school was, indeed, enormous. At the present time, however, most well-motivated students go to college, and the bright and aggressive ones generally enter graduate programs as well, thereby reducing the gap between doctors and others in society. Internship, residency, and subspecialty training add an additional two to six years to the medical training. Until the late 1950s this training was non-remunerative; interns and residents received room, board, and (sometimes) laundry with an additional modest stipend of, for example, $50 per month. By contrast, today, the average internship pays $20,500 per year, with salaries increasing to $25,300 for a fifth-year resident. Although from the point of view of anticipated reward this may not be a large salary, many Americans, including some with a PhD degree who become teachers, may wait years to achieve such an income.

Regarding the indebtedness of physicians at the time of graduation from medical school, varying estimates are given. In the late 1970s it was fashionable to extrapolate from then-current interest rates and inflationary costs to a prediction of $250,000 debts at graduation. However, according to a recent report,[1] three quarters of one graduating class was in debt, and the median indebtedness was $22,000. Even if this debt were to be carried throughout residency, the average income in the first year of practice should enable most of the debt to be paid off if the young doctor would be willing to wait one more year before upgrading his or her standard of living. Thus, physicians' claims that they have studied far longer than other members of society and without financial remuneration, and that they have completed their training with an enormous debt tend to be exaggerated.

The third rationalization is that of self-sufficiency. Doctors like to believe that they "did it all" themselves; that having put themselves through medical school and subsequent training entitles them to extraordinary financial reward. But medical students are no longer children of the Depression. At some medical schools, one third are themselves children of physicians, and it remains difficult to recruit qualified students from low-income and minority backgrounds.[2] Moreover, because subsidies from governmental and private philanthropic sources maintain medical schools, each student is far more dependent on society than he is on himself. Of the $40,000 estimated cost to support a medical student through each year of a four-year program, the student pays only a fraction in the form of tuition. The education is made possible and largely supported by society, and it is my belief that the physician remains in debt to society throughout his professional life.

The fourth rationalization is twofold, incorporating elements of "we-have-nothing-but-our-own-time" and "we-have-no-business-to-leave-our-children." Certainly the practice of medicine depends on the skills of the individual, and incapacity eradicates wage-earning potential. But physicians are not alone in this regard; the same is true of nearly all other professionals, ranging from accountants to violinists. The concept that doctors have a special need to accumulate wealth because they have no business to leave to their children is another anachronism. Doctors regularly own part or all of their offices and/or professional building, and often laboratories or partial interest in a hospital, as well. Even that is only one small part. Thanks to assets acquired during years of practice, most physicians have invested in a variety of business ventures. For example, many doctors have become part-owners of apartment complexes in sunbelt cities and regularly participate in real estate ventures and tax-shelters of various kinds. Thus, it does not seem reasonable to exempt the physician from charitable obligations either because he is especially dependent on his own labors or because he has no business assets to leave to his heirs.

The final rationalization is that doctors are nothing more than members of the upper middle class. Many physicians just do not consider themselves wealthy, an attitude that is belied both by their life style and the size of their incomes. Compartmentalization in accord with modern tax laws plays a role: the private corporation can be viewed as a separate entity which owns, among other things, an office and an automobile; investments are built up in a retirement plan; and the physician may receive an income that covers his costs but does not seem excessive to him. The argument that the medical profession will not fare as well in the future as in the past is also part of the network of rationalizations. I can not predict the economics of medicine in the next decade, but I remember my medical school classmates in the early 1960s arguing that federal interference had forever eliminated their ability to generate substantial income, a prediction that could hardly have been further from the truth.

In summary, then, physicians have developed a complex set of rationalizations that result in a feeling of entitlement. They tend to believe that they are entitled to being supported by society without a monetary payback. Although there is some historical basis for some of these concepts, none is valid today. It is hoped that an open assessment of these rationalizations will stimulate greater interest in the ethical obligations of the physician to participate in charitable causes.

DANIEL M. MUSHER, MD
Infectious Disease Section
Veterans Administration Medical Center
2002 Holcombe Blvd
Houston, TX 77211

1. Moss AJ: Moonlighting house officers: the silent majority. *N Engl J Med* 1984; 311:1375-1377.
2. Report of the Association of American Medical Colleges Task Force on Minority Student Opportunities in Medicine. Washington, DC, Association of American Medical Colleges, 1978.

The strengths of organized medicine

The following letter was written in reply to a physician who resigned from the Medical Society of the State of New York because of his view that traditional organized medicine is losing its relevance and legislative impact.

I sincerely regret your feeling that our being organized as a professional association renders us either impotent or irrelevant; particularly since we don't really have any other options for expressing our collective will on such matters as legislation or government regulation of the medical profession.

I appreciate your understanding of the antitrust implications of certain concerted actions by members of a medical society. However, the legal limitations are a function not of our organizational framework but of physicians' status as independent professionals rather than employees.

While some physicians (eg, employees of a health maintenance organization) are allowed under law to bargain collectively, their right to do so extends only to negotiations with their employer. A recognized employer-employee relationship must exist in order for a group of individuals to have the legal right to associate and function as a union.

So what are we to do? We can either work through our professional associations to address legislative and regulatory issues, or we can lie down and accept what comes in the way of governmental control of our practices.

Fortunately, while participating in organized medicine may be "the only game in town" as far as political action is concerned, it at least offers us the opportunity to win occasionally.

The nation's health care policymakers would hardly agree that the American Medical Association is either impotent or irrelevant. In a recent survey they acknowledged that it's the single most influential force in the health care field, aside from the Department of Health and Human Services.

With the enormous pressures to reduce costs in health care today, it is clear that the government will not cease in its attempts to exercise control over the dollars it spends on medical services. Yet just because we face an unending battle to defend physicians' rights in the face of government-mandated change, and because we don't always get our way does not mean that the effort should be abandoned.

All of organized medicine is unhappy with the Medicare fee freeze and the division of physicians into participating and non-participating categories. However, without the efforts of the AMA, the legislation passed by Congress last year would have revoked physicians' hospital privileges if they refused to accept assignment in all cases.

The "Mandatory Assignment" proposal will undoubtedly arise in Congress once again, as will proposals to extend the DRG program to cover physicians' services. If you, who feel so strongly about government control of medical practice, do not support the AMA, who will? And if not the AMA, who will speak for you?

Closer to home, the same argument prevails for MSSNY membership as your best means of obtaining further reform in the medical liability insurance system. While it is obvious that the problem is by no means solved, significant progress was made this year. I cannot understand why any member would choose this, of all times, to resign from MSSNY. Surely the 23% drop in the retroactive premium increase for 1984-1985 is worth many times more than the investment in society dues.

We have the governor's assurance that the legislation passed this summer is just the first step, and that he will continue to work on a better solution for introduction this fall. However, politics is politics, and if there was ever a time to demonstrate your solidarity with your colleagues in demanding a permanent resolution of this problem, it is now.

I urge you, therefore, to reconsider your decision to resign. Of course we don't win every battle, but think for a moment where physicians would be today if they didn't have their medical societies defending their interests.

GEORGE J. LAWRENCE, JR., MD
Executive Vice-President
Medical Society of the
State of New York
420 Lakeville Rd
Lake Success, NY 11042

Quantitative Doppler spectrum analysis of extracranial carotid artery stenosis

MALCOLM O. PERRY, MD, MICHAEL F. SILANE, MD, DAVID CALCAGNO, MD, SUSAN SPRADLIN

ABSTRACT. The clinical evaluation of suspected extracranial carotid artery occlusive disease is often inaccurate. This has led to a search for reliable noninvasive methods of examining these vessels. A blind comparison was made between spectrum analysis of continuous wave Doppler recordings from 155 carotid artery bifurcations and contrast arteriograms. There was overall agreement in 88%, but sensitivity to internal carotid arteries with severe stenoses was 92%, and to occlusions, 94%. Thirty-one of 34 true negative findings were identified (91% specificity).

The evaluation of asymptomatic carotid bruits, and the regular follow-up of patients who have undergone carotid surgery usually can be accomplished with Doppler spectrum analysis. Patients with specific symptoms are referred for arteriography.

(NY State J Med 1985; 85:577–580)

INTRODUCTION

Spectrum analysis of continuous wave Doppler recordings from the carotid bifurcation has proved useful in the assessment of carotid artery stenosis.[1–4] Although the ultrasonic duplex scanner that combines pulsed Doppler spectrum analysis and B-mode imaging may be a more accurate noninvasive method for the diagnosis of carotid bifurcation disease, the continuous wave Doppler technique, when used with real-time spectrum analysis, offers a useful and cost effective technique for screening patients with suspected carotid artery disease.

Previously reported studies have suggested that more than 90% of stenoses that cause significant hemodynamic changes can be detected with this technique.[5] Although continuous wave Doppler systems are not inexpensive, they cost approximately one fifth as much as the pulsed Doppler systems combined with B-mode scanners. In this study real-time spectrum analysis was used to classify carotid bifurcation disease in 155 consecutive carotid arteries examined by contrast arteriography.

PATIENTS AND METHODS

Direct Doppler examinations for suspected occlusive disease of the common carotid, internal carotid, and external carotid artery were performed in 155 carotid arteries that also were examined with arteriography. The patient population consisted of 47 men and 33 women who ranged in age from 38 to 76 years. Most of the patients were referred for evaluation of suspected cerebrovascular insufficiency,(52) the detection of a cervical bruit,(19) or because of multifocal atherosclerosis detected in other areas of the body.(9) Prior to angiography, the Doppler examinations were performed in the vascular laboratory by a noninvasive vascular technologist and were supervised by a vascular surgeon.

All the arteriograms were evaluated separately in a blind manner by the senior author, and measurements of the degree of diameter reduction were performed independently by the method described by DeWeese et al.[6] The most severe diameter reduction seen on the arteriogram was measured against the normal appearing internal carotid artery diameter distal to the stenosis. Usually this is best seen in the lateral films of the neck, but occasionally the anterior-posterior or oblique views are more revealing. Blind interpretation of the real-time spectrum analysis studies were also performed by the vascular technologist and independently by the vascular surgeon; the results were compared after all measurements had been recorded separately.

Technique. The method of direct carotid bifurcation examination employed in this study is similar to that described by Barnes and his associates[2] and by Bandyk et al.[1] The patients are placed in the supine position and are asked to rotate the head 30 degrees to the side opposite from that being studied. A hand-held 8 mHz directional Doppler probe is positioned over the common carotid artery low in the neck and is coupled acoustically to the skin with gel. The probe is then angled cephalad at 45 to 60 degrees to the skin surface to obtain the wave-form with the highest frequency. The probe is moved cephalad along the course of the common carotid artery until the carotid bifurcation is reached. The internal and external carotid arteries are identified by the audio and visual characteristics of the Doppler wave form. The internal carotid artery is followed cephalad as far as the signals can be elicited, and the external carotid artery is found by medial angulation and also followed toward the mandible as far as possible.

The signals of the continuous wave Doppler detector are processed by a real-time, fast Fourier transform spectrum analyzer and are displayed on a video monitor. The freeze-form mode on the video monitor is used to examine the wave form characteristics. A permanent record of the findings are obtained by self-developing photographs taken from the video monitor.

Interpretation of spectrum analysis. At the carotid bifurcation the normal Doppler spectrum consists of a systolic peak fre-

---

From the Division of Vascular Surgery, Department of Surgery, Cornell University Medical College, New York.

Address correspondence to Dr Perry, Division of Vascular Surgery, Department of Surgery, Cornell University Medical College, 1300 York Ave, F-2011, New York, NY 10021.

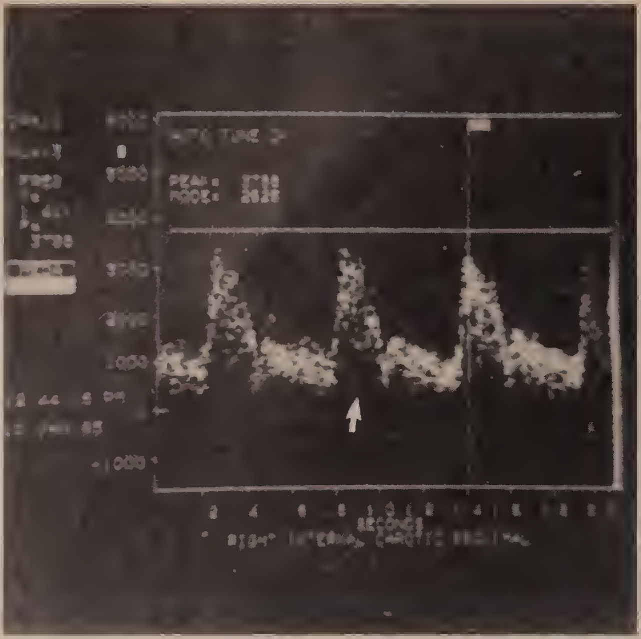


FIGURE 1. The spectral analysis obtained from a normal internal carotid artery. Low peak frequencies and a clear systolic window are seen (arrow).

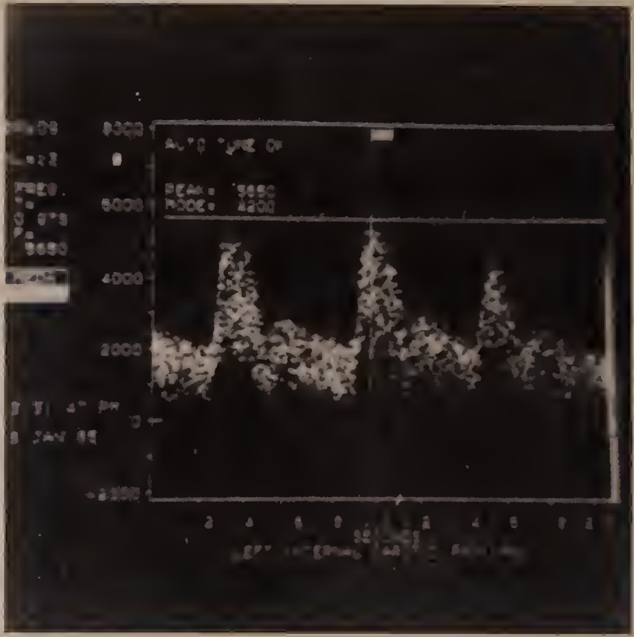


FIGURE 2. Mild stenosis at the internal carotid artery results in a small increase in the peak systolic frequency; some spectral broadening is present.

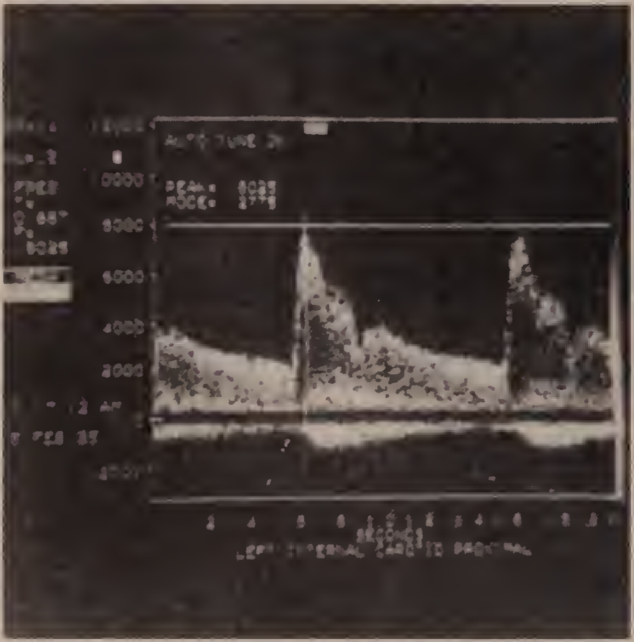


FIGURE 3. A moderate internal carotid artery stenosis changes the spectra by increasing significantly the peak frequencies and producing more spectral broadening. The systolic window is now filled in.

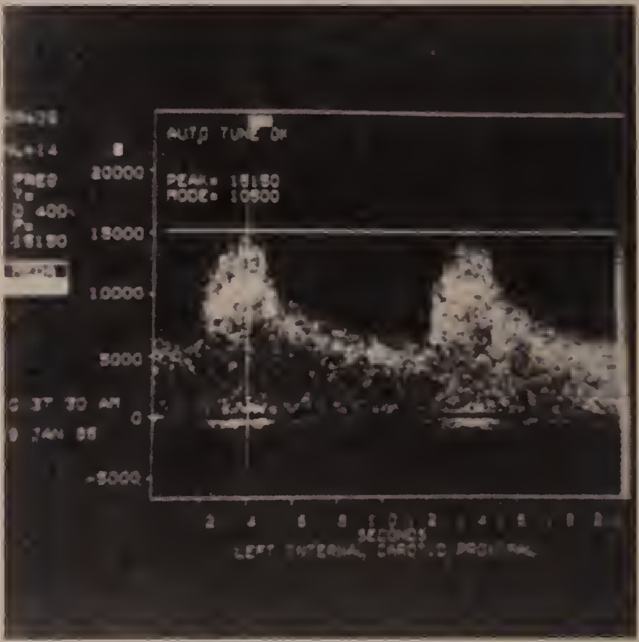


FIGURE 4. The high peak frequency and marked spectral broadening are striking in this photograph obtained from a patient with severe internal carotid artery stenosis.

quency up to 4 kHz accompanied by a narrow range of frequencies during systole which result in a clear area in the spectrum wave form just beneath the systolic peak (systolic window) (Fig 1). As shown in Figure 2, as the arterial stenosis increases in severity, flow is disturbed in the region immediately distal to the stenosis; this produces spectral broadening during systole. As the stenosis increases further the clear area underneath the systolic peak is filled in completely (no systolic window) (Fig 3). The systolic peak frequency increases with increasing severity of the stenosis; the changes in peak systolic and diastolic frequencies are measured at the site of stenosis (Fig 4).

In the presence of an occlusion of the internal carotid artery the Doppler flow signal may not be elicited in the usual position, and the signals from the common carotid artery will no longer exhibit prograde flow during diastole (Fig 5). In some cases contralateral carotid bifurcation flow may be increased in the presence of internal carotid artery occlusion, perhaps reflecting compensatory flow via collateral arteries. The characteristics of increasing stenosis as seen in the velocity spectra are described in Table I.

TABLE 1. Categories of Occlusive Disease in the Internal Carotid Artery

Arteriogram	Spectra
Normal	ICA* systolic peak frequency <4 kHz ICA to CCA† systolic peak frequency ratio <1.5 Systolic window present
Mild (20%–50% diameter reduction)	ICA systolic peak frequency ≤4 kHz Systolic spectral broadening present
Moderate (50%–80% diameter reduction)	ICA systolic peak frequency >4 kHz No systolic window Increase in ICA diastolic frequency
Severe (>80% diameter reduction)	ICA systolic peak frequency >10 kHz ICA diastolic peak frequency >4 kHz
Occlusion	No ICA flow signal No prograde flow in CCA during diastole

Modified from Bandyk et al.[1]

* ICA, internal carotid artery; CCA, common carotid artery.

RESULTS

The severity of the disease in the internal carotid artery as shown by arteriography is divided into mild stenosis (20% to 50% diameter reduction), moderate stenosis (50% to 80% diameter reduction), severe stenosis (greater than 80% diameter reduction), and occlusion.[1] The results of disease classification using quantitative Doppler spectrum analysis compared with contrast arteriography are shown in Table II. Among patients with lesions that altered hemodynamic variables (greater than 50% diameter reduction) there was agreement with arteriography in 88%, but sensitivity to arteries with severe stenoses (greater than 80% diameter reduction) was 92% (36 of 39). The sensitivity to occlusion of the internal carotid artery was 94% (17 of 18). The specificity of the test was 91% (31 of 34 true negative studies).

DISCUSSION

The clinical evaluation of suspected extracranial carotid artery atherosclerotic disease is often inconclusive. Distal internal carotid pulses are not accessible for examination. The presence of bruits heard over the carotid bifurcation is an indication of turbulence in blood flow, and may be a marker of arterial stenosis. This finding is nonspecific, and unreliable, because approximately one third of the patients with significant arterial occlusive lesions that have been identified by arteriography have no bruit.[2] Moreover, almost a third of the patients with bruits heard over the bifurcation will have no significant arterial lesions detected by arteriography. These confusing features have led to a search for more reliable noninvasive techniques.

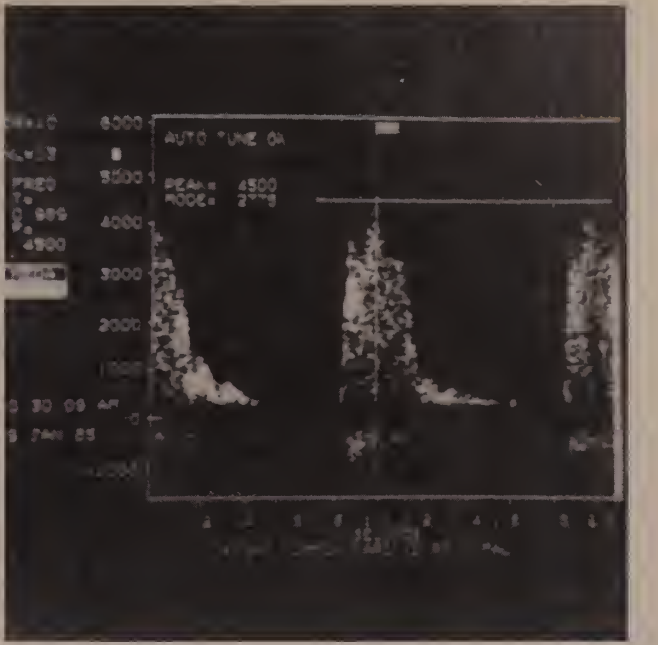


FIGURE 5. No internal carotid artery Doppler signal is elicited in this patient with occlusion of the artery. Also of diagnostic importance is the lack of prograde flow in the common carotid artery during diastole.

The most common indirect diagnostic method for evaluation of the carotid system is oculoplethysmography—either a method of measuring pressure in the retinal vessels or detecting delays in pulse wave transmission in the ophthalmic artery distribution.[7,8] These tests are useful mainly for exposing tight stenoses or occlusions in the internal carotid and ophthalmic arteries.

Direct methods include B-mode imaging, carotid waveform analysis, and real-time Doppler spectrum analysis.[1,2,9,10] The most sophisticated of the instruments currently in use is the duplex scanner—a machine that combines B-mode imaging and pulsed-wave Doppler spectrum analysis.[4] This requires equipment that costs nearly $100,000, but it clearly is an excellent method.

Continuous wave Doppler techniques for evaluating the carotid bifurcation spectra are useful as well, and recent studies demonstrate that more than 90% of arterial lesions in the internal and external carotid arteries that result in significant hemodynamic changes can be detected.[1]

Several important maneuvers are necessary to achieve acceptable results in screening tests performed with continuous wave Doppler systems. Not all these instruments are the same, and each diagnostic laboratory must vali-

TABLE II. Comparison of the Findings Obtained with Arteriography and Doppler Spectral Analysis (155 Internal Carotid Arteries)

Results of Arteriogram (Number)	Results of Doppler Spectrum Analysis				
	Normal	Mild	Moderate	Severe	Occlusion
Normal (39)	36	2	1	0	0
Mild (38)	5	30	3	0	0
Moderate (29)	1	5	21	2	0
Severe (31)	0	0	2	27	2
Occlusion (18)	0	1	0	0	17

date their methods and results.[10] The data presented in this report were acquired for that reason.

The changes in the Doppler spectra described in this study have proven to be reliable in screening patients for carotid bifurcation occlusive disease. As Barnes et al,[2] and Bandyk and his associates[1] emphasized, several features are more important when this method is used. Loss of the systolic window, increasing systolic and diastolic peak frequencies, and spectral broadening are the major changes used to detect lesions. When properly interpreted, keeping in mind the specific characteristics of the particular instrument being used, this test is quite useful as a screening procedure.

Patients with transient attacks of cerebral ischemia, previous strokes, or other problems in the carotid system require arteriography. Our experience strongly suggests that noninvasive techniques as described in this report are at least as useful as digital intravenous arteriography in evaluating carotid bifurcation occlusive disease. Such angiograms tend to be hampered by technical and procedural problems: patient movement, swallowing, superimposition of images, and computer artifacts. Conventional selective arteriography with or without digital computer assistance is our preferred mode of study in all patients with symptoms that suggest occlusion.

The evaluation of asymptomatic carotid bruits and the regular follow-up of patients who have undergone carotid surgery are often satisfactorily achieved with continuous wave Doppler spectrum analysis. This direct method can be enhanced by combining it with indirect techniques which are relatively insensitive, but highly specific for occlusion or a tight stenosis of the internal carotid artery (oculoplethysmography). Alterations in the tests that suggest progression of disease, or the appearance of new lesions, require arteriograms for final evaluation. Despite some reports to the contrary, in our experience, careful performance and inspection of biplanar carotid arteriograms yield a high correlation with the morbid findings at surgery.

Acknowledgments. The authors are grateful to Marian Napoletano for preparation and review of the manuscript, the tables, and the illustrations.

REFERENCES

1. Bandyk DF, Levine AW, Pohl L, Towne JB: Classification of carotid bifurcation disease using quantitative Doppler spectrum analysis. *Arch Surg* 1985; 120:306-313.
2. Barnes RW, Rittgers SE, Putney WW: Real-time Doppler spectrum analysis. Predictive value in defining operable carotid artery disease. *Arch Surg* 1982; 117:52-57.
3. Blackshear WM Jr, Phillips DJ, Thiele BL, et al: Detection of carotid occlusive disease by ultrasonic imaging and pulsed Doppler spectrum analysis. *Surgery* 1979; 86:698-706.
4. Blackshear WM Jr, Lamb SL, Kollipara VS, et al: Correlation of hemodynamically significant internal carotid stenosis with pulsed Doppler frequency analysis. *Ann Surg* 1984; 199:475-481.
5. Breslau PJ, Knox RA, Phillips DJ, Strandness DE: Ultrasonic duplex scanning with spectral analysis in extracranial carotid artery disease: comparison with contrast arteriography. *Vasc Diag Ther* 1982; 3:17-22.
6. DeWeese JA, May AG, Lipchik WO: Anatomic and hemodynamic correlations in carotid artery stenosis. *Stroke* 1970; 1:49-57.
7. Gee W, Mehigan JT, Wylie EJ: Measurement of collateral cerebral hemispheric blood pressure by ocular pneumoplethysmography. *Am J Surg* 1975; 130:121-127.
8. Kartchner MM, McRae LP, Morrison FD: Noninvasive detection and evaluation of carotid occlusive disease. *Arch Surg* 1973; 106:528-535.
9. Rutherford RB, Hiatt WR, Kreutzer GW: The use of velocity waveform analysis in the diagnosis of carotid artery occlusive disease. *Surgery* 1976; 80:328-335.
10. O'Donnell TF Jr, Parker SG, Callow AD, Kelly JJ, McBride KJ, Korwin S: The relative value of carotid noninvasive testing as determined by receiver operator characteristic curves. *Surgery* 1980; 87:9-19.

Use of gonadotropins in ovulation induction

Analysis of treatment protocols and pregnancy outcome

PAUL R. GINDOFF, MD, RAPHAEL JEWELEWICZ, MD

ABSTRACT. Experience with gonadotropin therapy using menotropins and human chorionic gonadotropin is reviewed for 51 patients with respect to pregnancy outcome, preovulatory estradiol level, days of therapy, number of cycles required for conception, and number of ampules of menotropins used. Overall success, regardless of underlying diagnosis, was 41%. The spontaneous abortion rate was 41%—noticeably higher than that occurring in the general population of spontaneously conceived pregnancies (20%-25%). Overall, 59% of all the conceptions resulted in viable offspring. The conception rate per cycle was substantially higher for the group with estradiol levels less than 1,500 pg/mL. The rate of spontaneous abortions tended to fall and that of term singleton pregnancies tended to rise at this level. The span of 10 to 15 days for menotropin administration proved more successful in obtaining conception. Successful conceptions usually occurred early in the treatment cycles regardless of outcome. In general, fewer ampules were required to induce conception when the pregnancy ended in spontaneous abortions rather than in viable offspring.

(NY State J Med 1985; 85:580-584)

From the Department of Obstetrics and Gynecology, The Sloane Hospital for Women at Columbia-Presbyterian Medical Center, New York.

Address correspondence to Dr Gindoff, Fellow, Division of Reproductive Endocrinology and Infertility, Department of Obstetrics and Gynecology, The Sloane Hospital for Women at Columbia-Presbyterian Medical Center, 630 W 168th St, New York, NY 10032.

INTRODUCTION

In the late 1950s a new era in therapy and hope for infertile women was initiated by Gemzell et al[1] in Sweden when he described the use of follicle stimulating hormone (FSH) derived from pituitary glands, followed by human chorionic gonadotropin (HCG) to induce ovulation in a large percentage of infertile and amenorrheic women. Lunenfeld and colleagues[2] in Israel and Rosemberg et al[3,4] in the United States isolated gonadotropic substances from the urine of postmenopausal women which, when followed with HCG, were capable of ovulation induction. Human menopausal gonadotropin (HMG) is available as a lyophilized form of follicle stimulating hormone and luteinizing hormone (LH) as the commercial preparation Pergonal® used in this center.

Infertility due to lack of ovulation is the chief indication for therapy with HMG. Best responders are those patients having persistently low or absent gonadotropins. This group, as classified by the World Health Organization and by Insler et al,[5,6] is comprised of women with hypogonadotropic hypogonadism, and primary and secondary amenorrhea. Patients with normal gonadotropin levels and some endogenous estrogen activity, typically with oligo-ovulatory disorders, respond less well to HMG therapy.[6-9] Treatment with HMG during the past 25 years has proven to be successful but fraught with the complications of an increase in spontaneous abortion, multiple gestation, and various side effects including the hyperstimulation syndrome which can be life-threatening. The spontaneous abortion rate obtained in this study (41%) is in fact greater than most of those reported in either normal or subfertile populations.

This study reviews all HMG treatment cycles in the private office of one of the investigators (RJ) in the year 1982. The data are analyzed with respect to the factors influencing the rate of spontaneous abortion, multiple birth, singleton conceptions, and hyperstimulation. Attention is directed to the influence of variations in the administration of HMG, pre-ovulatory estradiol levels, and possible causes for the observed outcomes.

PATIENTS AND METHODS

Fifty-one patients were treated during 190 cycles with Pergonal® during the year 1982 in the private offices of one of the investigators. The results of all semen analysis and post-coital tests were within normal limits. The patients were divided into two groups: group A representing the responders to HMG as evidenced by achieving conception, and group B representing the non-pregnant patients or non-responders (Table I). The patients were further subclassified according to endocrine diagnosis (Table II) including oligo-ovulation, anovulation, amenorrhea-galactorrhea, amenorrhea, poor corpus luteum function, and unexplained infertility.

TABLE I. Overall Pregnancy Rate Among 51 Patients

	No. of Women	Pergonal Cycles	Hyper-stimulation	Concep-tions	(% cycles) (% pts)
Pregnant patients	21	53	3	22	(41%) (41%)
Non-Pregnant patients	30	137	1	—	— —

TABLE II. Endocrinologic Diagnosis

	No. of Women in Pregnancy Group A	No. of Women in Non-Pregnancy Group B
Oligo-ovulation	15	16
Anovulation	3	6
Amenorrhea-galactorrhea	1	—
Amenorrhea	1	4
Poor corpus luteum function	1	3
Unexplained infertility	—	1
Total	21	30

Fifteen patients in group A and 16 in group B were classified as having oligo-ovulation disorders; three in group A and six in group B had anovulation disorders; amenorrhea associated with galactorrhea was diagnosed in one patient in group A; amenorrhea was found in one patient in group A and four in group B. Poor corpus luteum function was the cause of infertility in one patient in group A and three patients in group B, whereas unexplained infertility accounted for one patient in group B. Treatment with clomiphene was unsuccessful in four of the patients in group A. These patients failed to experience ovulation at progressively increasing doses of clomiphene, reaching 200 mg orally a day for five consecutive days on day five to nine of the menstrual cycle. One patient in group A with oligo-ovulation was noted to have polycystic ovaries, and another such patient had galactorrhea. This latter patient had undergone previous successful treatment with HMG. The patient in group A with amenorrhea-galactorrhea did not respond to bromocriptine management. The patients in group B had diverse problems: two patients had endometriosis; three were subsequently shown to have tubal disorders; two had galactorrhea; two had experienced poor response to clomiphene therapy; four had previously conceived following HMG therapy but are now unresponsive to the treatment; and one patient was subsequently shown to have elevated gonadotropin levels associated with anovulation.

Pergonal® was used as the HMG preparation in all patients. The administration schedule was individualized for each patient. A total of 51 patients received HMG in 190 cycles; ovulation was induced with a single dose of 10,000 IU of HCG. On occasion, if ovulation did not occur, an additional 5,000 IU of HCG was given on the third or fourth day after the initial injection. All patients were initially started on at least two ampules of HMG daily. Each ampule contains 75 IU of FSH and 75 IU of LH. The dose of HMG was adjusted periodically depending on the apparent clinical response. Patients received a range of 10 to 140 ampules, two to seven ampules daily, and were treated from 5 to 23 days during a single course of treatment. Ovulation was detected by monitoring with biphasic temperature charts, with late luteal serum progesterone levels, or with loss of cervical mucus ferning.

The degree of follicular development and/or gonadal stimulation was evaluated by the physiologic change in the cervical mucus at frequent intervals.[10] In addition, plasma estradiol measurements were monitored until follicular maturation was achieved, as indicated by plasma levels between 500 to 1,500 pg/mL.[9] Pre-ovulatory levels ranged from 263 to 9,500 pg/mL. Several investigators[11] have monitored treatment based on cervical mucus alone or in combination with estrogen excretion,[12] but monitoring ovarian response by measurement of plasma estrogen appears to be a more reliable index of follicular maturation and can reduce the complication rate of hyperstimulation. Radioimmunoassay was the technique used to measure estradiol at this institution; it is readily available and easy to perform.

RESULTS

Among the 51 women treated for infertility, 21 (41%) conceived, yielding 22 actual conceptions (Table I). One patient had two cycles

TABLE III. Individual Patient Pergonal Cycle Data

Conception Number	No. Days In Conception Cycle	No. Ampules Used in Conception Cycle	Pre-ovulatory Estradiol Levels
Singleton Births			
1	18	34	2,700
2	12	45	570
3	8	24	541
4	15	140	951
5	12	24	1,125
6	12	60	—
7	8	16	1,250
8	12	27	281
Multiple Births			
9	—	—	929
10	14	52	1,375
11	14	49	917
12	—	—	9,500
13	17	33	1,250
Spontaneous Abortions			
14	11	22	4,600
15	7	14	910
16	8	16	3,025
17	—	80	1,141
18	8	16	1,234
19	13	42	1,225
20	14	28	790
21	13	24	—
22	12	38	1,100

of HMG-HCG, with each culminating in a spontaneous abortion. Ovulation was induced in every treatment cycle by administration of HCG, usually in the dose of 10,000 IU IM. There were 53 cycles of HMG-HCG among the 21 women who conceived, yielding a 41% conception/cycle rate, and 137 cycles were administered for the non-pregnant group. Pregnancies that produced viable offspring accounted for 13 of the 22 conceptions or 59% of all conceptions. There were nine (41%) spontaneous abortions (including one molar pregnancy), eight (36%) singleton pregnancies that went to full term, and five (23%) multiple pregnancies (only one set of which ended in premature labor and delivery). The results of the two patient groups can be further broken down according to endocrinologic diagnosis (Table II).

TABLE IV. Number of Treatment Cycles Required to Achieve Conception

Cycle Conceived	No. of Conceptions	Viable Pregnancies	Spontaneous Abortions
1	14	7	7
2	5	5	—
3	—	—	—
4	1	—	1
5	1	1	—
6	1	—	1

TABLE V. Days of Pergonal Treatment Required to Attain Sufficient Ovarian Stimulation*

Days	Conceptions	Spontaneous Abortions (% conception)	Singletons (% conception)	Multiple Gestations (% conception)
<10	5	3 (60%)	2 (40%)	—
10–15	13	5 (39%)	5 (39%)	3 (22%)
16–25	1	—	1 (100%)	—

* Three conceptions had to be withdrawn from this table because of incomplete data.

There were three cases of hyperstimulation in group A. In one patient subclassified as hypothalamic amenorrhea without galactorrhea mild ovarian enlargement (<10 cm) developed—no ascites accumulated. Her pre-ovulatory estradiol level was 1,125 pg/mL. The second patient had a pre-ovulatory estradiol level of 9,500 pg/mL; mild hyperstimulation developed with ovarian enlargement less than 10 cm. She had amenorrhea-galactorrhea, had been unresponsive to bromocriptine treatment, and had undergone a successful trans-sphenoidal hypophysectomy. These two patients had successful outcomes to their HMG cycle, resulting in a singleton birth and a multiple birth, respectively. The third patient with hyperstimulation, which was also mild, suffered a spontaneous abortion. All three patients had been given 24 ampules of HMG and had been treated over 8 to 12 days. The hyperstimulation rate for group A was 3/21 or 13%—slightly higher than reported elsewhere. Further descriptive analysis of the patients in group A is shown in Table III.

In general, patients who did respond to HMG and achieve conception did so within the first few cycles (Table IV). While conceptions did occur in the earlier cycles, patients can still respond after multiple cycles, as shown here and in previous reports.[7] There was not a marked difference in the number of cycles required to achieve a pregnancy when viable pregnancies versus spontaneous abortions were compared.

The time span during which HMG is administered was analyzed. In previous work[7] it was shown that a span of 10 to 15 days proved optimal for induction of ovulation. This was also the case in this group of patients (Table V). Only one conception occurred after 15 days of treatment, and of the remaining 18 conceptions, 72% occurred in the 10 to 15 day period and 28% occurred earlier than 10 days (72% is significantly larger than 28% by a one-tailed binomial test, $P < .05$). Prior work[7] has also shown that a span of 10 to 15 days yielded a higher incidence of multiple pregnancies. Consistent with this finding, the three multiple gestations in the present study were conceived using HMG in the intermediate span of 10 to 15 days.

TABLE VI. Pre-Ovulation Estradiol and Outcome of Pregnancy

Estradiol Levels	# of Cycles	Conceptions (% cycles)	Spontaneous Abortions (% conception)	Singleton (% conception)	Multiple (% conception)
<1500 pg/ml*	31	16*(52%)	6*(37.5%)	6*(37.5%)	4*(25%)
>1500 pg/ml	16	4(25%)	2(50%)	1(25%)	1(25%)

* Two conceptions (#6, #21) were removed from this analysis since estradiol levels were not available for the conception cycle. In addition, four additional cycles had incomplete data, making the total number of cycles 47 for this table.

Table VI shows the extent of ovarian stimulation as judged by estrogen levels in the plasma in relation to pregnancy outcome. The first group represents levels less than 1,500 pg/mL (and greater than 500 pg/mL with the exception of patient 8 whose level was less than 500 pg/mL) which will empirically represent ovarian stimulation optimal for induction of ovulation. The other group represents stimulated ovaries with estradiol levels greater than 1,500 pg/mL. The overall conception rate per cycle was significantly higher for the group with average (lower) estradiol levels (0.52 versus 0.25; $z = 1.75$; $P < .05$ for a one-tailed test). The spontaneous abortion rate tended to be lower and the rate of singleton conceptions tended to be higher for this group—a finding reported earlier.[7,13]

Group B represents the 30 non-pregnant patients that were treated with HMG for a total of 137 cycles. The various factors responsible for the infertility in these patients could not be adequately corrected by ovulation induction. Six of the 30 patients that did not respond to HMG therapy had either tubal or peritoneal disorders. Four of the patients in group B who had previously conceived following HMG therapy now presented with apparently multifactorial causes for their infertility.

Finally, a comparison of the amount of gonadotropins used in each successful treatment cycle revealed that the mean number of ampules/cycle used was higher for the viable gestations as compared to the non-viable gestations: multiple gestations required an average of 45 ampules/successful cycle and singleton gestations required 46 ampules/cycle, whereas conceptions ending in spontaneous abortion required 31 ampules/cycle on the average. Although the mean difference in ampules/cycle is substantial, the difference between these means failed to meet traditional levels of statistical significance because of the large variation in ampules/cycle (variance = 842.5).

DISCUSSION

The results of ovulation induction with Pergonal® from several centers display a broad range. Pregnancy rates as low as 16.1% and as high as 58.6% have been reported with conceptions/total cycles reported to range from (8.08% to 27.9%).[7,14] In this study the individual pregnancy rate was 41%, with 41% of the cycles in group A patients culminating in conception. The overall conception rate/total cycles administered was 22/190 or 12%. It is difficult to adequately compare results of Pergonal® therapy among various investigators because of differences in patient selection and guidelines for HMG-HCG administration. In group A there were several patients who had been nonresponsive to clomiphene therapy or had been found to have polycystic ovarian syndrome. These patients are known to respond less well to HMG induction than a patient with amenorrhea and anovulation with low endogenous estrogen levels.[6–9] Whereas among patients in group B, the causes of infertility were often multiple, and therefore could not be corrected by HMG administration alone.

The spontaneous abortion rate was 41%—which is considerably higher than in spontaneous conceptions or in many subfertile populations. Ovulation that is induced by exogenous gonadotropin may produce an ovum that is intrinsically faulty and ill-prepared for appropriate fertilization and division. This theory is supported by failures in in vitro fertilization among patients who theoretically have no ovarian disorders and who have been treated with HMG. Explanations for failures in patients with anovulation/oligo-ovulation and polycystic ovarian syndrome may be due to high levels of androgen derivatives in the local environment of the developing follicle or to the presence of many follicles at various stages of development and subsequent superovulation of immature ova which will lead to complete failure of fertilization or set up an increased incidence of spontaneous abortion. In addition, these immature ova may be ovulated from physiologic immature or ill-prepared follicles whose corpus lutea can not support implantation.

The majority of patients who did respond to HMG did so within the first cycle. Certain patients—all of whom were classified as having oligo-ovulation—required four cycles, five cycles, and six cycles. This indicates that one should not consider HMG treatment unsuccessful after just a few cycles. One should continue therapy until pregnancy occurs if no other complicating factors are detected. There was not a significant difference in the number of cycles required to achieve a viable versus non-viable pregnancy.

Although the number of ampules, on the average, used to induce a conception cycle were roughly equivalent for the singleton and multiple gestation subgroups, the number was larger than that found for the subgroup whose pregnancies ended in spontaneous abortion. The length of HMG therapy seems to be more important in correlation with the outcome of pregnancy. The intermediate number of days (10 to 15), regardless of the number of ampules, seemed to correlate best with the physiologic state and yielded an increased number of conceptions. All the multiple gestations were in this group, and so length of treatment rather than number of ampules seemed to correlate with the type of viable gestation one can expect. A higher percentage of the conceptions achieved in the short cycle length (<10 days) ended in spontaneous abortion. This is probably related to premature induction of ovulation with HCG regardless of estradiol level. In fact, in these three patients the pre-ovulatory estradiol levels were 910, 3,025 and 1,234, respectively.

In the patients with high pre-ovulatory estradiol levels (greater than 1,500 pg/mL) the abortion rate tended to be higher and the singleton rate tended to be lower as compared to the group with lower levels of estradiol. Although the sample size for multiple gestations is small, others[15] have shown a direct relationship between the estradiol level and multiple pregnancy rates.

There were three cases of hyperstimulation among the group A patients (11%) and one case among the patients in group B. All incidences were mild, with ovarian enlargement less than 10 cm (about 5 × 5 cm), and no ascites or electrolyte imbalance was noted. The estradiol levels were all over 800 pg/mL, which correlates with a 90% incidence of hyperstimulation.[13] Ovarian hyperstimulation can be prevented by withholding HCG administration.

Since multiple pregnancy may not be adequately predicted by estradiol levels, it is recommended that adjunct evaluation of the physiologic response of the ovary be performed by ultrasound. This procedure may suggest the possibility of multiple pregnancy by detection of multiple follicles ready to ovulate.[16]

At the present, ultrasound is utilized in certain patients but not in all. Monitoring in this series was done by plasma estradiol levels exclusively. Ultrasound has been used in the last few years to assess follicular maturation and development. According to reports of spontaneous cycles,[17] the minimum size necessary for ovulation was 16 mm to 18 mm. Other authors[18] have shown that estradiol levels greater than 1,500 pg/mL may be associated with relatively immature follicles in certain patients.

Prior to ultrasound monitoring, HCG was administered when serum estradiol reached 500 to 1,500 pg/mL. With the use of sonography, it has become evident that multiple follicular development may account for an estradiol level in this range, but the individual follicles may all be small and immature. Therefore, HMG is administered in these individuals until the follicular size reaches a minimum of

18 mm. At this point, HCG is administered.

In addition, the dominant follicles which are induced following HMG stimulation are often smaller than those that occur during spontaneous cycles. Although multiple follicles will develop with HMG, not all follicles will become dominant or will mature. Interestingly, among women who are estrogen deficient, it is unusual to have more than two dominant follicles develop.[18]

With the use of ultrasound, one could follow not only follicular maturity to assess triggering with HCG at higher levels of estradiol but also evaluate the absolute number of mature follicles that develop. If multiple mature follicles do develop, HCG could be withheld to prevent potential multiple gestations.

In conclusion, the results indicate that ovulation induction is a successful procedure with an acceptable rate of hyperstimulation and abortion. Pergonal® was used as the gonadotropin preparation rather than FSH alone. The patients were monitored by clinical examination and serum estradiol levels. The endpoint of follicular maturation was the level of estrogens attained. LH was not measured as the endpoint to stop gonadotropin therapy. HCG was used as the ovulation triggering agent. Pre-ovulatory estradiol levels of less than 1,500 pg/mL and use of HMG for 10 to 15 days was shown to be significantly associated with conception success as compared to estradiol levels of more than 1,500 pg/mL and shorter courses of HMG. Proper selection of patients is necessary, but successes do occur even in repeated later cycles. In addition, the range of preovulatory estradiol levels and variation in dosage of Pergonal® used in this study imply that the development of hyperstimulation is as much an individual function of the particular patient as it is dependent on the absolute quantitative dosage of Pergonal® administered.

Acknowledgments. Dr Andrew Davidson provided statistical analysis and Ms Bambi Aharon helped in preparing the manuscript.

REFERENCES

1. Gemzell CA, Diczfalusy E, Tillinger G: Clinical effect of human pituitary follicle-stimulating hormone (FSH). *J Clin Endocrinol Metab* 1958; 18:1333-1348.
2. Lunenfeld B, Eshkol A, Donini P, Puzzudi D, Shelesnyack M: Studies on gonadotropins. Biological assessment of highly purified urinary extracts. *Harokeach Haivri* (Hebrew Pharmacist) *Sci Ed* 1963; 9:766.
3. Rosemberg E, Coleman J, Demany M, Garcia CR: Clinical effect of human urinary postmenopausal gonadotropin. *J Clin Endocrinol Metab* 1963; 23:181-190.
4. Rosemberg E, Maher RE, Stern A, Demany M: Clinical effect of gonadotropins of human origin. *J Clin Endocrinol Metab* 1964; 24:105-117.
5. Lunenfeld B, Insler V: Classification of amenorrhoeic states and their treatment by ovulation induction. *Clin Endocrinol* 1974; 3:223-237.
6. Insler V, Melmed H, Mashiah S, Monselise M, Lunenfeld B, Rabau E: Functional classification of patients selected for gonadotropic therapy. *Obstet Gynecol* 1968; 32:620-626.
7. Schwartz M, Jewelewicz R, Dyrenfurth I, Tropper P, Vande Wiele RL: The use of human menopausal and chorionic gonadotropins for induction of ovulation. *Am J Obstet Gynecol* 1980; 138:801-807.
8. Oelsner G, Serr DM, Mashiach S, Blankstein J, Snyder M, Lunenfeld B: The study of induction of ovulation with menotropins: analysis of results of 1897 treatment cycles. *Fertil Steril* 1978; 30:538-544.
9. Schwartz M, Jewelewicz R: The use of gonadotropins for induction of ovulation. *Fertil Steril* 1981; 35:3-12.
10. Spadoni LR, Cox DW, Smith DC: Use of human menopausal gonadotropin for the induction of ovulation. *Am J Obstet Gynecol* 1974; 120:988-993.
11. Taymor ML, Sturgis SH, Liberman BL, Goldstein DP: Induction of ovulation with human post-menopausal gonadotropin. *Fertil Steril* 1980; 33:387.
12. Taymor ML, Yussman MA, Gminski D: Estrogen monitoring in ovulation induction. *Fertil Steril* 1970; 21:759-762.
13. Jewelewicz R, Dyrenfurth I, Warren MP, Vande Wiele RL: Ovarian hyperstimulation syndrome. In: Rosenberg E (ed): *Gonadotropin Therapy in Female Infertility*. Amsterdam, Excerpta Medica, 1973, pp 235-245.
14. Tyler ET: Treatment of anovulation with metrotropins. *JAMA* 1968; 205:86-92.
15. Gemzell C, Roos P: Pregnancies following treatment with human gonadotropins. *Am J Obstet Gynecol* 1966; 94:490-496.
16. Smith DH, Picker RH, Sinosich M, Saunders DM: Assessment of ovulation by ultrasound and estradiol levels during spontaneous and induced cycles. *Fertil Steril* 1980; 33:387-390.
17. Hackelöer BJ, Fleming R, Robinson HP, Adam AH, Coutts JR: Correlation of ultrasonic and endocrinologic assessments of human follicular development. *Am J Obstet Gynecol* 1979; 135:122-128.
18. Marrs RP, Vargyas JM, March CM: Correlation of ultrasonic and endocrinologic measurements in human menopausal gonadotropin therapy. *Am J Obstet Gynecol* 1983; 145:417-421.

WHY KEEP MEDICAL RECORDS?

A physician licensed to practice medicine in New York State had treated Medicaid patients at nursing homes in Suffolk County since the early 1970s. A random audit of his records and Medicaid billings for the period January 1, 1979 through May 31, 1981 ultimately led to the determination that the physician's medical recordkeeping was so "completely and utterly deficient" that permanent disqualification from participating in the Medical Assistance Program was called for.

The Supreme Court Appellate Division, Third Department, in reviewing the administrative determination barring the physician permanently from the Medicaid program, found that the records "revealed nothing of the patients' general condition, chief complaints, symptoms, or any significant physical examination findings or therapy plans."

Although the court agreed that there was no indication that fraud was intended and that the physician was not guilty of moral turpitude, the court nevertheless ruled that disqualification from the Medicaid program was an appropriate penalty because the physician's records were so "monumentally deficient." The maintenance of proper records is a basic condition for participating in the State's Medical Assistance Program [18 NYCRR 540.7 (a)(8)].

Matter of Triant versus Perales, 491 NYS 2d 486 (A.D. 3d Dept., July 11 1985).

A 28-year-old man with a left-sided palatal mass

CASE PRESENTATION

Thomas Little, MD (Chief Medical Resident): A 28-year-old man was admitted for evaluation of a left-sided palatal mass. Four weeks earlier, the patient had been given penicillin for buccal and palatal swelling of the left upper oral cavity. Ten days later, the left third molar was extracted and an adjacent papular lesion on the hard palate was incised without purulent drainage. Continued pain and enlargement of the palatal lesion led to admission.

Past medical history included pneumonia in childhood and tuberculosis in 1977, treated with isoniazid and pyridoxine for one year. He smoked ten cigarettes daily for many years, and had previously been admitted to the hospital for alcohol detoxification. He had served in the Army in California, but had not traveled outside of the United States; he denied exposure to industrial toxins. His family history was unremarkable.

Physical examination revealed a well developed, well nourished man in no distress. His blood pressure was 126/94 mm Hg; respiratory rate, 20/min; pulse rate, 140/min; and oral temperature, 98.8° F. His pupils were normally reactive; the extraocular movements were normal. The tympanic membranes were normal. The left side of the palate was distorted by a 3 × 3.5 cm ulcerated mass that protruded into the oral cavity and was surrounded by edematous mucosa. Trismus was present. The nasopharynx and larynx were normal. There were no masses, thyroid enlargement, or adenopathy in the neck region. The lungs and heart were normal on examination. The abdomen contained no palpable masses, tenderness, or enlarged organs. The genitalia were normal. The extremities showed no clubbing, cyanosis, or edema. Neurologic examination revealed no focal deficits.

Urinalysis revealed a specific gravity of 1.026 with a one-plus positive test for protein and only rare leukocytes on smear. The hemoglobin was 14.6 grams/dL, and the white blood cell count was 8,400/mm^3, with 52% neutrophils, 1% band forms, 37% lymphocytes, 5% monocytes, 3% eosinophils, and 2% basophils. The platelet count was 487,000/mm^3. The serum levels of sodium, potassium, chloride, bicarbonate, glucose, creatinine, calcium, and bilirubin were within normal limits. The blood urea nitrogen was 2 mg/dL; serum uric acid, 8.5 mg/dL (normal range, 3.9-9.0 mg/dL); serum alkaline phosphatase, 155 units/L (normal range, 30-115 units/L); serum lactic dehydrogenase, 216 units/mL (normal range, 100-225 units/mL); and the aspartate aminotransferase (AST), 51 units/mL (normal range, 10-50 units/mL). The serum vitamin B_{12} level was 310 pg/mL (normal range, 200-900 pg/mL); serum folic acid, 3.4 ng/dL (normal range, 4-16 ng/dL); serum T4, 11.7 μg/dL (normal range, 4.5-11.5 μg/dL); and resin T3 uptake, 25% (normal range, 25%-35%). Serum antibodies to hepatitis B surface antigen and core antibody were absent.

The electrocardiogram demonstrated left ventricular enlargement and the chest radiograph was normal. Radiographs of the paranasal sinuses demonstrated opacification of the left maxillary sinus (Fig 1). A computerized tomographic (CT) scan showed an enhancing mass in the left palate, extending posteriorly to the adenoid region and containing several low density areas (Fig 2). The mass filled the maxillary sinus, causing erosion and bowing of the medial wall, and extended into the floor of the left orbit.

Two biopsies of the lesion were made. The initial specimen revealed necrotic tissue and a small focus of well-differentiated nonkeratinizing squamous cell carcinoma. The second biopsy contained necrotic tissue without malignant cells, prompting the pathologist to recommend an additional biopsy. After review of the biopsy specimens, a pathology consultant suggested the possible diagnosis of amelanotic melanoma. Cisplatin (120 mg/m^2 of body surface) was given with intravenous hydration and mannitol. Five days later oliguria developed, with elevation of serum creatinine to 5.2 mg/dL (normal range, 0.7-1.4 mg/dL), and blood urea nitrogen to 33 mg/dL. Urinalysis at that time showed a specific gravity of 1.007, a one-plus positive test for protein, a one-plus positive test for hemoglobin, and a few leukocytes and granular casts on smear of the urine sediment. The serum creatine phosphokinase (CPK) activity was increased at 182 units/mL (normal range, 2-82 units/mL), with a 100% MM isoenzyme fraction. The serum protein electrophoresis was normal.

The patient's renal failure was attributed to cisplatin therapy and was treated with both hemodialysis and peritoneal dialysis. Normal renal function returned within several days. The patient underwent a left maxillectomy and had an uncomplicated postoperative course. After being fitted with a prosthetic palate, he left the hospital on a weekend pass and did not return.

Based on the clinicopathologic conference (CPC) held at the Buffalo Veterans Administration Medical Center, Buffalo, NY, on February 2, 1984.

Case Presentation: Thomas Little, MD, Chief Medical Resident, *Radiology Presentation:* David M. Wiechec, MD, Attending Radiologist; *Housestaff Clinical Discussion:* Maria Julia Westerink, MD, Junior Medical Resident; *Attending's Clinical Discussion*: John Lore, MD, Professor and Chairman, Department of Otolaryngology, State University of New York at Buffalo School of Medicine; *Pathology Presentation:* Mario Montes, MD, Clinical Professor of Pathology, State University of New York at Buffalo School of Medicine (deceased).

Editor: JOHN T. FLYNN, MD

Address correspondence to Dr Flynn, Consulting Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

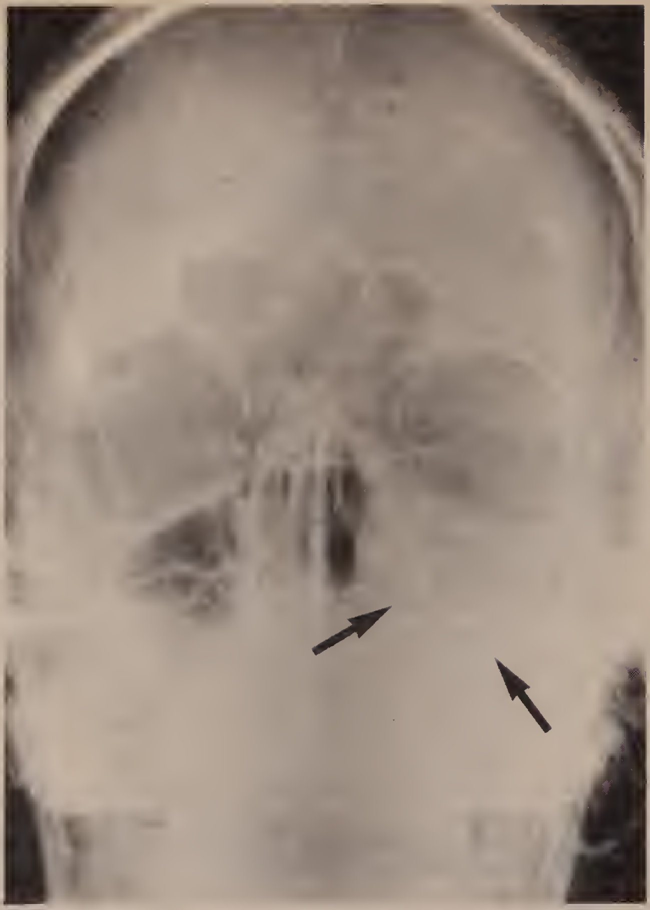


FIGURE 1. Modified (30°) Caldwell view demonstrating complete opacification of left maxillary sinus (arrows).

Radiology Presentation

David M. Wiechec, MD (Attending Radiologist): The chest film on admission showed blunting of the right costophrenic angle, a finding seen on previous studies and consistent with pleural scarring and a history of pleurisy. A few bihilar, postinflammatory granulomata were also present. Radiographic examination of the paranasal sinuses demonstrated total homogeneous opacification of the left maxillary antrum (Fig 1), indicative of sinus disease. The most important radiographic finding to identify in sinus disease is destruction of the sinus wall. Simple sinusitis without mucocele formation—whether it is allergic, bacterial, or viral—does not cause bone destruction. When bone destruction is present, more serious conditions, including neoplasms and granulomatous diseases, must be considered. Among the latter, fungi such as mucormycosis, aspergillosis, and histoplasmosis should be considered, as well as other granulomatous diseases such as sarcoidosis, syphilis, rhinoscleroma, tuberculosis, and Wegener's granulomatosis. However, the differential diagnosis is of secondary importance. Once observed, bone destruction indicates the need for biopsy. Unfortunately, when the sinus is totally opacified the detection of bone destruction without tomography may be very difficult, as in the present case.

The CT examination did not demonstrate conclusively a lesion of the palate (Fig 2). However, the left maxillary sinus appeared to be the site of the disease. On an axial cut through the mid-portion of the maxillary sinuses, the left maxillary sinus was filled with an abnormal mass, in which some low densities consistent with tissue necrosis and high densities consistent with calcification were present. The medial aspect of the antral wall was displaced into the nasal cavity. There was also evidence of bone destruction involving the medial and lateral walls with an abnormal tissue mass seen extending beyond the lateral margin of the maxillary antrum and involving the infratemporal fossa. The fibrofatty tissue plane of the parapharyngeal space was indistinct on the left side, suggesting tumor invasion or edema. This space was displaced medially, also suggesting disease lateral to it in the infratemporal fossa.

In summary, the CT examination demonstrates disease of the left maxillary sinus associated with bone destruction. These findings are consistent with a primary tumor arising at and extending through the infratemporal fossa and causing a deformity in the region of the nasopharynx.

Housestaff Clinical Discussion

Maria Julia Westerink, MD (Junior Medical Resident): The differential diagnosis of a mass lesion in the oral cavity and paranasal sinuses includes bacterial infection, granulomatous disease, and neoplasm. I will begin with a consideration of infection.

Bacterial infection with subsequent osteomyelitis seems unlikely without a history of previous sinusitis or purulent drainage from the nasal cavity. The possibility of tuberculosis is suggested by the patient's prior history of the disease and by the calcifications noted within the oral lesion. However, recurrent tuberculosis in the sinuses is rare and highly unlikely without such systemic findings as fever and weight loss. Fungal infection of the upper respiratory tract should be considered. Actinomycosis, the most com-

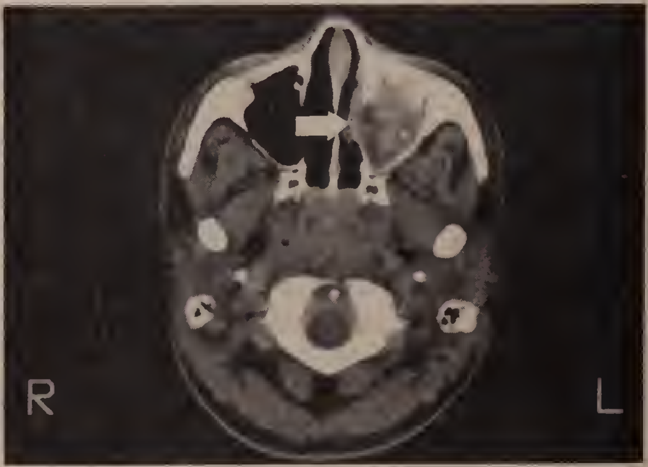


FIGURE 2. Computed tomographic (CT) scan of the head, with mass filling the left maxillary sinus, causing bone destruction and bowing of medial antral wall (arrow). Areas of radiolucency and calcification are within the mass.

mon of such diseases found in the oral cavity, usually involves the mandible and manifests multiple draining sinuses and characteristic sulfur granules in biopsy material. These findings were lacking in the present patient. Other fungal infections causing bony destruction include blastomycosis and histoplasmosis, both of which are rare in this geographical area. Mucormycosis is most often seen in poorly controlled diabetic patients and also in the setting of hematologic malignancies. I would consider this patient an otherwise normal host.

Wegener's granulomatosis involves the sinuses in 90% of cases, and may cause considerable bony destruction along with pulmonary and renal involvement. Although this patient developed acute renal failure, the urinary sediment was inactive, and the findings were of short duration and associated with chemotherapy. There was neither clinical nor histologic evidence of vasculitis to suggest Wegener's granulomatosis. Lethal midline granulomatosis is more common in older individuals and women. These patients usually experience nasal symptoms and progress to ulceration and destruction of the nasal mucosa and septum. Although midline granuloma may manifest as a buccal lesion, disease as extensive as seen in this case would rarely spare the nose.

Sarcoidosis should also be considered in this young man. However, the absence of hilar lymphadenopathy and noncaseating granulomas on biopsy eliminate this diagnosis.

The most likely diagnosis is a tumor. Benign tumors of the paranasal sinuses, such as inflammatory polyps and giant cell reparative granulomas, may cause destruction of bone and soft tissue. This patient's mass grew much too rapidly to support these diagnoses. Although lymphoma is common in this age group, Hodgkin's lymphoma is unlikely due to the absence of systemic symptoms and adenopathy, both clinically and radiographically. Histiocytic lymphoma, accounting for five percent of non-Hodgkin's lymphomas, arises in bone rather than soft tissue and may cause bony destruction. This is frequent in widespread disease but uncommon at initial presentation.

Esthesioneuroblastoma is the most common malignancy in this region in the younger age group. It usually appears between 20 and 30 years of age and arises from the olfactory nerves. Clinically and histologically it resembles adrenal neuroblastoma, but it may be confused with undifferentiated carcinoma or lymphoma. However, in view of the normal olfactory function of this patient, this diagnosis seems unlikely. Although it was suggested by one of the pathologists, malignant melanoma is rarely seen in the paranasal sinuses and represents less than 1% of neoplasms in this area. A wide range of soft tissue and bone sarcomas occur in the nasal cavity and paranasal sinuses, including chondrosarcoma, osteosarcoma, and soft tissue sarcoma; however, the tissue biopsy lacked typical cells seen in these lesions.

The most common malignant tumor of the paranasal sinuses is squamous cell carcinoma. It typically occurs in men over age forty and increases in frequency due to alcoholism and smoking, both of which are present in this case. It may progress to the orbit and oral cavity. This patient lacks the nasal findings of squamous cell carcinoma that include bloody nasal discharge, chronic sinusitis, and progressive destruction of the nasal structures. I favor the diagnosis of squamous cell carcinoma on epidemiologic grounds, and I was impressed by the biopsy findings in support of this possibility.

ATTENDING'S CLINICAL DISCUSSION

John Lore, MD (Professor and Chairman, Department of Otolaryngology, State University of New York at Buffalo School of Medicine): This man had a condition characterized by a rapidly growing, space-occupying lesion. It involved the palate, the maxillary sinus, a portion of the pterygomaxillary space, and the nasopharynx. The patient's trismus suggests tumor involvement of the pterygoid muscles as well. Several observations indicate that the lesion was a neoplasm. It was rapidly expanding and produced radiographic evidence of bony destruction. Fever, local inflammation, and leukocytosis which would likely accompany an infectious process were not evident. The lesion was not purulent, and there was no description of an infectious or granulomatous process in the first biopsy which provided evidence of a malignancy interpreted at the time as squamous cell carcinoma or possibly melanoma. Although the lesion was first noted in the palate, it may have originated in the paranasal sinuses or the nasal cavity. I will approach the diagnosis from each of these anatomical sites.

Benign tumors of the palate include torus palatinus, a clinically insignificant bony protuberance of the midline, and minor salivary gland tumors. Mixed salivary gland tumors account for 90% of the latter group with adenomas and cytadenomas completing this category. The benign minor salivary gland tumors may produce symptoms due to their enlargement, that requires a lengthy period of time due to their slow growth. For the sake of completeness, I will also mention papillomas, skin tags, and fibromas that are other benign lesions of the palate but not the diagnosis in the present patient.

Malignant tumors of the palate occur typically in the sixth and seventh decade of life and have a higher incidence in men. Squamous cell carcinoma is the most common of these tumors and often produces a fungating mass similar to this patient's. However, the rapid growth seen here over a period of weeks does not occur with squamous cell carcinoma. Malignant minor salivary gland tumors of the oral cavity occur most frequently on the hard palate, which was the presenting site for this patient's mass. Adenoid cystic carcinoma is the most common of these tumors followed by mucoepidermoid adenocarcinoma and malignant mixed cell tumors. These are slow growing and therefore unlikely in this case.

Primary mucosal melanoma arises most often in the head and neck, and the palate is its most common site in this region. However, mucosal melanoma of the head and neck occurs rarely in blacks or in individuals under 30 years of age. Most of the patients suffering from primary mucosal melanoma are over 50 years of age, report minimal symptoms, and have stage I disease at time of diagnosis. Amelanotic melanoma resembles other poorly differentiated neoplasms on histological examination. This diagnosis can be a difficult one to make with certainty and may explain the need for the third biopsy.

Tumors of the nasal cavity and paranasal sinuses are similar. A number of benign lesions may occur in these sites, such as mucosal cysts, dermoid cysts, papillomas, adenomas, mixed tumors, and myxomas. Osteomas are benign bony lesions surrounding the sinuses and constitute the second most common tumor of the paranasal sinuses.

Malignancies of the nasal cavity and paranasal sinuses have been associated with nasal polyps, chronic sinusitis, and chronic exposure to inhaled dust. Eighty percent of paranasal neoplasms are squamous cell carcinoma. As in the case of tumors of the palate, they occur typically in older men and are slow growing. A notable exception is the highly anaplastic spindle-cell carcinoma of the antrum that can be acutely invasive. This diagnosis is appealing as that it includes the most probable histologic group, and it accounts for the rapid and extensive spread of the lesion.

The glandular elements of the nasal and paranasal mucosa give rise to adenocarcinomas, cylindromas, acinic-cell carcinomas, and mucoepithelial carcinomas. A number of soft tissue and bony sarcomas occur in this area including rhabdomyosarcoma, chondrosarcoma, osteosarcoma, and Ewing's sarcoma. Of these, osteosarcoma is more prevalent in the mandible than in the maxilla, although it is rare in the facial bones. It may appear osteoblastic or osteolytic on radiograph. Sarcomas in general are diseases of young adults and are capable of the rapid proliferation seen in this case. They must, therefore, be considered seriously. Melanoma usually has an obvious nasal mass that was not seen in this case. As already discussed, however, this diagnosis cannot be excluded. Plasmacytomas may arise from paranasal lymphoid tissues and grow to several centimeters in diameter. They may be isolated to the nose or associated with disease elsewhere. Lymphomas, particularly histiocytic lymphomas that arise in bone, may also be seen here and remain a possibility due to the patient's age and the ability of these neoplasms to grow rapidly.

Esthesioneuroblastoma occurs most often in this patient's age group. The tumor arises in olfactory epithelium high in the nose, producing nasal stuffiness, epistaxis, rhinorrhea, and anosmia. As the tumor expands, it may produce frontal headache and diplopia. It is a slow-growing tumor and bony destruction is a late finding. Both midline lethal granulomatosis and Wegener's granulomatosis can produce bony destruction in the sites seen in this case. However, although the patient did have an episode of renal failure, this appeared to be related to the chemotherapy, and was not accompanied by active urinary sediment or by pulmonary symptoms, as would be expected with Wegener's granulomatosis. My diagnosis would be anaplastic spindle-cell carcinoma. Other possibilities are lymphoma or osteogenic sarcoma.

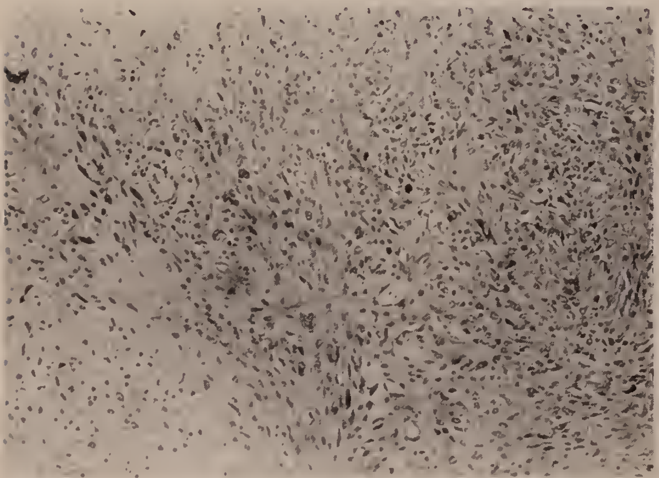


FIGURE 3. Spindle-cell sarcomatous structure with malignant cartilage at lower left. (hematoxylin and eosin stain; original magnification × 100)

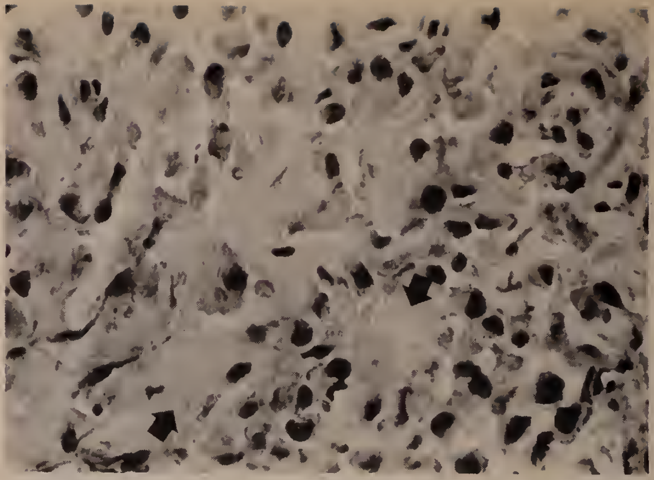


FIGURE 4. Sarcomatous structure forming osteoid (light homogeneous areas at arrows). (hematoxylin and eosin stain; original magnification × 440)

CLINICAL DIAGNOSES

Squamous cell carcinoma, left maxillary sinus.

Neoplasm involving the left maxillary sinus, most probably anaplastic spindle-cell carcinoma; possible lymphoma or osteogenic sarcoma.

PATHOLOGY PRESENTATION

Mario Montes, MD (Clinical Professor of Pathology, State University of New York at Buffalo School of Medicine): The first two biopsies consisted of necrotic tissue and atypical cells, but were inadequate for proper histopathologic study. In the third biopsy, the squamous mucosa contained a cellular tumor structure with necrosis and chondroid areas. The tumor was made up of spindle-shaped mesenchymal elements within a loose stroma (Fig 3). Under high magnification, these spindle-shaped cells appeared malignant (Fig 4). Some bizarre giant cells were present, as well as both normal and pathologic mitoses. The tumor cell nuclei were hyperchromatic and eccentric. These cells were forming osteoid that appeared as pink, condensed, homogeneous material that was partially calcified. The chondroid areas consisted of malignant cartilage. These findings were diagnostic of an osteogenic sarcoma, chondroblastic in type.

The surgical specimen consisted of the left maxilla. The maxillary sinus was completely filled with a pinkish-grey, fleshy tumor that had invaded the walls of the sinus, and had broken through at several sites. There were two tumor nodules similar to the one just described behind the second molar, measuring about 1 cm and 2.5 cm in diameter, respectively. The larger nodule protruded on the surface of the palate. The histology of the tumor in the maxillary sinus showed malignant cartilage, bone with calcific material, and malignant spindle-shaped cells forming dense osteoid. These findings were again diagnostic of osteogenic sarcoma, chondroblastic in type.

Conventional osteogenic sarcoma has a peak incidence in the second decade of life and arises most frequently in the metaphyses of long bones, particularly in the distal femur and proximal tibia. It may arise in association with Paget's disease, or as a complication of radiation therapy. The first clinical manifestation of the disease is usually pain, followed soon after by localized soft tissue swelling. The serum alkaline phosphatase activity is elevated in 60% of patients. The radiographic appearance may be lytic or sclerotic. Pathologic fractures are uncommon. Osteogenic sarcoma involves the marrow cavity and breaks through the cortex. The cut surface exhibits focal calcification, dense bone, chondroid tissue, and areas of fibrosis, necrosis, hemorrhage, and telangiectases. The microscopic appearance is that of a malignant spindle-cell tumor producing osteoid. The tumor may be subclassified as osteoblastic, chondroblastic, or fibroblastic according to the predominance of these elements. Benign multinucleated giant cells may also be observed.

The differential diagnosis includes chondrosarcoma, fibrous dysplasia, exuberant callus, myositis ossificans, and metastatic carcinoma. The majority of patients develop pulmonary metastases early in the course of their disease. The five-year disease-free survival rate is 20%.

In a series of 962 cases of osteogenic sarcoma, Dahlin and Unni reported 66 cases localized in the jaws.[1] These were predominantly chondroblastic with limited osteoid production. Compared to conventional osteogenic sarcoma of long bones, these jaw tumors occurred in older patients and exhibited less hematogenous spread and a better prognosis.

Fu and Perzin[2] reported 256 nonepithelial tumors of the nasal cavity, paranasal sinuses, and nasopharynx; 11 were osteosarcomas. In seven of these cases, the lesions were located in the maxillary sinus. Facial swelling was present in eight cases and local pain in three. Other symptoms included epistaxis, nonhealing wounds, ocular complaints, and numbness of the upper lip. The duration of symptoms prior to diagnosis ranged from one month to one year. All of the patients had a recognizable mass. Eight cases were treated with radical surgical resection. Survival was less than two months in all but one patient who was without recurrence ten years later. Radiation therapy and chemotherapy were ineffective in the remaining three cases.

Among six cases of maxillary osteosarcoma reported by LiVolsi,[3] three were associated with antecedent radiation therapy, Paget's disease, or maxillary trauma. The lesions spread to the ethmoid sinus in two cases, to the nasopharynx in one case, to the opposite antrum in five cases, and to the brain in one case. Metastasis occurred to the lungs and to other bones. Various combinations of surgery, radiation therapy, and chemotherapy resulted in a maximum survival of only three years.

Russ and Jesse[4] reported 30 cases of osteosarcoma of the maxilla and mandible. The median age at diagnosis was 37 years and the sex distribution was equal. Most patients presented with a mass in the gum that was often painless. The duration of symptoms was usually less than six months. As in the present case, a dentist was often the first health professional contacted, and 25% of the patients had dental extractions prior to diagnosis. Previous conditions at the site of the tumor included an ossifying fibroma, a giant-cell epulis, an osteochondroma, an epidermoid carcinoma of the nasopharynx, and skin cancer. Seventeen patients had tumor originating in the maxilla. Of ten patients treated with maxillectomy, with or without orbital exenteration, five survived for five years. The remaining patients had local recurrences and one patient developed pulmonary metastases. Radiation therapy proved useful in the management of primary and recurrent osteosarcoma in the maxilla and mandible, whereas chemotherapy was of no benefit.

Clark et al[5] reported 66 patients with osteosarcoma of the jaw treated at the Mayo Clinic. The 42 male and 24 female patients had a mean age of 34.2 years. The tumors were equally distributed between the maxilla and the mandible. Seven patients had previous radiation therapy and one had Paget's disease. On radiograph the mandibular lesions were most often lytic, whereas the maxillary lesions were usually sclerotic. Less than 5% of the lesions displayed a classic sunburst appearance. The histology of these tumors was similar to osteosarcoma of the long bones with osteoblastic, chondroblastic, and fibroblastic types observed. Fifty percent of the jaw tumors exhibited predominantly chondroid differentiation and 50% were high-grade by Broders' criteria, compared to 80% high-grade lesions in conventional osteosarcomas. Distant metastases were found in only four patients. The overall five-year survival rate in this series was 40%. The best survival rate was noted in patients treated with surgery alone. Patients in whom radiation and/or chemotherapy was administered along with radical resection did less well, possibly due to more extensive disease. The results of local surgery or radiation alone were very poor. Most deaths resulted from uncontrollable local recurrences.

In summary, this is a case of osteosarcoma of the left maxillary sinus, chondroblastic in type.

FINAL DIAGNOSES

Osteogenic sarcoma of the left maxilla, chondroblastic in type, with extension to the palate and pterygoid muscle.

REFERENCES

1. Dahlin DC, Unni KK: Osteosarcoma of bone and its important recognizable varieties. *Am J Surg Pathol* 1977; 1:61–72.
2. Fu Y, Perzin KH: Non-epithelial tumors of the nasal cavity, paranasal sinuses, and nasopharynx: a clinicopathologic study. II. Osseous and fibro-osseous lesions, including osteoma, fibrous dysplasia, ossifying fibroma, osteoblastoma, giant cell tumor, and osteosarcoma. *Cancer* 1974; 33:1289–1305.
3. LiVolsi VA: Osteogenic sarcoma of the maxilla. *Arch Otolaryngol* 1977; 103:485–488.
4. Russ JE, Jesse RH: Management of osteosarcoma of the maxilla and mandible. *Am J Surg* 1980; 140:572–576.
5. Clark JL, Unni KK, Dahlin DC, Devine KD: Osteosarcoma of the jaw. *Cancer* 1983; 51:2311–2316.

Institutional ethics and graduate medical education*

ALFRED GELLHORN, MD

The State of New York holds a unique position in American medicine by virtue of its size, quality, and complexity. After World War II the winds of change were behind the medical enterprise, speeding it on its way. Now headwinds face medicine everywhere, and the effect in New York dictates change. Since graduate medical education has a profound influence on the future practice of medicine, it is important for the medical profession to understand the factors that will modify the current status of residency programs in New York.

GRADUATE MEDICAL EDUCATION IN NEW YORK STATE TODAY

There are about 82,000 medical residents in the United States and New York has 14.7% of them, or about 12,000. New York is the Empire State, and so it may be appropriate for it to have more men and women in clinical training than any other state in the Union. But in reality, the state's graduate medical enterprise is bloated in relationship to its population. It already has more than 280 physicians for every 100,000 persons and at the current rate will exceed 330 per 100,000 in the next two decades—the optimal suggested by the Graduate Medical Education National Advisory Council (GMENAC) was between 180 and 190 per 100,000. Its residency programs offer 50% more positions than there are American medical school applicants to fill them. These values are compared with those of two states which are most similar to New York quantitatively (Fig 1).

One of the explanations put forth in New York is that its statewide residency programs are of such high quality that graduates from all over the country flock to New York for their training. In part, this is true, as indicated by the 1984 National Residency Match. Last year, for the first time in many years, less than half of the graduates of New York medical schools (48%) left the state for residency training and it is significant that slightly more medical graduates from schools in other states came to New York than the number of New York medical graduates that went elsewhere (Personal communication from John S. Graettinger, MD, Director, National Residency Match Program). In other words, New York was in positive balance with regard to the exchange of medical school graduates. Despite this, there were hundreds of residency positions that went begging and, as nature and hospitals abhor a vacuum whether of empty beds or unfilled residencies, foreign medical school graduates filled the gap. It may be helpful to look at the foreign medical graduates (FMGs) more closely, since actions which affect them will affect all teaching programs, regardless of whether or not they have FMG residents.

In the past 20 years there have been two streams of foreign medical graduates entering the United States. In the 1960s and early 1970s, there was active recruitment of physicians from abroad to meet the rising demands in the US for access to medical care. To facilitate the entry of FMGs, the US immigration laws were made very permissive so that in 1973, the peak year of foreign physician influx, 7,097 of the 8,145 were admitted as immigrants.[1] In 1968, 23% came from Europe, 19.5% from Canada and Central America, and 43% from Asia (predominantly India and the Philippines). In 1976 Congress revoked the preferential professional inducements provided in the earlier amended Immigration and Nationality Act, and in Title VI of PL 94-84 stated ". . . there is no longer an in-

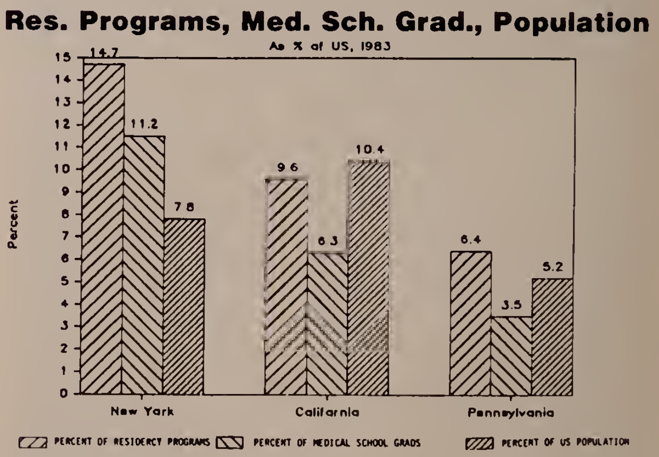


FIGURE 1. Residency programs, American medical school graduates, and state population expressed as a percent of the total in each category in the United States.

Address correspondence to Dr Gellhorn, Director of Medical Affairs, New York State Department of Health, Tower Building, Empire State Plaza, Albany, NY 12237.

* This article is an edited version of the Samuel Belkin Memorial Lecture delivered at the Albert Einstein College of Medicine, April 2, 1985.

sufficient number of physicians and surgeons in the United States." By 1978 the number of immigrant physicians admitted had dropped to 4,435; the proportion from Europe had decreased to 12.3%, but those from Asia increased to 52.5%.

Like the overflowing pot of cereal in the *Sorcerer's Apprentice*, it has not been easy to control the growth of physician manpower in the United States. The first factor that has kept the pot bubbling was the incentives provided to American medical schools to increase their enrollment and generous financial assistance for the creation of new medical schools. The response was prompt and enthusiastic, with the result that in the 1970s, medical school output doubled from about 8,000 graduates to 16,000.

Other factors that might have influenced the second stream of foreign medical graduates to the United States were the coming of age of the post-World War II "baby boom" and the enactment of the Medicare and Medicaid legislation by Congress. This legislation has had a favorable impact on physician income which, when added to the social prestige of the profession, made medicine one of the most popular career aspirations of young Americans (or of their parents, or of both) in the 1970s. It became so desirable, in fact, that many of those who failed to gain admission to an American medical school enrolled in foreign schools. In the late 1960s and early 1970s, these American students usually went to medical schools in Europe often after paying a fee to an agency in the United States which facilitated the matriculation. Eventually, however, the population of American students was too great on the already overcrowded medical schools in Italy and Spain, and admission of foreigners was curtailed. Guadalajara's Universidad Autonoma in Mexico had always been a popular medical school for disappointed North American medical school applicants, and its example stimulated the creation of a number of "offshore" enterprises in the Caribbean which were given charters by the country of residence to bestow an MD degree.[2] Thus was developed the second stream of foreign medical graduates to American shores with, however, the distinct difference from other FMGs in that they are US citizens with legal and lobbying power disproportionate to their number. The other distinct difference of the offshore medical schools, as pointed out by the Association of American Medical Colleges, is that although they are foreign chartered, they operate in the United States,[3] as shown in Table I.

The quantitative impact of these two migrations of foreign medical graduates can be characterized in several ways. Table II shows the number of FMGs in New York residency positions as a proportion of all residents in New York and contrasts this with the entire United States.

In 1984, the New York State Department of Health made a survey of FMG residents in all of the teaching hospitals and found that 25 of the 86 hospitals have 60% or more FMG residents. These residents were located predominantly in hospitals with a small number of beds and residents.

The issue of foreign medical graduates concerns the responsibility of government to protect the public from medical practitioners whose undergraduate and/or graduate medical education does not meet the standards that are required of American medical school graduates. There was a time from the latter half of the 19th century to the second decade of the 20th century when graduates from European medical schools with training in European hospitals and clinics were far better qualified than American graduates, and those physicians were welcomed here. But now, with more than 1,400 medical schools in the world, in almost every country, it is necessary to raise questions regarding the adequacy of the education of foreign medical graduates. For the most part, foreign medical schools are not monitored by national or international accrediting organizations. By contrast, all American and Canadian medical schools undergo a rigorous evaluation by the Liaison Committee on Medical Education (LCME) at seven-year intervals.

The most recently established foreign medical schools, the Caribbean proprietary institutions, have limited, sub-

TABLE I. A Partial List of Foreign-Chartered Medical Schools with Offices in the United States, 1984

Country Where Chartered	Medical School	US Address
Antigua	University of Health Sciences Antigua	Atlanta, GA
Dominica	Ross University (Formerly University of Dominica)	New York, NY
Dominican Republic	CIFAS University	Los Nietos, CA
Dominican Republic	Universidad de Nordestana	Brooklyn, NY
Dominican Republic	Universidad Technologica de Santo Domingo (UTESA)	Whittier, CA
Dominican Republic	World University	Washington, DC
Grenada	St. George's University	Bay Shore, NY
Mexico	Universidad Autonoma de Guadalajara	San Antonio, TX
Mexico	Universidad del La Salle	New York, NY
Mexico	Universidad del Noreste	New York, NY
Mexico	Universidad Valle del Bravo	McAllen, TX
Montserrat	American University of the Caribbean	Miami, FL
St. Lucia	Spartan Health Sciences University	El Paso, TX

Source: Imperato PJ: An overview of New York State and the offshore medical schools. *NY State J Med* 1984; 84:337-344.

TABLE II. Number of FMG Residents and Their Percent of Total

	1979		1982		1983	
	No.	%	No.	%	No.	%
New York	3,779	43.1	4,147	40.4	4,028	38.2
United States	12,065	20.2	13,123	19.0	13,221	18.4

Source: *American Medical Association Directory of Residency Training Programs*, 1984-85.

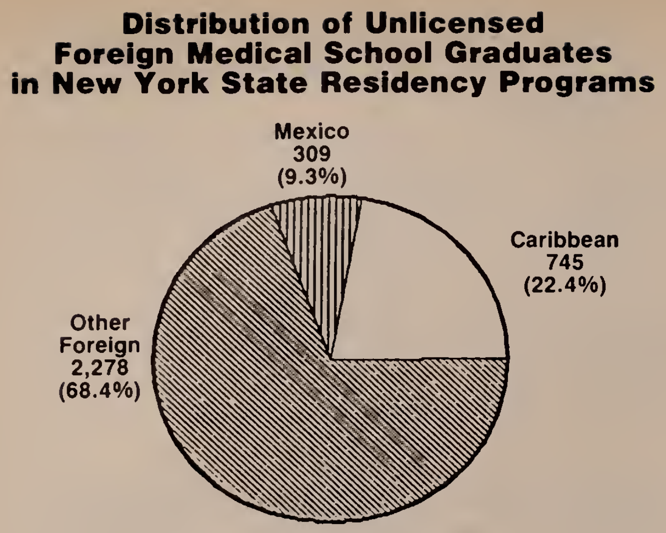


FIGURE 2. Distribution of unlicensed foreign medical school graduates in New York State residency programs. FMGs must have three years of approved residency training before being eligible for medical licensure in New York; USMGs are eligible after one year of residency. (Source: New York State Department of Health Survey of Unlicensed Physicians in Hospital Training, 1984.)

marginal basic science educational resources and, with inadequate clinical facilities, they must send their students to the United States (and in large numbers to New York and New Jersey) for the clinical clerkships of the third and fourth year. Because these clerkships were often unsupervised either by the hospital personnel or anyone related to the school, the New York State Commissioner of Education established a regulation which mandates that clinical clerkships cannot be served in New York hospitals unless the medical school and the hospitals have been approved. Only one medical school in Mexico has been approved. At the present time, however, the distribution of FMG residents shows the significant representation of Caribbean graduates (Fig 2).

There are many hundreds of medical schools throughout the world that do not depend on clinical clerkships in the US, and until such time as the Commission on Foreign Medical School Education (the analog of the LCME), which has been created by the Federation of State Medical Boards, begins to evaluate the foreign medical schools that train American citizens, the adequacy of the education provided must be assessed by examinations of the Educational Commission for Foreign Medical Graduates (ECFMG). The ECFMG examinations have been criticized because they were less difficult and comprehensive than the National Boards taken by American medical students. Despite this, the performance of FMGs was far from outstanding, as shown by the results in Table III.

In July 1984, a new type of examination was given by the ECFMG which used the National Board bank of questions and was more closely akin to Parts 1 and 2 of the National Board of Medical Examiners. On their first try with this examination 28.6% of the foreign born (alien) medical school graduates (AFMGs) and 28.5% of the US foreign medical school graduates (USFMGs) passed the basic science component; 39.4% of the AFMGs and 20.5% of the USFMGs passed the clinical science part. Both parts of the examination were taken by 7,423 of the 12,388 foreign medical school examinees: 17.4% of the AFMGs passed both parts, whereas only 3.8% of the USFMGs were successful.[4] In contrast, 86% of American medical school candidates passed Part 1 and 98% passed Part 2 of the National Boards in 1984.[5]

A Commission on Graduate Medical Education in New York, created at the request of Governor Cuomo, has considered the question of foreign medical graduates and, having come to the conclusion that undergraduate medical education must meet certain standards of quality before a graduate undertakes the responsibilities of residency training, has under consideration more stringent requirements for eligibility of FMGs for residency positions that currently exist.

There is a second set of facts that needs to be discussed before looking at the institutional ethics of graduate medical education.

The distribution of house officers is skewed in several ways. For example, as the number of beds in a hospital increases, the number of residents per unit of beds increases. In hospitals that have from 50 to 500 beds and have residency programs, the ratio of beds per resident ranges from 6.3 to 10.6, with a mean of 8.0. In contrast, in hospitals that have from 600 to 1,200 beds, the ratio of beds per resident is from 2.4 to 3.8, with a mean of 3.1. This reflects the larger number of residency programs in the major medical centers, and explains why more than half of all the residents in the state will be concentrated in slightly less than a quarter of the teaching hospitals.

Another fact relates to the decrease in the number of hospital beds in New York City over time, together with an increase in the number of residents. Between 1978 and 1984, the number of hospital beds decreased by 1,000; the number of residents increased by 700. How can these data be rationalized? Two factors, at least, appear to have contributed to this dichotomy. First has been the reimbursement pass-through phenomenon. Since compensation for the house staff—both salary and indirect costs related to their education—are included in the per diem reimbursement (New York State Department of Health, Office of Health Systems Management. 1983 New York's Prospective Hospital Reimbursement Methodology [NYPHRM]) and since, until recently, the reimbursement was on a cost plus basis, there was no incentive for hospital administrators or clinical chiefs to be restrained. And population control has not been a prominent characteristic of those of us in academic medicine when related to a replication of our own kind.

TABLE III. Results of ECFMG Examinations in 1980

Type of Examination	Number Examined	Number Passing	Percent Passing
All initial examinees	4,803	2,396	49.9
All repeat examinees	4,774	1,180	24.7

Source: Educational Commission for Foreign Medical Graduates: *The Changing Role of Foreign Medical Graduates in the Practice of Medicine in the U.S.A.*, 1981.

A second factor, often cited by the major medical centers, has been the increase in labor-intensive technological advances. This is undoubtedly true, but in many instances the end results of the technology in terms of human health have not been sufficiently evaluated to justify its widespread use.

Another factor that contributes to the skewed distribution of hospital staff is that the house officers in New York State are disproportionately distributed in metropolitan New York, where 80% of the residents are concentrated. This is not proportional to the population distribution (7 million people in metropolitan New York; 10 million in the rest of the state) and is more an accident of history. Until the State University of New York schools were created, there were only small private medical schools in Albany, Buffalo, and Syracuse—with the remainder in New York City. This concentration may be a financial advantage to New York City, attracting money, providing employment, and creating medical prestige, but it is an expensive luxury that leads to duplication, competition, and lapses in meeting community responsibilities. There are two general types of community responsibilities.

Physician availability. The Health Planning Commission of the New York State Department of Health published a Primary Care Profile of the entire state in 1984.[6] The analysis combined need indices and physician and other service supplier availability in 700 small, sub-county areas. For each area, the consumer demographics included age, sex, and race; income, in which poor was defined as $7,412 yearly for a family of four; and low birthweight, defined as the percent of all live births weighing less than 2,000 grams. The latter was used as the surrogate for health status. The physician supply data included age, specialty, and direct patient care activity. The final analysis concluded that 224 of the 700 small state areas had a high priority for physician placement. These priority areas, with a total population of 4 million, had a shortage of primary care physician service, problems with low birthweight babies, and large portions of their population existing at or below an annual income of $7,412. Two thirds of these areas are located in New York City, with a total population of over 2.6 million residents. Of the areas in New York City, those with the largest priority for remediation have only 335 active care physicians for 1.3 million people, or less than 29 per 100,000.

A slightly lower priority grouping of 82 rural communities with 1.6 million residents is characterized by physician shortage, poverty, and a high percentage elderly persons.

Physician training. A recent report on the general medical care of patients in the major New York State inpatient psychiatric centers on Long Island[7] revealed substandard primary medical care in those wards in which "medically well" patients were housed, and an impressive variety of acute illnesses in the secondary care unit, a number of which were misdiagnosed and poorly managed by the staff physicians. About 65% of the doctors in these centers were foreign medical graduates, and they were isolated from peer support by the community or nearby university physicians. Most striking was the absence of integration of medical and psychiatric care. It was as though Descartes was the chief of service saying that the brain is the seat of the soul and should be left to the church, whereas the rest of the body is like a machine and should be taken care of by physicians. Thus, the opportunity to educate medical doctors to be more than mechanics and to become complete physicians and the opportunity for psychiatric doctors to become more than purveyors of narcoleptic drugs was lost. The report recommended that a university medical school teaching service be developed to teach general medical care to house staff and attendings.

TABLE IV. Primary Care Specialty Residency Positions in New York State

Primary Care	Number	Percent
Medicine	3,217	29.2
Pediatrics	1,052	9.6
Family Practice	422	3.9
Obstetrics/Gynecology	722	6.6
Total	5,413	49.2

Source: Council for Graduate Medical Education: *American Medical Association Directory of Residency Training Programs*, 1984–85.

The skewed distribution of house officers can also be explained by the following: the distribution of residents among the medical disciplines in New York indicates that about half are in the primary care specialties and the other half are in all the other disciplines (Table IV). This illustrates the overriding need for generalists to provide continuity of care, to have comprehensive ability to diagnose and manage 85% of the common but not trivial medical problems of a population, and to use preventive and rehabilitative means when appropriate. Unfortunately, the statistic is deceptive. Internal medicine, like surgery, is a many splendored thing, and the majority of medical residents proceed into subspecialties. Only two of these subspecialties, allergy/immunology and cardiology, have specific residencies—all the others take the form of fellowships or residency positions which are not formally recorded. The other 50% of residency positions are in surgery and other specialties (Table V).

TABLE V. Residency Positions Other Than Primary Care Specialties in New York State

Discipline	Number	Percent
Surgery	1,522	13.8
Subspecialties	1,063	9.7
Psychiatry	1,053	9.6
Radiology	627	5.8
Anesthesiology	500	4.5
Pathology	386	3.5
Medical Subspecialties	355	2.9
Other	110	1.0
Total	5,588	50.8

Source: Council for Graduate Medical Education: *American Medical Association Directory of Residency Training Programs*, 1984–85.

IMMINENT CHANGE

Washington is concerned about money—money that is spent on things other than military matters. Since 1965, graduate medical education has been paid from the Medicare Hospital Trust Fund, together with a proportional share from all other third party payers. The impending insolvency of the Medicare Trust Fund, escalating health care costs, and the national debt, have led to a number of suggestions pertaining to funding of graduate medical education:

- In 1982, the Social Security Advisory Council recommended that Medicare funds be discontinued for educational costs.
- The Inspector General of the Department of Health and Human Services recommended that Medicare fund only the first year of hospital training, and thereafter the house staff would support itself by direct billing of patients.
- Last year, Senator Durenberger's Finance Committee suggested that block grants be made to the states and that the state governments decide the allocations for graduate medical education.

These proposals have been put aside, but a new suggestion by Senator Dan Quayle of Indiana is attracting attention and support. Senator Quayle is concerned with more than money: to wit, the excess production of physicians and the forecasts of GMENAC regarding the doctor glut; the overspecialization in the medical profession; and the quality of residency training. He proposes to link Medicare support for graduate medical education programs to "institutional compliance with national guidelines reflecting sensitivity to physician manpower issues:"

- The number of first year residency positions in the United States should approximate the number of graduates of American LCME-approved schools and never should be less.
- Eligibility for Medicare funds to support graduate medical education programs will require that the majority of positions offered be in the primary care specialties, and "majority" is suggested to be 70%. The distribution of the remaining 30% would be an institutional decision.
- The duration of graduate medical education support from Medicare funds would be restricted to no more than three years for each resident.
- In view of the excess of physicians projected by the end of the 1980s, graduate medical education payments by Medicare would be restricted to those programs with 75% or more American medical graduates.
- To assure nationwide compliance and the quality of graduate medical education, and to integrate better undergraduate and graduate medical education, the responsibility for coordinating graduate medical education programs would be vested with the nation's medical schools, such that all teaching hospitals would be affiliated with a medical school.

ETHICS AND GRADUATE MEDICAL EDUCATION

In his exciting and informative book, *Physicians, Law and Ethics*,[8] Carlton Chapman has traced the ethical concepts of the medical profession from the Sumerians in 3000 BC, through Hippocrates in 500 BC, to the present moment. Through a variety of influences, including social, legal, scientific, technological, and philosophical, Dr Chapman shows that at long last it is recognized that the physician's basic ethical consideration is for the welfare of the patient, not the profession. The commitment to the medical care and well being of the individual patient, with the latter's consent, combined with the Hippocratic admonition "To do no harm" constitute powerful and often difficult guidelines to follow.

In contemporary times, the ethical obligation to the individual patient is made complex and confusing when issues of equity of access to medical care, distributive justice, and the allocation of scarce resources and cost containment arise. The following discussion is focused on the ethical and social obligations concerned with access to high quality medical care. Untouched is the current and pressing ethical issue of cost containment which pits the physician's obligation to benefit his or her patient regardless of cost against society's need to set limits on health care expenditures in order to fund other valued goods such as education, housing, transportation, museums, and symphonies.

Physicians bear obligations not only to the individual patient but to society as well. Their education during medical school is subsidized by state funds (and until the current Administration, by federal capitation grants). As for house officers, federal, state, and private funds provide them with a salary which makes it possible to continue their education and to learn by doing. The knowledge and skills which they have acquired from teachers and from patients should not be considered as personal property which can be sold in the open marketplace. In very significant part, they have an obligation to share this admittedly hard-earned knowledge with society. And what about the institutions of which they are a part? Public and private non-profit hospitals are dependent on public as well as private support, which create obligations to the surrounding neighborhoods and communities. Because of the sheer size and grandeur of great institutions, it is easy to be drawn into the intramural life and forget about the pressing needs of those on the outside who also have a claim to physician concern.

If physicians accept that their obligations to society are not satisfied by locating their practices exclusively among affluent populations where physician income is high, where there are richly endowed clinical facilities, hospitals, ancillary services, peer reinforcement, and referral networks, what changes in graduate medical education might provide some different perspectives?

As indicated above, residency positions will almost surely be curtailed in the next few years in New York. FMGs are concentrated in residencies in smaller community hospitals, whereas USMGs in New York are concentrated in university medical centers where the experience emphasizes tertiary, high technology care. A redistribution of house staff will have to occur through a meaningful affiliation of community hospitals and community and state ambulatory care facilities with the university medical centers, assuring excellence of educational experience; the development of knowledge and skills in primary and secondary medical problems; continuity of relationships with community populations, together with a major improvement in the medical care provided. Such a system of health care which is not fixed on death prevention alone will begin to make distinctions between maximal treatment and optimal care. Research should be carried out in technology assessment, in determining the health status of the community and, in developing health education and preventive measures, possibly even reducing the requirements for some forms of tertiary care.

This realignment of residency programs with meaningful affiliations between medical center hospitals and community facilities will be encouraged by the reimbursement policies of the federal government, state governments, or both. Even more important would be if the leadership of major residency programs would better recognize the societal needs, in order to diversify the educational experience of their house staff colleagues.

At present, the direct cost of graduate medical education in New York is estimated to be $350 million. The approximate distribution of fund sources is 40% Medicare, 35% private insurance, and one quarter Medicaid. The state pays one half of the Medicaid bill, or about $44 million annually, for residents' salaries. The question is whether or not the sole obligation of the state's tax dollars and the public's insurance premiums is to support physician education, regardless of how that education may be used subsequently by the resident. The state and the public must recognize that the house officer is providing service while gaining knowledge and skills and, in many or perhaps most instances, providing this service to a poor population which might otherwise be deprived. But the realities of the medically underserved poor persons in New York State are issues which neither the State nor teaching hospitals can ignore without peril to aspirations for a humane society. Federal and State health policy initiatives are now being directed toward the problem of geographic distribution of physicians. In addition to financial incentives, loan forgiveness, and revitalization of national or state health service corps, opportunities should be developed for house officers to have a learning-service experience in an underserved area. This might be combined with instruction in the methodology of health services research so that insights could be acquired of facets of research other than at the bench or bedside.

Mention has been made of what are important aspects of graduate medical education—namely, awareness and sensitivity to the medical and health needs of populations. If the generations of physicians now in graduate medical education are to provide the leadership, as they should, in areas of social reform leading to more equity in access to medical care, more understanding of the issues in distributive justice, and greater recognition of those factors other than medical care which contribute to health, then the experiences and horizons of graduate medical education may have to be expanded to accommodate this vision.

There was a time when physicians, and particularly those connected with a university, were recognized not only as physicians but as citizens and leaders in civic affairs. It is my impression that physicians have become so preoccupied with the technology and the business of medicine that they have retired from active participation in communities and even in informed understanding of national and international issues.

The roles of students and house officers should be more than reacting to alternatives that time and circumstances place before them. And, in part, the teaching hospital which assumes the responsibility for education should provide a focused, systematic, even aggressive concern for the manner in which society is evolving—a concern for values, for the problems society faces, and for strategies to define those values and to solve the problems.

Physicians should be engaged not only in the one-on-one care of patients but also in the seeking of new solutions to the health problems of our time. Medicine offers myriad opportunities, none more exciting than participating as a physician and a citizen in the design of the future.

REFERENCES

1. Annual Reports 1970–77, Immigration and Nationalization Service, US Department of Justice.
2. Imperato PJ (ed): The offshore medical schools. Current issues and controversies. *NY State J Med* 1984; 84:337–371.
3. Luginbuhul WH, Cohen DH, Deal W, Jones JE, Petersdorf RG: Quality of preparation for the practice of medicine in certain foreign-chartered medical schools. *J Med Educ* 1981; 56:963–979.
4. Steele WS (ed): Results of first administration of FMGEMS. *Outlook* 1984; 2:4.
5. Report on National Board Examinations for 1984. *The National Board Examiner* 1984; 31:1–4.
6. New York State Health Planning Commission Primary Care Profile, January 1984.
7. Gellhorn A: Report to the New York State Commissioner of Health: General Medical Care in Long Island Regional Psychiatric Centers. June 1984.
8. Chapman CB: *Physicians, Law and Ethics*. New York, New York University Press, 1984.

Epidermoid cysts of the cranium

V. JAGADHA, MD, T. POON, MD

Epidermoid cysts are rare lesions of ectodermal origin that account for approximately 1% of all intracranial tumors.[1] The identification of these lesions presents both clinical and radiologic problems; they tend to escape attention for long periods of time by the virtue of slow growth. Two cases of intracranial epidermoid cysts are presented below with a comment on the pathogenesis and clinicopathologic features of this curious entity.

CASE REPORTS

Patient 1. A 31-year-old woman complained of persistent frontoparietal headaches of nine months' duration. The headaches were unrelated to time of day and posture; there was no relief with analgesic agents. She also complained of pain in both eyes with diminished visual acuity on the left side. There was also an indefinite history of increased urinary frequency without polyuria. Her medical history was otherwise unremarkable.

Physical examination revealed no significant abnormalities. Neurologic examination showed intact motor and sensory responses with no cranial nerve deficits. The pupils were equal on both sides and reacted normally. Visual acuity and visual fields were normal. The fundus examination was unremarkable. A complete laboratory workup including blood count, serum chemistry, urinalysis, and coagulation profile was normal. There were no electrolyte abnormalities and no clinical indication of diabetes insipidus. A computed tomography (CT) scan of the skull revealed a large suprasellar cyst with a solid component (Fig 1). Angiograms were not conclusive.

A right frontotemporal craniotomy was performed. A small, encapsulated, whitish cyst was seen laterally and inferiorly adjacent to the carotid artery. The cyst cavity measured 1.5 × 1 cm; approximately 1.5 to 2 ml of cheesy material was suctioned from the cavity. The complete removal of the cyst was not possible since the wall of the cyst was attached to the optic chiasm and the anterior cerebral artery. Pathologic examination revealed the cyst wall to be lined with stratified squamous keratinizing epithelium (Fig 2). Squamous cells and keratinous material were identified in the lumen.

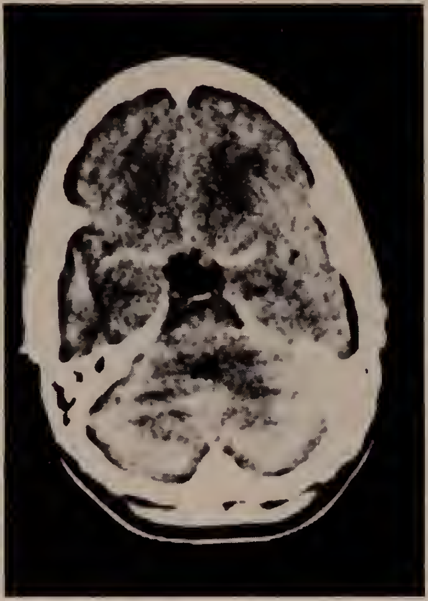


FIGURE 1. CT scan of the skull revealing a suprasellar cyst in Patient 1.

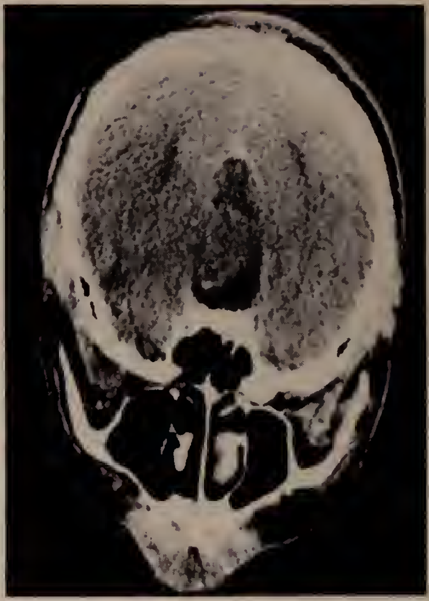


FIGURE 3. CT scan showing a right posterior fossa mass in Patient 2.

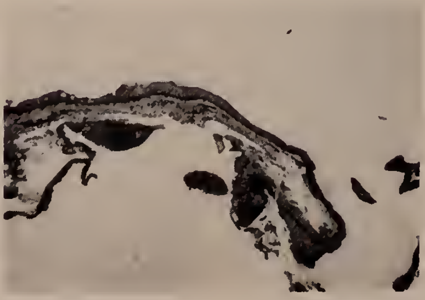


FIGURE 2. Cyst wall lined by stratified squamous keratinizing epithelium in Patient 1. (hematoxylin-eosin; magnification × 25)

Patient 2. A 25-year-old man was admitted to the hospital with frontoparietal headaches of four to five months' duration that varied in site and intensity. There were no specific aggravating or relieving factors. The patient was diagnosed as having sarcoidosis two months previously and was taking aspirin, prednisone, and isoniazid. His medical history was unremarkable.

Physical examination was normal except for a grade II/VI systolic murmur in the precordium. The electrocardiogram revealed no significant abnormalities. Hepatosplenomegaly was present (liver, 18 cm in the midclavicular line; spleen, 16.8 cm in maximum vertical dimension); bilateral uveitis was also noted.

The neurologic examination revealed the patient to be well oriented to time, place, and person; there was no sensorimotor deficit. The deep tendon reflexes were intact. The gag reflex could not be elicited.

Laboratory workup showed interstitial infiltrates on the chest roentgenogram that later resolved with prednisone therapy. There was also evidence of cutaneous anergy on the skin test panel. A gallium scan revealed homogenous radionucleotide uptake

---

From the Department of Pathology, New York Medical College, Metropolitan Hospital Center, New York.

Address correspondence to Dr V. Jagadha, Division of Neuropathology, Toronto General Hospital, 222 Elm St., Box 402, Toronto, Ontario M5T 1K5 Canada.

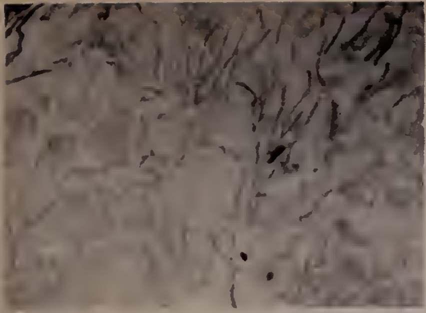


FIGURE 4. Cystic contents with keratinous material in Patient 2. (hematoxylin-eosin; magnification × 125)

consistent with sarcoidosis. The liver function tests were abnormal (alkaline phosphatase, 1,300 units/L; SGPT, 110 units/L; SGOT, 60 units/L; and GGT, 1,189 units/L). Liver and bone marrow biopsies revealed noncaseating granulomas.

The anteroposterior, lateral, and Towne projection roentgenograms of the skull were negative. The CT scan showed a low density, extra-axial, right posterior fossa mass that displaced the brainstem, and compressed and displaced the fourth ventricle (Fig 3). There was no enhancement after metrizamide injection. Angiography was consistent with an extramedullary posterior fossa lesion. The patient also had abnormal brainstem auditory-evoked responses that suggested a delay in brainstem pathways.

The contents of the lesion were aspirated—due to its strategic location—and the postoperative recovery was uneventful. Pathologic examination revealed keratinous material (Fig 4).

DISCUSSION

Jean Cruveilhier is credited with the first description of epidermoid cysts in 1829 when he named them "the pearly tumors"; in 1838 Muller proposed the term cholesteatoma to describe a tumor of the cranial bone containing a mass of cholesterine (as cited by Obrador).[2] Subsequently, similar formations in the mastoid and the middle ear related to chronic ear disease came to attention. Since that time, this lesion has aroused considerable clinical interest and has been the subject of several reviews.[1-4]

Some confusion exists over whether the cranial epidermoid cysts and their otic counterparts are either the same or separate entities. While some individuals believe that cholesteatomas of the middle ear are collections of desquamated epithelium caused by ingrowing squamous epithelium due to chronic inflammation,[5] it is generally held that aural cholesteatomas, like their cranial counterparts, are inclusion cysts caused by epithelial entrapment at heterotopic sites.[4] Two mechanisms may explain the pathogenesis of intracranial epidermoid cysts; the occurrence of congenital epithelial nests or the mechanical displacement of the epithelium in later life.[4,6] The occurrence of epithelial nests in the pituitary stalks of newborns supports a congenital origin.[7] Examples to support the concept of trauma or mechanical displacement include the finding of epidermoid cysts in children who had repeat lumbar punctures,[6] and the occurrence of epidermoid cysts at the site of the primary suture of a scalp laceration.[4]

The most common sites of occurrence of epidermoid cysts include the cerebellopontine angle, the suprasellar region, the fourth ventricle, the pineal region, and over the convexity of the hemispheres.[2] The cysts within the skull and spinal canal may be connected to the skin by a congenital sinus, thus posing the threat of infection. Associated congenital anomalies have been reported by MacCarty and colleagues[3] and include anomalies of vertebral fusion, septum pellucidum cysts, the presence of a dermal sinus, harelip, pilonidal sinus, cervical rib, meningocele, spina bifida, and cysts under the occipital portion of the scalp.

The usual age for the identification of epidermoid cysts is between 20 to 40 years.[2] These lesions remain clinically silent for long periods of time despite their considerable size. When symptomatic, they may present as optic atrophy, hemianopias, epileptic attacks, hemiparesis, speech disturbances, cranial nerve palsies, and cerebellopontine symptoms.[2] Mental symptoms may also be present.[8] Cysts of the suprasellar or cerebellopontine regions may be recognized relatively early—as in our patients—because of their proximity to important structures. Radiographs may not reveal the lesion; computed tomography is the method of choice since epidermoid cysts appear as low density lesions with absorption values similar to or slightly lower than those of cerebrospinal fluid.[9] There is no enhancement of the contrast medium due to the avascularity of the lesion.[10,11] Lesions such as meningiomas, chordomas, and neuromas, on the other hand, are characterized by increased density on CT scan.[10] As in Patient 1, the differential diagnosis of a suprasellar lesion may include Rathke's cleft cysts, craniopharyngiomas, germinal tumors, colloid cysts, and pituitary tumors.

On gross examination epidermoid cysts are globular, cystic, and well circumscribed with a smooth glistening surface and cheesy contents. They are easily separated from neighboring structures but since they may grow along nerve roots or other vital structures, complete excision may be impossible. Under microscopic examination the capsule consists of stratified squamous keratinizing epithelium and fibrous tissue; the cyst lumen contains squamous cells without nuclear cytoplasmic details. Under the electron microscope, the cyst lining is shown to be made up of typical keratinizing squamous epithelium.

Surgery is the most effective mode of therapy. As shown in our patients, removal of the entire cyst may not be possible in the suprasellar or cerebellopontine locations. In these instances, the contents should be drained; due to the slow growth of the cysts even incomplete removal often yields good results for many years.[12] Malignant degeneration of epidermoid cysts is rare although cases have been reported.[4,13]

REFERENCES

1. Ulrich J: Intracranial epidermoids. A study on their distribution and spread. *J Neurosurg* 1964; 21:1051-1058.
2. Obrador S, Lopez-Zafra JJ: Clinical features of the epidermoids of the basal cisterns of the brain. *J Neurol Neurosurg Psychiat* 1969; 32:450-454.
3. MacCarty CS, Leavens ME, Love G, Kernohan JW: Dermoid and epidermoid tumors in the central nervous system of adults. *Surg Gynecol Obstet* 1959; 108:191-198.
4. Toglia JU, Netsky MG, Alexander E Jr: Epithelial (epidermoid) tumors of the cranium. Their common nature and pathogenesis. *J Neurosurg* 1965; 23:384-393.
5. Wallenfang T, Schürmann K: Discovery of an epidermoid of the skull concomitant with a homolateral subacute traumatic subdural hematoma. *J Neurol* 1979; 221:213-218.
6. Choremis K, Economos D, Papadatos C, Gargoulas A: Intraspinal epidermoid tumours (cholesteatomas) in patients treated for tuberculous meningitis. *Lancet* 1956; 2:437-439.
7. Goldberg GM, Eshbaugh DE: Squamous cell nests of the pituitary gland as related to the origin of craniopharyngiomas. A study of their presence in the newborn and infants up to age four. *AMA Arch Path* 1960; 70:293-299.
8. Zivin I, Caprini JA: Craniopharyngioma: A review. *GP* 1967; 36:100-105.
9. Davis KR, Roberson GH, Taveras JM, New PFJ, Trevor R: Diagnosis of epidermoid tumor by computed tomography. *Radiology* 1976; 119: 347-353.
10. Corriero G, Maiuri F, Giamundo A, Colella G: Intracranial cholesteatoma extending in the middle and posterior fossa. *Acta Neurol (Napoli)* 1982; 37:180-184.
11. Gellad F, Rao C, Arora S, Chiantella N, Salcman M: Epidermoid tumor of the fourth ventricle: use of metrizamide-computed tomography. *CT* 1982; 6:231-235.
12. Love JG, Kernohan JW: Dermoid and epidermoid tumors (cholesteatomas) of central nervous system. *JAMA* 1936; 107:1876-1883.
13. Davidson SI, Small JM: Malignant change in an intracranial epidermoid. *J Neurol Neurosurg Psychiat* 1960; 23:176-178.

Primary osteogenic sarcoma of the skull

Five-year disease-free survival following surgery and high dose methotrexate therapy

NARAYAN SUNDARESAN, MD, JOSEPH H. GALICICH, MD, GERALD ROSEN, MD,
ANDREW G. HUVOS, MD

Osteosarcoma is the most common malignant tumor of bone; its incidence in the United States is estimated to be one per 100,000 of the population.[1] The majority of these tumors occur in the long bones of the extremities, and less than 10% originate in the craniofacial bones.[2] We report on a patient with primary osteosarcoma of the skull in a young patient who has remained disease-free five years following subtotal surgical resection and chemotherapy.

CASE REPORT

A 13-year-old girl complained of pain and swelling in the right temporal region after being hit by a softball in that area. Initial skull films were negative. A local physician diagnosed a possible ear infection. Following another episode of trauma, a right-sided mass was noted and skull films showed thinning of the right temporal bone. A neurologic examination was unremarkable except for a mild decrease in sensation along the maxillary division of the right trigeminal nerve. A computed tomographic (CT) scan showed a large cystic tumor with calcification involving the floor of the middle fossa (Fig 1). The clinical diagnosis was that of an aneurysmal bone cyst or giant cell tumor.

A right temporal craniotomy was performed, and the tumor was noted to involve the base of the skull medially to the gasserian ganglion. A subtotal excision of the tumor was performed, and a small portion involving the cavernous sinus was left untouched. Pathologic studies (Fig 2) revealed an osteoblastic osteogenic sarcoma.

The patient had an uneventful recovery following surgery. An extent of disease evaluation consisted of a bone scan, which was negative for metastatic disease, as was chest tomography. One month after the craniotomy high-dose methotrexate therapy was instituted at a dose of 12 g/m^2. Because the patient had some intracranial edema secondary to surgery, high-dose dexamethasone (16 mg/day) was given together with the methotrexate treatment. Each methotrexate treatment was followed by calcium leucovorin rescue, starting 24 hours after the beginning of the methotrexate infusion at doses of 10 mg orally every six hours for a total of 11 doses. Three days following each high-dose methotrexate treatment, the patient was instructed on the amount of fluid to drink to produce an alkaline urine output of approximately 1,800 cc/M^2 per 24 hours for three days following therapy. The urine was made alkaline by having the patient take oral sodium bicarbonate and checking the pH of each voiding. If the void did not show a pH of 7 or greater, the patient was instructed to take an additional dose of sodium bicarbonate. As a result, the patient took between 2 to 3 milliequivalents per kilogram body weight of sodium bicarbonate for 24 hours.

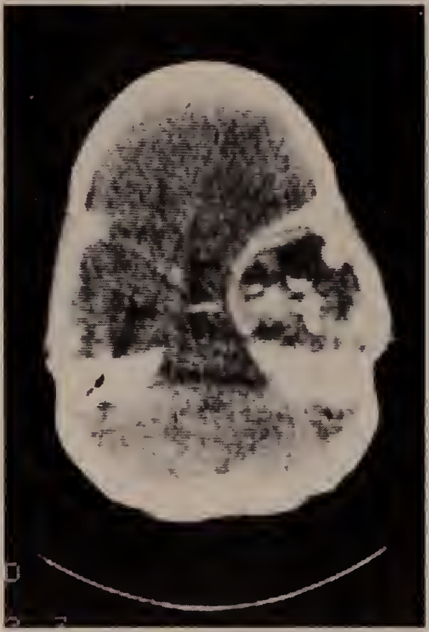


FIGURE 1. Axial computed tomographic scan with contrast showing lytic tumor with calcification in right temporal region.

After eight high-dose methotrexate treatments, an attempt was made to treat her with doxorubicin, which was part of our T_7 protocol.[6] This had to be discontinued after two doses because of persistent vomiting. As a result, high-dose methotrexate was continued for a total dose of 29 consecutive treatments bimonthly. Following cessation of treatment, she was followed at regular intervals by CT scans, bone scans, and chest tomography. She is currently free of disease 5½ years following treatment. Endocrine and skeletal growth are normal.

DISCUSSION

Osteosarcoma may arise de novo, may be secondary to a variety of bone disorders—such as Paget's disease and fibrous dysplasia—or may follow radiation therapy.[3] The majority of patients are initially seen with a painless skull mass, and neurologic symptoms are present in approximately 40% of patients at the time of initial presentation.[2] Unfortunately, due to the disease's relative rarity, the diagnosis is seldom entertained before surgery, and only limited surgical excisions are performed. Palliative radiation therapy has been advocated following surgery, but little evidence of its value can be found in the literature. For these reasons, osteosarcoma arising in the cranial bones has had a dismal prognosis, with five-year survival rates of approximately 10%.[4] During the past decade, however, a variety of chemotherapeutic agents have been found to be effective

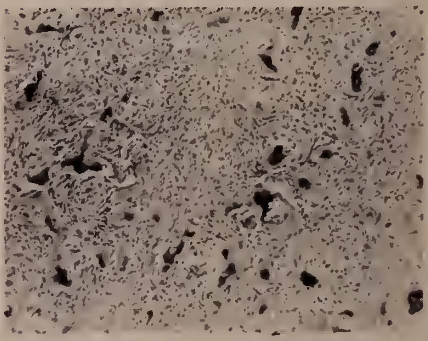


FIGURE 2. Malignant osteoblastic osteogenic sarcoma with calcified tumor osteoid formation. (hematoxylin and eosin stain; original magnification × 100)

From Memorial Sloan-Kettering Cancer Center, New York.
Address correspondence to Dr Rosen, Medical Director, Comprehensive Cancer Centers Inc, 407 N. Maple Dr, Beverly Hills, CA 90210.

in the treatment of this tumor.[5,6] These, coupled with advances in skull base surgery,[7] should now allow more aggressive and successful treatment of this tumor.

In a previous review of a series of 43 patients with osteosarcoma arising from the craniofacial bones at this institution, Caron found only one long-term survivor for calvarial tumors.[4] Similarly, Thompson noted only one 10-year survivor in a series of 14 patients with sarcomas arising from the calvarium; this patient had undergone complete surgical excision but received no other therapy.[8] There appears to be some heterogeneity in this tumor, and occasional long-term survivors have been reported among patients with predominantly extracranial tumors that do not involve the dura. For such patients, adequate surgical excision should preferably be an en bloc resection of the scalp, skull, and dura.[7,8] With the widespread availability of computerized tomography to assist in surgical planning, it is now possible to carry out more planned en bloc excisions than were possible previously.

Prior to 1972, the outlook for most patients with osteosarcoma was bleak, even following adequate surgery. In tumors of the extremities, the five-year survival rate was 20% because of early onset of pulmonary metastases. Modern chemotherapy regimens began in 1972, when high-dose methotrexate in dosages of 200 to 300 mg/kg was used to treat patients with osteogenic sarcoma.[9] Objective responses were observed in 4 of 13 patients treated with two doses of approximately 300 mg/kg. Following this report, Rosen and associates[6] developed combination chemotherapy protocols that incorporated high-dose methotrexate as the major treatment regimen for this tumor. The effective dose of high-dose methotrexate varies between 10 to 20 g for most patients, and translates to a dose of 8 to 9 g/m^2 for fully grown adolescents and adults and 12 mg/m^2 for children who have not reached adult stature. Systemic alkalinization with sodium bicarbonate is used to prevent the drug from precipitating in the renal tubules, thus preventing renal toxicity. In addition, calcium leucovorin is used to minimize systemic toxicity, and serum methotrexate levels are measured for three days following the start of treatment. With leucovorin rescue, high-dose methotrexate is administered weekly up to four consecutive doses. For patients who exhibit evidence of renal toxicity, alternate therapy should be substituted—for example, doxorubicin during the interim period. With the use of combination chemotherapy, Rosen reported an 82% continuous disease-free survival rate in 61 patients treated with the T_7 chemotherapy protocol with a median follow-up time of 36 months.[6]

At the present time, emphasis has shifted to treating most patients with biopsy-proven osteosarcoma with chemotherapy before surgery for periods ranging from six to eight weeks. Originally designed for patients scheduled for limb salvage surgery, this strategy (termed neoadjuvant chemotherapy) has several advantages: early treatment of presumed systemic micrometastases; reduction in primary tumor bulk; and identification of effective postoperative adjuvant chemotherapy based on tumor response. In addition, preoperative chemotherapy can be used to identify a high-risk group that does not respond to high-dose methotrexate. In these patients, alternative therapy with drugs such as doxorubicin and *cis*-platinum is substituted. With this approach continuous disease-free intervals of five years or more are now feasible for 80% of patients with lesions in the extremities.[11] As this case illustrates, a combination of aggressive surgery and chemotherapy may improve the prospects for long-term palliation and cure in most patients with osteosarcoma, regardless of the site of the lesion.

REFERENCES

1. Huvos AG: *Bone Tumors - Diagnosis, Treatment and Prognosis.* Philadelphia, WB Saunders, 1979, pp 47-127.
2. Huvos AG, Sundaresan N, Smith J, Bretsky SS, Butler A: Osteogenic sarcoma of the skull: a clinico-pathologic study of 19 patients. *Cancer.* (in press)
3. Huvos AG, Butler A, Bretsky SS: Osteogenic sarcoma associated with Paget's disease of bone. *Cancer* 1983; 52:1489-1495.
4. Caron AS, Hajdu SI, Strong EW: Osteogenic sarcoma of the facial and cranial bones: a review of 43 cases. *Am J Surg* 1971; 122:719-725.
5. Rosen G: A comprehensive guide to the therapeutic use of methotrexate in osteogenic sarcoma. *Pharma Libri,* Morristown, NJ, 1984.
6. Rosen G, Marcove RC, Huvos AG, et al: Primary osteogenic sarcoma: eight-year experience with adjuvant chemotherapy. *J Cancer Res Clin Oncol* 1983; 106s:55-67.
7. Voorhies R, Sundaresan N: Skull tumors, in Wilkins R, Rengacharry S (eds): *Textbook of Neurosurgery.* Baltimore, Williams & Wilkins, 1984.
8. Thompson JB, Patterson RH, Parsons H: Sarcomas of the calvaria: Surgical experience with 14 patients. *J Neurosurgery* 1970; 32:534-538.
9. Jaffe N: Recent advances in the chemotherapy of metastatic osteogenic sarcoma. *Cancer* 1972; 30:1627-1631.
10. Rosen G, Marcove RC, Caparros B, Nirenberg A, Kosloff C, Huvos AG: Primary osteogenic sarcoma: the rationale for preoperative chemotherapy and delayed surgery. *Cancer* 1979; 43:2163-2177.
11. Rosen G: Preoperative (neoadjuvant) chemotherapy for osteogenic sarcoma: a ten year experience. *Orthopedics* 1985; 8:659-664.

Silicone granulomas of the breast

GLADYS CRUZ, MD, JOHN F. GILLOOLEY, MD, MARIAN WAXMAN, MD

Since intramammary silicone injections have become popularized for augmentation mammoplasty, many complications of this procedure have been reported.[1-10] Local complications have included cyst formation, subcutaneous migration of the silicone from the intended site to other locations, hyperpigmentation of the skin over the injection site, infection, and gangrene.[2] Systemic complications due to embolization have included blindness and pulmonary edema—sometimes fatal.[3] The local complications, in which the injected silicone has induced severe granulomatous inflammation with subsequent formation of hard fibrous tumefactions in the breasts, are likely to pose a clinical problem. In a patient with a lump in her breast, it may be impossible to distinguish this inflammatory fibrous mass from a solid neoplasm, not only by pal-

---

From the Department of Pathology, St. Vincent's Hospital and Medical Center, New York.

Address correspondence to Dr Waxman, Department of Pathology, St. Vincent's Hospital and Medical Center, 153 West 11th St, New York, NY 10011.

pation but also using mammography. In this report we describe three patients with silicone-induced pseudotumors of the breast and discuss the diagnostic and therapeutic aspects of these often difficult cases.

CASE REPORTS

Patient 1. A 33-year-old woman was admitted to the hospital for removal of bilateral breast masses. She had received bilateral intramammary silicone injections in her native Taiwan ten years previously. The masses appeared about five years later and have enlarged gradually for five years since then. They were mobile, firm, non-tender, did not change with the menstrual cycle, and were not associated with nipple discharge or abnormalities of the overlying skin. The patient had a 17-month-old child whom she breast-fed. She had used birth control pills for two years in the distant past. She underwent cervical conization for carcinoma-in-situ one year prior to this admission. The abnormal physical findings were limited to the breasts: three separate masses—the largest 7 cm in size—were felt in the right breast, and two masses—the larger 5 cm in size—were found in the opposite breast. No mammogram was performed. All the masses were resected together with some adjacent breast parenchyma. The masses were firm, and at the cut section they appeared well-demarcated, with white glistening surfaces (Fig 1). The microscopic picture (Fig 2) was characterized by multiple, small cyst-like vacuoles of varying sizes and without epithelial lining. The frozen section slides showed outlines of an almost transparent foreign material (presumably the silicone mixture) within these vacuoles; the material was not refractile under the polarized light. This material was no longer seen on the sections cut from the paraffin blocks. The intervening stroma contained inflammatory exudate composed mainly of lymphocytes and macrophages, including multinucleated histiocytic giant cells of a foreign body type. In some areas the stroma showed dense collagenous fibrosis with few or no residual inflammatory cells. Irregular coarse calcification was seen occasionally within these fibrotic areas. The breast parenchyma within the masses was mostly destroyed, except for occasional surviving ducts and acini. The patient's recovery was uneventful, but she has failed to appear for the follow-up visits.

Patient 2. A 36-year-old woman sought treatment for a slowly enlarging mass in the left breast of six months' duration, not associated with nipple discharge and not related to menstruation. She had had bilateral silicone injections in her breasts seven years prior to this admission. A mammogram, performed five years after the injections, showed bilateral irregular densities consistent with reaction to silicone. Her medical history and family history were not contributory.

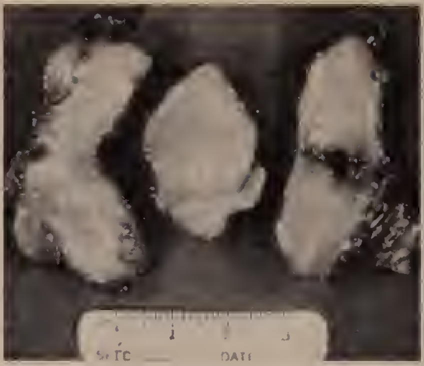


FIGURE 1. Cut surfaces of breast masses from Patient 1 showing solid uniform appearance.

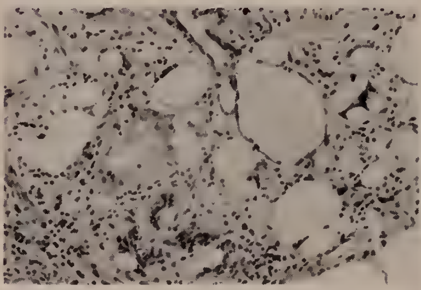


FIGURE 2. Multiple vacuoles and extensive inflammatory infiltrate with occasional multinucleated histiocytic giant cells seen in sample from Patient 1. A few residual ducts are also seen (bottom). (original magnification X 100)

Physical examination showed a distinct, firm, slightly tender area, about 3 cm in size, located in the midline of the left breast just above the areola. The right breast did not contain any distinct masses, and there were no palpable lymph nodes. The remainder of the examination was unremarkable. In addition to the bilateral irregular densities seen previously, a repeat mammogram demonstrated a new distinct mass in the left breast. Although there was no evidence of calcification or vascularity, and although this finding was consistent with reaction to silicone, the possibility of malignancy could not be excluded.

The mass was removed. It was a lobulated, rubbery, yellowish solid mass measuring 2.4 cm in largest diameter. Histologic analysis revealed the characteristic vacuoles with extensive fibrosis and with sparse inflammatory cells. The follow-up (six months) has been uneventful.

Patient 3. A 43-year-old woman was admitted to the hospital for treatment of bilateral breast deformities. She had had bilateral silicone injections into her breasts in Hong Kong 17 years prior to this admission. Both breasts were diffusely fibrotic and, in addition, the left breast contained a large firm mass in the left upper quadrant. The mammogram showed changes consistent with reaction to silicone. Surgery consisted of bilateral subcutaneous mastectomy and augmentation mammoplasty using silicone bag implant. Both specimens were similar in appearance, showing replacement of most breast parenchyma by a pale pink, dense, rubbery, firm tissue. There were occasional cystic spaces filled with clear, sticky, thick material. Microscopic examination showed extensive chronic granulomatous inflammation of foreign-body type, multiple vacuoles, small and large cystic spaces, and areas of fibrosis, with focal involvement of the adjacent pectoral muscle fibers. The follow-up (six months) has been uneventful.

DISCUSSION

The advantage of using pure form silicone (dimethylpolysiloxane) in breast augmentation has been that it is an inert substance which is not significantly metabolized in the body,[1] does not produce local inflammation, and does not evoke antibody formation.[1] The most undesirable property of the pure silicone, however, is its tendency to migrate in the tissues from the injected site to other locations,[5] with loss of the intended cosmetic effect. Certain oils or fatty acids have been added to the silicone with the idea of provoking a local inflammatory reaction that leads to fibrosis and fixation of the silicone to the injected site (Sakurai formula).[1] Although the migration is thus prevented, other complications may ensue, such as scar contraction with deformities or formation of fibrous masses (silicone granulomas) such as those that have occurred in these patients. The firm consistency of these tumors is due to dense collagenous fibrosis which is the final stage of the healing process of these silicone granulomas.

Several reports on silicone granulomas of the breast have been found in the literature.[1,6-9] In one patient a single "siliconoma" simulated carcinoma of the breast on both physical examination and mammography.[7] Another report describes migration of the injected silicone from the breast to the abdominal wall and inguinal area.[6] Other local complications described following silicone injections were infection, cyst formation, skin changes including hyperpigmentation, lymphedema, vesicle formation, ulceration, and even gangrene.[2] Four cases of silicone embolism were recently described.[3] These young women developed acute respiratory distress syndrome four hours to seven days after the injections, manifested by dyspnea, cough, blood-streaked sputum, vomiting, and chest pain. There was electrocardiographic evidence of typical pulmonary embolism in two patients.

Three patients survived, but one expired 36 hours after the onset of symptoms.[3] Similarly, acute pneumonitis and respiratory failure have been described in transsexual men within 24 hours of silicone injections into breast and trochanteric areas.[10] Interestingly, spilled silicone particles from the blood-pump tubing caused granulomatous hepatitis with fibrosis in hemodialysis patients.[11]

There is no evidence of carcinogenicity of injected silicone. Although breast cancer has developed in some patients following augmentation mammoplasty, the relationship of these events appeared to be incidental.[12] However, since the breast is an organ so prone to development of malignancy, the pressure of such an inflammatory-fibrous mass may pose a difficult clinical problem of differentiating it from a carcinoma in any individual patient. The most important clues in the diagnosis of silicone granuloma that help to distinguish it from cancer are the following: a history of silicone injections (not always volunteered by the patient); bilaterality and multiplicity of the lesion; and diffuse irregular nodularity of the breasts. Characteristic mammographic appearances of silicone-injected breasts[7] have been described. Sometimes, however, the mammogram can be equivocal (as in patient 2) or even suggestive of cancer.[7] Only pathologic examination of the excised mass can assure one of the benign nature of the lesion. The diagnosis can be made with certainty by frozen section.

A patient with a known history of intramammary silicone injections in whom a breast lesion develops could be managed without surgery if the lesion is diffuse, bilateral, has a typical mammographic appearance consistent with a silicone reaction, and occurs without a discrete mass.

Finally, silicone-induced fibrosis and granulomas of the breast may cause a delay in discovery of a small carcinoma because of diffuse nodularity of the breast. This should be kept in mind when treating women with a high risk for breast cancer (based on a positive family history) before selecting silicone injections as a method of augmentation mammoplasty.

REFERENCES

1. Nosanchuk JS: Silicone granuloma in breast. *Arch Surg* 1968; 97:583-585.
2. Kopf EH, Vinnik CA, Bongiovi JJ, Dombrowsky DJ: Complications of silicone injections. *Rocky Mt Med J* 1976; 73:77-80.
3. Lee C-M, Kou H-T, Lin C-C, Huang W-C: Adult respiratory distress syndrome following augmentation of breast. *J Form Med Assoc* 1982; 81:1043-1047.
4. Terino EO: Essential concepts in prosthetic breast surgery. *Aesthetic Plast Surg* 1982; 6:25-32.
5. Mason J, Apisarnthanarax P: Migratory silicone granuloma. *Arch Dermatol* 1981; 117:366-367.
6. Delage C, Shane JJ, Johnson FB: Mammary silicone granuloma. Migration of silicone fluid to abdominal wall and inguinal region. *Arch Dermatol* 1973; 108:105-107.
7. Kuiper DH: Silicone granulomatous disease of the breast simulating cancer. *Mich Med* 1973; 72:215-218.
8. Baruch J, Wechsler J, Bodin B, Raulo Y: Slowly evolving breast siliconomas. *Ann Chir Plast* 1982; 27:183-184.
9. Symmers W St C: Silicone mastitis in "topless" waitresses and some other varieties of foreign-body mastitis. *Br Med J* 1968; 3:19-22.
10. Chastre J, Basset F, Viau F, et al: Acute pneumonitis after subcutaneous injections of silicone in transsexual men. *N Engl J Med* 1983; 308: 764-767.
11. Leong AS, Disney AP, Gove DW: Spallation and migration of silicone from blood-pump tubing in patients on hemodialysis. *N Engl J Med* 1982; 306:135-140.
12. Hoopes JE, Edgerton MT Jr, Shelley W: Organic synthetics for augmentation mammoplasty: their relation to breast cancer. *Plast Reconstr Surg* 1967; 39:263-260.

Erythroleukemia in the adjuvant treatment of breast cancer

STUART M. LICHTMAN, MD, PHILIP SCHULMAN, MD, DANIEL R. BUDMAN, MD

The development of acute nonlymphocytic leukemia as a consequence of the treatment of neoplastic[1,2] and nonneoplastic[3] illness is a well recognized phenomenon that occurs in the treatment of multiple myeloma,[4] Hodgkin's disease,[5] non-Hodgkin's lymphoma,[6] ovarian carcinoma,[7,8] and other malignancies.[1] In most instances, the development of treatment-related leukemia has been associated with the use of alkylating agents. In recent years, multidrug protocols, often combined with radiation, have become a significant part of the therapy for many solid tumors. In this setting an increased incidence of treatment-induced leukemia may be expected.

We recently observed a patient with breast carcinoma who developed acute myelogenous leukemia after receiving adjuvant therapy.

CASE REPORT

A 62-year-old woman sought treatment for dysarthria, lethargy, and right-sided weakness in July 1983. She had undergone a left radical mastectomy in December 1980 for a 6.5-cm mass. Pathologic analysis of the mass revealed infiltrating ductal carcinoma with vascular and lymphatic invasion. Axillary nodes were negative, and the tumor was positive for both estrogen and progesterone receptors. After surgery she received phenylalanine mustard, 10 mg/day for six days every six weeks for one year (266 mg/m^2 total dose).

In December 1981 she complained of right hip pain. Roentgenographs were consistent with metastatic disease to the right pubic ramus; she received 2,400 rad to that area and was placed on tamoxifen. In September 1982, pain recurred, and roentgenographs showed mottling of the right hemipelvis. In November 1982, she received 2,500 rad to that area and was switched to fluoxymesterone.

The patient's condition was stable, including a normal white blood cell count and platelet count, until July 1983 (the current admission), 32 months after the initial diagnosis of breast cancer. A hemogram showed a hemoglobin level of 9.6 gm%; white blood cell count, 7,900/μL (70% polymorphonuclear leukocytes, 10% band forms, 15% lymphocytes, 3% monocytes); and platelets,

---

From the Don Monti Division of Oncology, Department of Medicine, North Shore University Hospital, Manhasset, NY, and the Department of Medicine, Cornell University Medical College, New York, NY.

Address correspondence to Dr Lichtman, Division of Oncology, North Shore University Hospital, 300 Community Dr, Manhasset, NY 11030.

TABLE. Breast Cancer and Acute Leukemia

	Number of Patients	Treatment*	Interval[†] (Months)	Type[‡]
Preisler et al[15]	1	Radiation therapy	NS	ANLL
		Alkylating agents		
Portugal et al[16]	4	Radiation therapy (0)	—	ANLL (4)
		Chemotherapy (2)	72	
		Both (2)	67	
		None (0)	—	
Ershler et al[17]	11	Radiation therapy (1)	96	ANLL (11)
		Chemotherapy (3)	54	
		Both (5)	64	
		None (2)	18	
Smith et al[18]	1	Radiation therapy		
		Alkylating agents	28	
Rosner et al[19]	24[§]	Radiation therapy (11)	85	
		Chemotherapy (5)	61	ALL (1)
		Both (2)	45	ANLL (23)
		None (6)	111	
	54[‖]	Radiation therapy (16)		
		Chemotherapy (8)		ALL (3)
		Both (6)	NS	ANLL (23)
		None (8)		NS (13)
		NS (16)		
Della Cuna et al[20]	1	Alkylating agents	13	ANLL (1)
Gori et al[21]	1	Alkylating agents	39	ANLL (1)
Smadja et al[22]	2	None (1)	0	ANLL (1)
		Both (1)	54	ALL (1)
Bezwoda et al[23]	2	Both (1)	18	ANLL (2)
		Chemotherapy (1)	14	

* Indicates number of patients receiving each therapy.

[†] Interval is from date of breast cancer diagnosis to diagnosis of acute leukemia. Numbers indicate averages depending on therapy employed.

[‡] Indicates number of patients having specified type.

[§] Rosner et al's own series.

[‖] Rosner et al's literature review.

NS = Indicates data not sufficient.

16,000/μL. A peripheral blood smear contained nucleated red blood cells and no erythroblasts. A computed tomographic (CT) scan of the head showed a large area of intracerebral bleeding. Bone marrow aspiration revealed erythroid hyperplasia consisting of 75% erythroblasts, 20% normal erythroid elements, and 5% normal myeloid elements. The blasts were positive for periodic acid-Schiff (PAS), consistent with a diagnosis of erythroleukemia. Cytogenetic analysis revealed two abnormal clones, 45,xx,−7 and 45,xx,−7,19q+, and a normal, 46,xx clone. Therapy was initiated with high-dose cytosine arabinoside. The patient was given an abbreviated course of four doses because of central nervous system toxicity, possibly exacerbated by the underlying structural lesion. A bone marrow aspiration study on day 17 showed 8% blasts; on day 24 the test findings were compatible with complete remission. Cytogenetic analysis at that time was normal. After discharge and restoration of normal hemopoietic parameters, a follow-up CT scan was consistent with metastatic breast carcinoma. Peripheral blood counts were normal. By November 1983, the leukemia was in relapse. The patient expired in December 1983.

Discussion

The patient described parallel previous reports of leukemia developing concurrently with myeloma or ovarian cancer. The patient underwent prolonged alkylating therapy followed by local radiation. Thirty-two months after initiation of treatment, the patient presented with acute nonlymphocytic leukemia (ANLL). She had multiple cytogenetic abnormalities and marked dyserythropoiesis. Cytochemical stains were consistent with erythroleukemia (M6 by FAB).[9] Despite the lack of a pancytopenic phase, this patient represents treatment-induced leukemia because of the combined modality therapy, including alkylating agents, an appropriate time interval from onset of therapy, and the cytogenetic abnormalities reported. This patient also confirms the ability of high dose cytosine arabinoside to induce remission in secondary ANLL.

Since the 1970 report of Kyle et al linking ANLL with the treatment of multiple myeloma,[4] the leukemogenic potential of chemotherapy, particularly alkylating agents, has been increasingly recognized. Casciato and Scott[1] noted that the usual patient was treated with chemotherapy for 42 months, and developed leukemia 48 months after starting chemotherapy (60 months after the diagnosis of the underlying disease). A preceding cytopenic period can be seen as in other preleukemia states. Associated bone marrow abnormalities, such as hyperplastic marrow with severe dysplasia of cell lines, are usually noted prior to frank leukemia. The majority of the patients reported developed symptoms associated with erythroleukemia and myelomonocytic leukemia. A similar preceding pancytopenia is also seen with exposure to bone marrow toxins such as benzene. Secondary ANLL manifests nonrandom chromosomal abnormalities involving chromosomes 5 and 7.[10] These same abnormalities may also be seen with toxic exposure. It has also been established that chemotherapeutic agents themselves may be carcinogenic. The alkylating agents are felt to have the highest leukemogenic risk as determined by both in vivo animal studies and clinical reviews of patients treated with these agents. Several agents used in the adjuvant treatment of breast carcinoma are among those found to be carcinogenic. These include cyclophosphamide, doxorubicin hydrochloride, and vincristine sulfate.[11]

A review of the literature on breast carcinoma and leukemia is seen in the Table. In several cases the two diagnoses were made simultaneously. Cytogenetics has not been evaluated systemically. Because of the diversity of therapy for breast carcinoma, no statistically significant conclusions can be drawn except for the very common occurrence of alkylating agents. Survival rates in the reported cases were uniformly poor and rarely exceeded five months. Most of the reports shown are retrospective and lack accurate reporting of leukemic subtype, dose or type of chemotherapeutic drug, dose of radiation therapy, latency time between therapy and development of leukemia, and cytogenetic evaluation. Therefore, the association of breast cancer therapy and ANLL is difficult to glean from the literature. The recently published National Cancer Institute's Surveillance, Epidemiology, and End Results Program (SEER) was an attempt to quantify leukemic risk with various solid tumors after one course of treatment.[12] A significantly increased risk was found in the group treated with chemotherapy as a first course. There was also an increase in the observed cases of ANLL for the group treated with radiation alone. Although leukemia was observed in the group treated with surgery alone, the number did not increase over the rate in the general population. The report by Boice et al[13] concerning the possible leukemogenic effect of semustine in the adjuvant therapy of gastrointestinal cancer is an outstanding example of the studies required for adjuvant breast cancer therapy to better define the incidence, type of leukemia, time of leukemia, and therapy. Nowell et al[14] did cytogenetic analyses of patients' bone marrow after adjuvant therapy alone and found no abnormalities in a limited series. However, nonrandom chromosomal abnormalities have been associated with secondary leukemia. Thus it is possible that chemotherapy induces a chromosomal aberration that may cause abnormal stem cells to proliferate. Pancytopenia and hypercellular marrow with dysplasia in all three cell lines may ensue. These abnormalities may be clues to diagnosing secondary ANLL.

It would therefore seem that one or both of the following mechanisms are operating. The first is that chemotherapy causes an evolution from normal bone marrow to a leukemic transformation by creating a dysmyelopoietic state which then follows its own natural history, with many patients developing ANLL. Nowell's findings of abnormal marrow genetics after adjuvant therapy may be a reflection of sampling for cytogenetic changes at too early a time period to detect stem cell damage. A second possibility is that these patients either are predisposed to or already have an unrecognized dysmyelopoietic state that is aggravated or accelerated by adjuvant therapy.

With an increasing number of patients with breast carcinoma being entered into adjuvant regimens, the problem of treatment-induced leukemia is of major concern. Many of these adjuvant courses of therapy contain carcinogenic drugs. Also, the nature of adjuvant therapy in certain settings remains in question. The value of chemotherapy in postmenopausal patients has been difficult to substantiate. This is particularly true of those patients with favorable prognostic factors (ie, negative nodes, positive estrogen receptors). Therefore, further prospective randomized controlled trials of adjuvant breast cancer therapy, with accurate recording of the variables that will help establish the causes of therapy-induced leukemia, are needed. Then one can determine whether the risk of secondary leukemia is outweighed by the increased survival and improved quality-of-life offered by adjuvant chemotherapy.

Acknowledgment. The authors thank Dr Eugene Gillman for referring this patient.

REFERENCES

1. Casciato DA, Scott JL: Acute leukemia following prolonged cytotoxic agent therapy. *Medicine* 1979; 58:32–47.
2. Zarrabi MH, Rosner F: Acute myeloblastic leukemia following treatment for non-hematopoietic cancers. *Am J Hematol* 1979; 7:357–367.
3. Grünwald HW, Rosner F: Acute leukemia and immunosuppressive drug use. *Arch Intern Med* 1979; 139:461–466.
4. Kyle RA, Pierre RV, Bayrd ED: Multiple myeloma and acute myelomonocytic leukemia. *N Engl J Med* 1970; 283:1121–1125.
5. Aisenberg AC: Acute nonlymphocytic leukemia after treatment for Hodgkin's disease. *Am J Med* 1983; 75:449–454.
6. Zarrabi MH, Rosner F, Bennett JM: Non-Hodgkin's lymphoma and acute myeloblastic leukemia. *Cancer* 1979; 44:1070–1080.
7. Reimer RR, Hoover R, Fraumeni JF Jr, Young RC: Acute leukemia after alkylating-agent therapy of ovarian cancer. *N Engl J Med* 1977; 297:177–181.
8. Greene MH, Boice JD Jr, Greer BE, Bleising JA, Dembo AJ: Acute nonlymphocytic leukemia after therapy with alkylating agents for ovarian cancer: a study of five randomized clinical trials. *N Engl J Med* 1982; 307:1416–1421.
9. Bennet JM, Catovsky D, Daniel MT, et al: Proposals for the classification of the acute leukaemias. *Br J Haematol* 1976; 33:451–458.
10. Pedersen-Bjergaard J, Philip P, Pedersen NT, et al: Acute nonlymphocytic leukemia, preleukemia, and acute myeloproliferative syndrome secondary to treatment of other malignant diseases. *Cancer* 1984; 54:452–462.
11. Schilsky RL, Erlichmann C: Late complications of chemotherapy: infertility and carcinogenics, in Chabner B (ed): *Pharmacologic Principles of Cancer Treatment*. Philadelphia, WB Saunders Co, 1982, pp 109–131.
12. Curtis RE, Hankey BF, Myers MH, Young JL Jr: Risk of leukemia associated with the first course of cancer treatment: an analysis of the Surveillance, Epidemiology, and End Results Program experience. *JNCI* 1984; 72:531–544.
13. Boice JD Jr, Greene MH, Killen JY Jr, et al: Leukemia and preleukemia after adjuvant treatment of gastrointestinal cancer with semustine (methyl-CCNU). *N Engl J Med* 1983; 309:1079–1084.
14. Nowell P, Glick JH, Bucolo A, Finan J, Creech R: Cytogenetic studies of bone marrow in breast cancer patients after adjuvant chemotherapy. *Cancer* 1981; 48:667–673.
15. Preisler HD, Early AP, Raza A, et al: Therapy of secondary acute nonlymphocytic leukemia with cytarabine. *N Engl J Med* 1983; 308:21–23.
16. Portugal MA, Falkson HC, Stevens K, Falkson G: Acute leukemia as a complication of long-term treatment of advanced breast cancer. *Cancer Treat Rep* 1979; 63:177–181.
17. Ershler WB, Robins HI, Davis HL, et al: Emergence of acute non-lymphocytic leukemia in breast cancer patients. *Am J Med Sci* 1982; 284:23–31.
18. Smith AG, Prentice AG, Lucie NP, Browning JD, Dagg JH, Rowan RM: Acute myelogenous leukaemia following cytotoxic therapy: five cases and a review. *Q J Med* 1982; 51:227–240.
19. Rosner F, Carey RW, Zarrabi MH: Breast cancer and acute leukemia: report of 24 cases and review of the literature. *Am J Hematol* 1978; 4:157–172.
20. Della Cuna GR, Cuzzoni Q, Parieri L, Marin G: Acute nonlymphoblastic leukemias. *Eur J Cancer* 1978; 14:1157–1158.
21. Gori S, Donti E, Venti G, Mecucci C, Crinó L, Tonato M: Acute myeloblastic leukemia after adjuvant chemotherapy with melphalan in breast cancer. Case report with cytogenetic analysis. *Tumori* 1983; 69:117–122.
22. Smadja N, Krulik M, Genot JY, Audebert AA, Debray J: Hematologic and cytogenetic study of two cases of acute leukemia associated with breast cancer. *Cancer Genet Cytogenet* 1983; 9:185–196.
23. Bezwoda WR, Hofman K, Bothwell TH, Derman DP, MacPhail AP, Mendelow B: Bone marrow damage due to melphalan and other cytostatic agents. *S Afr Med J* 1980; 58:479–484.

Massive lower gastrointestinal hemorrhage secondary to colonic varices

CHARLES C. CANVER, MD, JAMES F. UPSON, MD

Massive rectal bleeding often presents difficult diagnostic and therapeutic problems. Varices of the colon are a rare cause of hemorrhage, although bleeding is a common symptom of this condition. In 2,912 consecutive autopsies, the frequency of colonic varices has been found to be 0.07%.[1] Since the first report in 1954[2] there have been 33 cases cited in the world literature. We present the case of a patient with micronodular cirrhosis in whom severe colonic hemorrhage developed from right-sided colonic varices. This case illustrates the necessity of considering this entity in patients with cirrhosis who are seen because of persistent lower gastrointestinal bleeding.

CASE REPORT

A 49-year-old woman with a history of alcohol abuse and micronodular cirrhosis was admitted to the hospital for treatment of abdominal discomfort in the right lower quadrant and rectal bleeding. A week previously, she had been admitted to another hospital with rectal bleeding. Endoscopy at that time showed nonbleeding esophageal varices and a prepyloric channel ulcer. A colonoscopic examination was done to the level of the midtransverse colon. The colonic mucosa was completely normal without evidence of diverticula, vascular malformations, dilated submucosal veins, or mucosal erosions. Bright red blood was seen coming from the hepatic flexure into the transfer colon. Because of the large amount of blood coming from the hepatic flexure, the examination could not be extended to the ascending colon and cecum. A barium enema was unremarkable. The patient required 14 units of blood transfusion during a six-day hospital stay and was then transferred to the Buffalo General Hospital.

On the day of admission she felt well except for mild lower abdominal discomfort. On physical examination she was noted to be very obese and alert with stable vital signs. Her abdomen was soft and nontender, but did have a fluid wave and shifting dullness to percussion suggestive of ascites. The liver was palpable just beneath the right costal margin and had a 12-cm span to percussion. The splenic tip was also palpable. She had a serosanguineous drainage from the umbilicus. On rectal examination no obvious external hemorrhoids or palpable anorectal masses were found, but there was bright red blood on the examining finger.

The results of laboratory studies on admission showed a hematocrit of 31.0%, and a hemoglobin level of 10.6 g/dL, with normal serum electrolyte, blood urea nitrogen, and serum creatinine levels. Other laboratory test results were as follows: serum albumin, 2.0 g/dL; serum total bilirubin, 5.0 mg/dL; serum glutamic oxaloacetic transaminase, 52 IU/L; and alkaline phosphatase, 69 IU/L.

Initially, transfusion with four units of packed red cells was carried out, but the patient continued to have intermittent bright red rectal bleeding; hemoglobin and hematocrit values dropped to 9.4 g/dL and 28.7%, respectively. On the morning of the first hospital day, the patient underwent abdominal angiography. The inferior and superior mesenteric arteries and celiac artery were selectively catheterized. Filming was carried out through the venous phase. No intraluminal extravasation of contrast material was shown. Large, dilated cecal and ascending colonic veins were identified. No other mesenteric varices were seen.

She required eight units of packed cells during the first 24 hours of hospital admission. At laparotomy, 36 hours after admission, a large cirrhotic liver was noted. An end-to-side portacaval shunt was made. The bleeding stopped following portacaval shunting, but refractory ascites persisted, and the patient developed marked oliguria. She underwent a peritoneo-venous shunt (Denver) procedure for the refractory ascites. Her condition improved gradually, and she had an uneventful hospital course. She was sent home free of complications and remains well without further rectal bleeding, anemia, or guaiac-positive stools.

DISCUSSION

Extra-esophageal portosystemic anastomoses or varices are a well-recognized but uncommon feature of portal hypertension. Communications between the portal and systemic venous systems, which normally exist only as potential anastomoses, occur in the esophagus, terminal ileum, ascending and descending colon, retroperitoneum, and anterior abdominal wall.[3] These veins may dilate and become varices as the result of increased pressure or flow associated with portal hypertension, mesenteric vein obstruction, congestive heart failure, congenital venous anomalies,[1] and intraperitoneal adhesions.[4]

Portal hypertension secondary to cirrhosis is the most common cause of colonic varices. As the portal blood flow diminishes secondary to increasing hepatic resistance, flow and subsequently pressure are increased within portosystemic collateral channels. These veins, which are not well supported by surrounding tissue, become ectatic and varicose. The most significant portosystemic collateral anastomosis is between the left gastric and coronary veins and the azygos system via the esophageal veins. Utilizing splenoportography, Britton[5] showed that esophageal collaterals are the primary anastomotic channels in at least half of all patients with portal hypertension. The other main channel was the inferior mesenteric left iliac venous system. This system was involved as a primary collateral in about one third of the patients.

This patient also had varices of the right colon associated with portal hypertension and ascites. The initial symptom was bright red bleeding from the rectum. Diagnosis of colonic varices may be done through radiographic or

---

From the Department of Surgery, State University of New York at Buffalo, The Buffalo General Hospital.

Address correspondence to Dr Canver, 1459 Parker Boulevard, Buffalo, NY 14223.

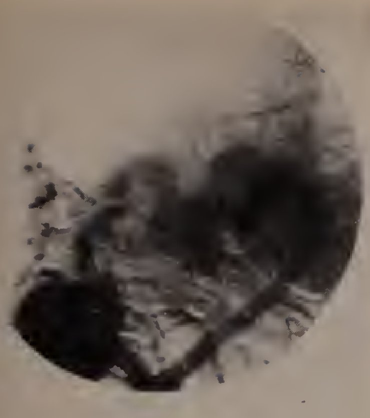


FIGURE 1. Markedly dilated and tortuous veins can be seen in the ascending colon and cecum.

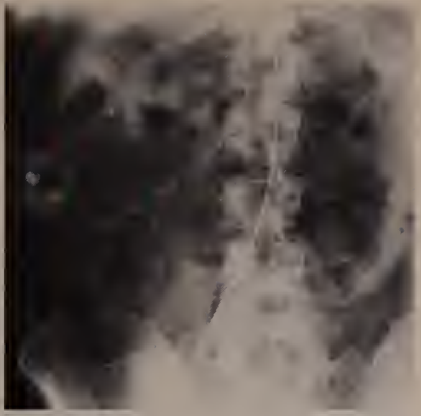


FIGURE 2. Venous phase of superior mesenteric angiogram showing cecal varices.

endoscopic means. In this case, a barium enema was reported as being negative and colonoscopy failed to confirm a diagnosis. Despite previous reports, we believe that colonoscopy, though widely available, is unlikely to provide definitive diagnosis in patients who are bleeding vigorously from mesenteric varices. An unprepared colon filled with stool and clots is an unacceptable endoscopic field. Barium enema was a poor diagnostic tool because of misinterpretation of findings. Some varices were called air bubbles or fecal material, others were thought to be carcinoma of the colon.

The role of angiography is firmly established in diagnosing arterial hemorrhage from the lower gastrointestinal tract. In this case, utilizing arteriography, the varices were visualized in the venous phase (Figs 1,2). Venous phase angiography seems to be the most reliable and accurate diagnostic tool in diagnosis of right colonic varices.

Controversy exists over whether primary resection or portacaval shunting is the procedure of choice when patients fail to respond to medical therapy. Despite the occurrences of death in patients who underwent colon resection, the patients who underwent portosystemic shunting have all survived. No long-term follow-up is available, but it seems that portacaval shunting is safer in the short-term for massive variceal bleeding. With primary portal decompression, resection of unprepared bowel with its attendant morbidity is avoided. The advantages of a shunt procedure outweigh the potential disadvantages, such as encephalopathy and liver failure. In the unprepared bowel, portal venous surgical decompression is preferred to colonic resection.

Despite its rarity in patients with portal hypertension, colonic varices should be considered as another potential source of massive lower gastrointestinal hemorrhage, and appropriate diagnostic and therapeutic steps should be taken.

REFERENCES

1. Feldman M, Smith VM, Warner CG: Varices of the colon. *JAMA* 1962; 179:729-730.
2. Case Records of the Massachusetts General Hospital. (Case #40102). *N Engl J Med* 1954; 250:434-436.
3. Edwards EA: Functional anatomy of the portasystemic communications. *Arch Intern Med* 1951; 88:137-154.
4. Burbige EJ, Tarder G, Carson S, Eugene J, Frey C: Colonic varices—a complication of pancreatitis with splenic vein thrombosis. *Dig Dis* 1978; 23:752-755.
5. Britton RC: Influence of portal-systemic patterns and distribution of varices on results of surgical treatment of bleeding esophageal varices. *Surgery* 1963; 53:567-574.

Massive hemoptysis due to retained rib fragment 40 years after thoracotomy

FREDERICK A. BEVELAQUA, MD, ROSEMARY WIECZOREK, MD, ARTHUR D. BOYD, MD

The major causes of massive life-threatening hemoptysis are tuberculosis, lung abscess, bronchiectasis, and carcinoma.[1] A less frequent cause of bleeding from the bronchial tree is erosion of a broncholith through the wall of a bronchus. Foreign body aspiration and pulmonary arteriovenous fistula are also uncommon causes of massive hemoptysis. We report on a patient with life-threatening hemoptysis due to a retained bone fragment from a rib resection that had been performed for drainage of an empyema 42 years previously. The rib fragment had eroded through the bronchial wall causing an area of marked, localized bronchiectasis that was associated with the development of a bronchial arteriovenous malformation.

CASE REPORT

A 49-year-old man was admitted to the hospital because of massive hemoptysis. Two days previously he had had an episode of massive hemoptysis (more than 600 cc), which subsided spontaneously.

Forty-two years earlier, at the age of 7, he had a left-sided rib resection for drainage of an empyema which left him with a left anterior chest wall deformity. He had had epi-

---

From the Departments of Medicine (Dr Bevelaqua), Surgery (Dr Boyd), and Pathology (Dr Wieczorek), New York University Medical Center, New York.

Address correspondence to Dr Bevelaqua, 15 Park Ave, Suite 1-N, New York, NY 10016.

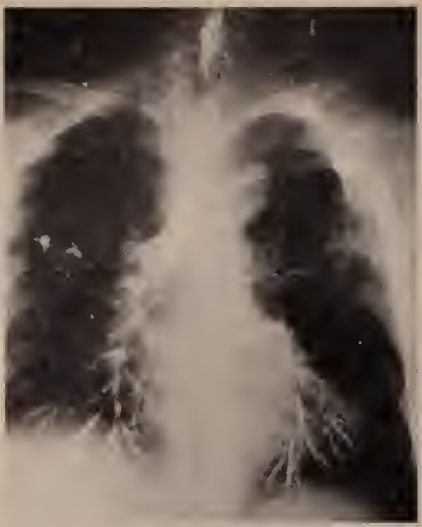


FIGURE 1. Bronchogram showing mild bronchiectatic changes in the right upper lobe. The left upper lobe was not visualized, but the remainder of the bronchogram was normal.

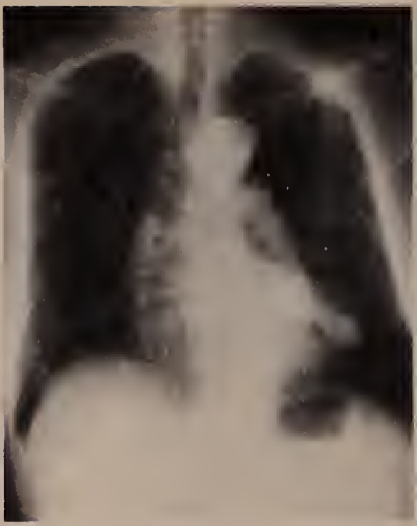


FIGURE 2. Chest roentgenogram on admission showing deformity of the left anterior chest wall.

sodes of intermittent mild hemoptysis since the age of 20.

Following admission to the hospital, the bleeding again subsided spontaneously, only to recur 24 hours later. This time the bleeding resulted in respiratory arrest, necessitating the insertion of an endotracheal tube and the use of mechanical ventilation. Once again the hemoptysis ceased spontaneously.

While the endotracheal tube was in place bronchograms were obtained. Mild bronchiectatic changes were noted in the right upper lobe (Fig 1). The right middle, right lower, and left lower lobes were unremarkable; however, the left upper lobe was not well visualized.

After three days the endotracheal tube was removed. Within two days he experienced a fourth episode of massive hemoptysis and respiratory decompensation which resulted in reinsertion of the endotracheal tube, using a double lumen tube. When his condition had stabilized, the patient was brought to the operating room for rigid and flexible bronchoscopy, but the source of the bleeding could not be determined. The bleeding again stopped spontaneously, and the patient was transferred to New York University Hospital for further diagnostic evaluation and treatment. Prior to his transfer he had received a total of five units of blood.

Figure 2 shows a chest film taken on admission to University Hospital. There is evidence of left chest wall deformity and pleuroparenchymal scarring due to the thoracotomy he had undergone in childhood. A computerized axial tomographic (CAT) scan of his chest (Fig 3) done with contrast showed the left anterior chest wall deformity. The depression caused by the chest wall deformity looked like a cavity on CAT scan, but this was not confirmed by the whole lung linear tomography which was also done. Neither the CAT scan nor the linear tomography detected any endobronchial lesions. Minimal bronchiectatic changes were noted on CAT scan in the right upper lobe. No definite evidence of an arteriovenous malformation was seen on either the CAT scan or the linear tomograms.

The patient next underwent a pulmonary angiogram to look for evidence of an arteriovenous malformation that might have been responsible for the bleeding. The pulmonary angiogram showed a persistent "blush" in the left upper lobe but no definite evidence of a pulmonary arteriovenous malformation (Fig 4). Following this, he underwent a bronchial arteriogram which showed definite evidence of a bronchial arteriovenous malformation in the left upper lobe which was presumed to be responsible for the bleeding (Fig 5).

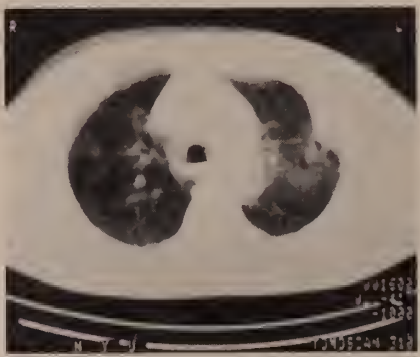


FIGURE 3. CAT scan of the chest with contrast showing deformed ribs of the left anterior chest wall and a cavity-like area in the left upper lobe. No evidence of arteriovenous malformation is seen and no foreign body is apparent.

With this information, a thoracotomy and resection of the left upper lobe were performed. At the time of surgery a jagged, irregular bone fragment measuring 3.5 × 0.7 × 0.3 cm was found penetrating the bronchus leading to the anterior segment of the left upper lobe (Fig 6). There was marked focal dilation of the bronchus at the point of attachment of the bone, with a great deal of surrounding fibrosis and chronic inflammation. There was also evidence of recent hemorrhage in the bronchus.

A radiograph of the surgical specimen showed the bone to be densely radiopaque. Study of the gross morphologic and microscope features of the bone together with the clinical history suggested that this fragment was derived from rib that had been curretted during the drainage of the patient's empyema 42 years previously. Since the removal of his left upper lobe more than one year ago the patient has had no further episodes of hemoptysis.

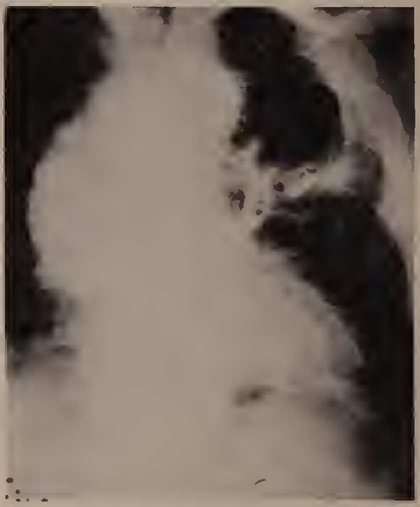


FIGURE 4. Pulmonary angiogram showing persistent "blush" suggestive of increased vascularity in the anterior segment of the left upper lobe.

DISCUSSION

Hemoptysis may be the presenting complaint in a patient who has no other symptoms. Any portion of the respiratory tract may be the source of the bleeding. On occasion, blood from the nose or nasopharynx is aspirated and then expectorated. However, bleeding from the upper respiratory tract can usually be distinguished from the lower respiratory tract on the basis of the patient's history and the physical examination. If the upper airway can be ruled out as the site of the beeding, then a

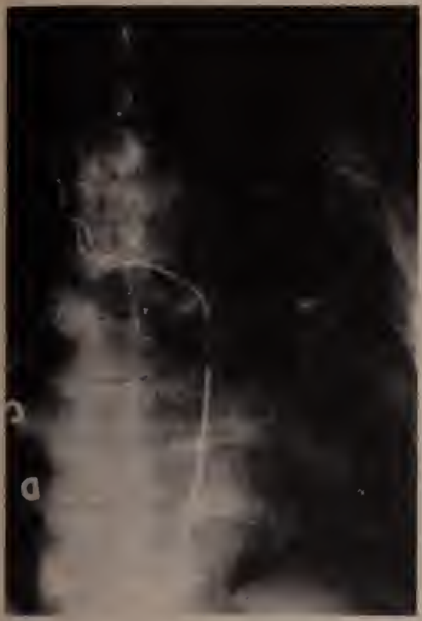


FIGURE 5. Bronchial arteriogram showing small bronchial arteriovenous malformation in the left upper lobe.

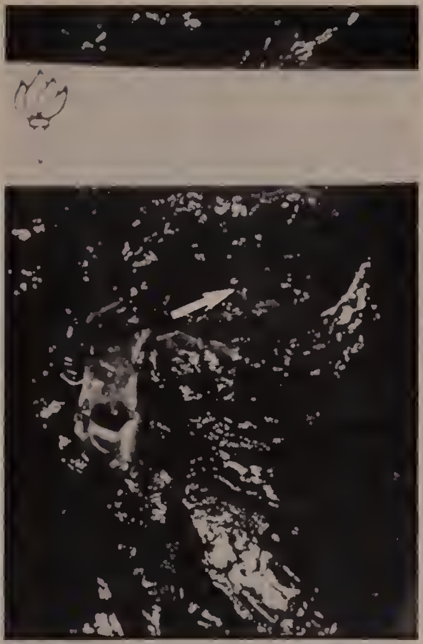


FIGURE 6. Resected left upper lobe with bone fragment (arrow) in the anterior segmental bronchus.

conscientious effort must be made to localize the intrapulmonary source. The causes of lower airway bleeding are numerous and include such relatively common entities as acute and chronic bronchitis, tuberculosis, mycetoma, pneumonia, pulmonary infarction, bronchogenic carcinoma, cardiovascular disorders, and bronchiectasis. Foreign body aspiration, broncholiths, blood dyscrasias, and Goodpasture's syndrome are other less common causes of hemoptysis.

Documenting the location of the bleeding in hemoptysis can be difficult if the cause is not immediately obvious on the basis of the patient's history, physical examination, and chest radiograph. Fortunately, most episodes of hemoptysis are not life threatening, and most spontaneous bleeding stops of its own accord.[1] However, massive hemoptysis with the production of 400 cc to 600 cc of blood or more is associated with high mortality. Crocco and associates[2] found a mortality rate of 75% in patients who lost more than 600 mL of blood within 16 hours. Asphyxia caused by the aspiration of blood is the usual cause of death in these situations. The most likely causes of such massive hemoptysis are tuberculosis, lung abscess, carcinoma, and bronchiectasis. Of these, bronchiectasis is probably the most common, but it may also be the most difficult to document and to localize.

In any case of massive hemoptysis the critical problem is to determine the source of the bleeding. The fiberoptic bronchoscope has enhanced the physician's ability to identify the site of bleeding in hemoptysis, and it should be employed in all patients with hemoptysis of unknown cause. However, even with bronchoscopy, the cause of the bleeding may be missed. Some studies have indicated that the site of the bleeding can be visualized or the segment identified by means of bronchoscopy in 80% to 90% of patients with hemoptysis.[3,4] On the other hand, Gong and Salvatierra[5] were able to identify the active site of bleeding in only 40% of patients.[5] If bronchoscopy fails to reveal the source of the bleeding, then bronchography may be needed to identify the presence of an endobronchial lesion or bronchiectasis. However, computed tomography of the thorax is becoming a more effective tool for documenting small endobronchial lesions and bronchiectasis.[6,7] If these studies fail to reveal the cause or location of the bleeding, then pulmonary angiography and bronchial arteriography may be necessary to demonstrate the presence of abnormal vessels. Occasionally arteriography and embolization have been used to treat patients with massive hemoptysis who have not been candidates for surgery.[8]

Hemoptysis tends to be a recurring problem in patients with bronchiectasis, and the source of the bleeding is often difficult to demonstrate even by the techniques described above. Although hemoptysis due to bronchiectasis usually ceases spontaneously, massive episodes can occur because of the bronchial arterial collateral circulation and bronchial-pulmonary artery anastomoses that can develop as a result of the chronic inflammation.[9,10] Bronchiectasis can be the result of a number of processes. It sometimes develops following endobronchial infection or pneumonia. It can also be caused by congenital or hereditary disorders that result in bronchial abnormalities which lead to recurrent infection. Cystic fibrosis, immune deficiency states, vascular and lymphatic abnormalities, chronic bronchitis, and long-standing bronchial asthma can all lead to the development of bronchiectasis.

The patient described here depicts a rather unusual cause of recurrent, life-threatening hemoptysis due to bronchiectasis that developed as a result of a foreign body. At surgery the patient was unexpectedly found to have a rib fragment lodged in the anterior segment of the left upper lobe. The severity of the bronchiectatic changes indicated that the bone had been present for a long time, and the presence of fresh clot established this location as the source of the bleeding. Morphologic studies and the patient's history led to the conclusion that this bone fragment came from rib that had been curretted during the patient's rib resection for empyema drainage in childhood. The bone must have penetrated the lung and entered the anterior segmental bronchus of the left upper lobe leading to the development of severe bronchiectasis over the course of many years.

This case demonstrates the importance of careful investigation to localize the site of bleeding in patients with hemoptysis. In this instance the chest film, apart from showing the old left anterior chest wall deformity, was not helpful in determining the location of the bleeding. The limited bronchograms that were obtained showed some evidence of bronchiectasis in the right upper lobe, but since the left upper lobe was not visualized this area could not be excluded as the source of bleeding. Although the recurrent episodes of hemoptysis were massive, intubation with a double lumen tube also failed to elucidate the site of the bleeding because the episodes ended before a determination could be made as to whether the blood was emanating from the right or the left lung. Bronchoscopy, both rigid and flexible, did not reveal the site of bleeding or the bone fragment in the anterior segment of the left upper lobe. This may have been due to the fact that this segment is sometimes difficult to visualize even with the flexible bronchoscope. Neither thoracic CAT scan with contrast nor linear tomography of the lung showed any parenchymal abnormalities to account for the bleeding. The bone fragment was relatively small, and the concave, anterior chest wall deformity may well have obscured the anatomy enough to prevent detection of the bone fragment. The vascular abnormalities detected on the pulmonary angiogram and the bronchial arteriogram were too subtle to be demonstrated on the CAT scan even with contrast; however, they were obviously significant enough to be responsible for the episodes of massive hemoptysis which the patient experienced. The finding of a bone fragment lodged within this patient's bronchus was totally unexpected at surgery, but it easily accounts for the severity of the bronchiectasis and the associated vascular abnormalities which led to the life-threatening hemoptysis.

Acknowledgments. Peggy Caldwell, MA and Irving Seidman, MD reviewed the pathologic specimens. Dorothy McCauly, MD and Robert Rosen, MD reviewed the radiologic studies.

REFERENCES

1. Fishman AP: Approach to the patient with pulmonary disease, in Fishman AP (ed): *Pulmonary Diseases and Disorders*. New York, McGraw-Hill Book Co, 1980, pp 72–78.

2. Crocco JA, Rooney JJ, Fanhushen DS, DiBenedetto RJ, Lyons HA: Massive hemoptysis. *Arch Intern Med* 1968; 121:495–498.

3. Purcel SE, Lindskog GE: Hemoptysis: a clinical evaluation of 105 patients examined consecutively on a thoracic surgical service. *Am Rev Respir Dis* 1961; 84:329–336.

4. Smiddy JF, Elliot RC: The evaluation of hemoptysis with fiberoptic bronchoscopy. *Chest* 1973; 64:158–162.

5. Gong H Jr, Salvatierra C: Clinical efficacy of early and delayed fiberoptic bronchoscopy in patients with hemoptysis. *Am Rev Respir Dis* 1981; 124:221–225.

6. Naidich DP, McCauley DI, Siegelman SS: Computed tomography of bronchial adenomas. *J Comput Assist Tomogr* 1982; 6:725–732.

7. Naidich DP, McCauley DI, Khouri NF, Stitik FP, Siegelman SS: Computed tomography of bronchiectasis. *J Comput Assist Tomogr* 1982; 6:437–444.

8. Bredin CP, Richardson PR, King TKC, et al: Treatment of massive hemoptysis by combined occlusion of pulmonary and bronchial arteries. *Am Rev Respir Dis* 1978; 117:969–973.

9. Tadavarthy SM, Klugman J, Castaneda-Zuniga WR, Nath PH, Amplatz K: Systemic-to-pulmonary collaterals in pathologic states. *Radiology* 1982; 144:55–59.

10. Liebow AA, Hales MR, Lindskog GF: Enlargement of the bronchial arteries and their anastomoses with the pulmonary arteries in bronchiectasis. *Am J Pathol* 1948; 25:211–219.

See also p 573.

THE GREAT AMERICAN SMOKEOUT

The ninth annual Great American Smokeout, the American Cancer Society's (ACS) effort to encourage persons to give up smoking for the day, will take place on Thursday, November 21, 1985.

The idea for the smokeout originated in Minnesota in 1974. Lynn R. Smith, editor of the *Monticello Times*, solicited pledges from among his 3,000 subscribers, asking that they quit "cold turkey" for one day. He called the event "D-Day," and several hundred persons participated. When it was repeated in 1975, the entire state went along. Thousands of Minnesotans took part, and the Minnesota Division of the American Cancer Society became involved. In 1976, the California division of the ACS borrowed the idea and gave the event its present name. When almost 1.5 million Californians participated, the smokeout received national attention.

By 1980, the Smokeout was a national event, with participation by more than 14 million persons.

Surveys show that most people who smoke would like to stop, and the smokeout provides a target date to stop each year. Many individuals report that it takes more than one attempt to stop completely; therefore, the smokeout provides a meaningful target date, bolstered by community support.

In 1983, the national smokeout program added the concept of "Adopt-A-Smoker." This gives the entire public an opportunity to take an active role in the smokeout by "adopting" a smoking friend or coworker for the day. Their role is to encourage their "adoptee" in every way possible to give up cigarettes.

On the eve of the Smokeout in 1984, the ACS, the American Lung Association, and the American Heart Association cosponsored "Breathing Easy," a nationally televised program on smoking, aimed at teenagers and children.

Few national programs achieve the same level of public awareness, degree of participation, and stimulus for better health as the Great American Smokeout. One indication of its success is that in 1984 the Tobacco Institute chose smokeout day to announce to the media the results of a survey it commissioned, reporting that more than 70% of the Americans surveyed felt that the funds allocated by ACS to the Smokeout ($250,000) would be better spent on research.

DAVID LEHMANN
American Cancer Society
4 West 35 Street
New York, NY 10001

This section is designed to offer rapid publication (usually within 1-3 months) of new clinical, epidemiologic, and laboratory observations, as well as of comments on articles published in the Journal *or on any topic related to the role of the physician. Letters should be typed double-spaced and should include the professional affiliation, highest academic degree, address, and signature of each author. Preference is given to letters not exceeding 450 words. All letters are personally reviewed and acknowledged by the Editor. Address correspondence to Editor,* New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042.*

BRIEF REPORTS

Porcelain gallbladder in a 24-year-old woman

TO THE EDITOR: Porcelain gallbladder, or calcification in the gallbladder wall, is an unusual radiologic finding. It almost always occurs in the presence of chronic obstruction of the cystic duct, but rarely causes symptoms. Hence its detection is usually adventitious. Porcelain gallbladder is more than a curiosity, however, because of its association with carcinoma of the gallbladder.[1,2] Gallbladder wall calcification is found predominantly in women and almost always in patients over age 40. We report on a young woman with a calcified gallbladder who had no known predisposition to liver disease or cholelithiasis.

Case Report. A 24-year-old woman was admitted to Jacobi Hospital because of abdominal pain. She had experienced such pain intermittently for seven years. She was not obese and she had had no children. A plain film of the abdomen demonstrated a curvilinear calcification in the right upper quadrant (Fig 1). Oral cholecystography did not enable visualization of the gallbladder. A cholecystectomy was performed; pathologic examination revealed calcification in the gallbladder wall and impaction of the cystic duct by a single calculus. She has had no signs or symptoms of hepatic abnormalities in the eight years since the operation.

Discussion. Calcified gallbladder was first recognized in 1797 and first described based on radiologic findings in 1909.[3] The term porcelain refers to the gallbladder's translucent bluish tint and its fragility at gross examination. Porcelain gallbladder is a rare disorder, found in only 0.07% of cholecystectomy specimens. Obstructing calculi are invariably present, yet these stones are seldom of sufficient opacity to be seen on plain radiographs. Since the cystic duct is blocked when the gallbladder wall is calcified, the lumen of the gallbladder will not be filled by contrast material during an oral cholecystographic examination. Gallbladder wall calcification is usually distinctive on a plain film of the abdomen, appearing as an ovoid or pear-shaped density delimited by slightly irregular and often discontinuous lines of radiopacity. The differentiation between gallbladder calcification and other cystic calcification in the right upper quadrant is achieved with oblique films. Cysts in the right kidney and the right adrenal gland will remain close to the spine on this projection, while the gallbladder will move anteriorly.

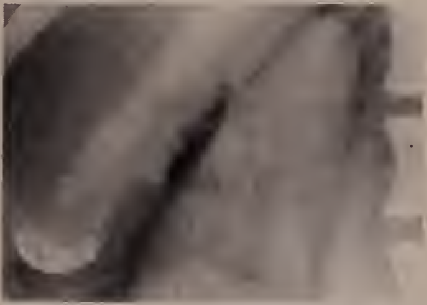


FIGURE 1. Oblique plain film of the abdomen demonstrates the anterior location of the calcified gallbladder. The right renal shadow is directly posterior to the gallbladder.

In addition to the present case there have been only two other examples of porcelain gallbladder in patients under age 40. Agarwal[4] studied a 38-year-old woman with a calcified gallbladder who was otherwise well. Aksoy[5] reported gallbladder calcification in a 35-year-old woman with thalassemia intermedia. The patient presented here is apparently the youngest individual with a porcelain gallbladder yet reported in the English literature.

Cholecystectomy appears warranted in healthy young adults and middle-aged patients with gallbladder wall calcification. The risk of carcinoma is probably higher in this group of patients, and the gallbladder is non-functional. However, elective cholecystectomy for elderly or debilitated patients with this disorder should not be an automatic procedure. The inevitability of malignant transformation remains to be established and the incidence and severity of complications of cholecystectomy increase in patients with other medical problems.

STEPHEN R. BAKER, MD
SHALOM BUCHBINDER, MD
Albert Einstein College of Medicine
1300 Morris Park Ave
Bronx, NY 10461

1. Cornell CM, Clarke R: Vicarious calcification involving the gallbladder. *Ann Surg* 1959; 149:267-272.
2. Etala E: Cancer de le vesicula biliar. *Prensa Med Argent* 1962; 49:2283-2299.
3. Ochsner SF, Carrera GM: Calcification of the gallbladder ("porcelain gallbladder"). *Amer J Roentgen* 1963; 89:847-853.
4. Agarwal R, Pandey RP, Gupta SN: Porcelain gall bladder. *J Indian Med Assoc* 1982; 79:80-81.
5. Aksoy M, Camli N, Dirvana S, et al: "Porcelain gallbladder" in a case of thalassemia intermedia. *Radiology* 1970; 95:265-266.

Thrombophlebitis: An unusual cause of chest pain

TO THE EDITOR: Thrombophlebitis of the chest wall is a rare cause of chest pain and may simulate pleuropulmonary disease. Originally described by Mondor,[1] the most important predisposing factors are trauma and infection. It is a benign, self-limiting condition lasting a few weeks or months and requiring no specific therapy.[2]

Case Report. A 29-year-old man sought treatment with a complaint of severe right-sided chest pain. He was found to have a painful, tender erythematous and an indurated cordlike structure extending from the lower axilla approximately six centimeters in length. There was no adenopathy or evidence of skin infection. The patient was otherwise in good health and had no clinical evidence of underlying disease. He did not smoke and consumed six to eight ounces of scotch weekly. There was no history of operations, allergy, trauma, or intake of medications. He had had type IV hyperlipidemia. Laboratory studies revealed a normal complete blood cell count, SMA 6, SMA 12, protein electrophoresis, and cryoglobulin level. The erythrocyte sedimentation rate was 6 mm/h. A chest film and electrocardiogram were within normal limits. A biopsy showed evidence of sclerosing thrombophlebitis (Fig 1). The patient had a complete recovery within three months.

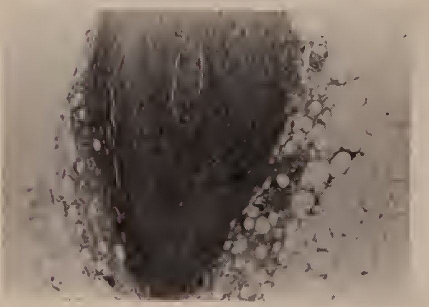


FIGURE 1. Sclerosing superficial thrombophlebitis; small subcutaneous vein obliterated by dense fibrous tissue with perivascular foci of lymphocytes and plasma cells (hematoxylin and eosin; original magnification X 100).

DISCUSSION

Thrombophlebitis of the chest wall is a clinical curiosity whose importance lies in the fact that it may simulate more serious disease. In 1939, Mondor[1] reported on four patients who had cordlike lesions in the subcutaneous lateral thoracic wall. They each had a history of trauma, and each had a complete recovery. In 1962, Bircher et al[3] compiled seven cases of Mondor's disease; each patient had been in good general health. None of the patients had a history of trauma, and all the lesions cleared within a few months. Mondor's disease does not harbor an underlying process.[3] Clinical awareness of thrombophlebitis as a cause of chest pain provides reassurance to both patient and physician.

DANIEL FAIERMAN, MD
The Mount Sinai School of Medicine
One Gustave L. Levy Place
New York, NY 10029

JOE H. DWEK
Booth Memorial Medical Center
Main St at Booth Memorial Ave
Flushing, NY 11355

1. Mondor H: Phlebiteen cordon de la paroi thoracique. *Mem Acad Chir* 1944; 70:96-98.
2. Ludbrook J, Jamison JC: Disorders of veins, in Sabiston DC Jr (ed): *David-Christopher Textbook of Surgery*. Philadelphia, WB Saunders Co. 1981, pp 1815-1816.
3. Bircher J, Schirger A, Clagett OT, Harrison EG: Mondor's disease: a vascular rarity. *Proc Mayo Clin* 1962; 37:651-656.

A Large Swallowed Foreign Body in the Esophagus

TO THE EDITOR: Swallowed foreign bodies in the esophagus are common in children and mentally disturbed adults. Bones, coins, and pins are among the most commonly swallowed foreign bodies. An unusually large foreign body that lodged in the esophagus of a schizophrenic young man is reported below.

Case Report. A 20-year-old man had allegedly swallowed a knife three days previously. He complained of painful swallowing, hematemesis, tachypnea, and dyspnea. Chest examination revealed a right-sided hydropneumothorax. However, examination of the abdomen and cardiovascular system were within normal limits. Psychiatric evaluation revealed features of schizophrenia. Radiograph studies revealed a large radiopaque object lying in the chest and abdomen (Fig 1).

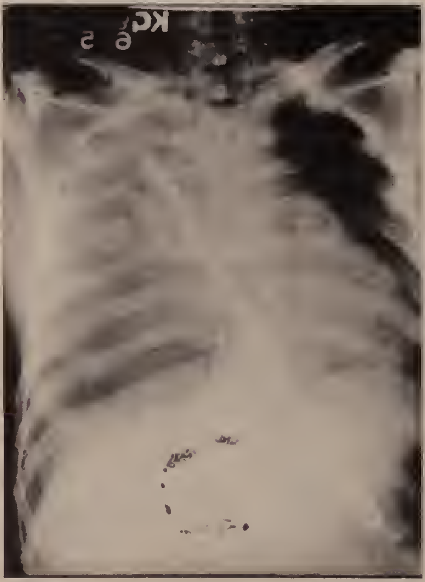


FIGURE 1. Roentgenogram showing a large radiopaque foreign body lying in the chest and the abdomen.

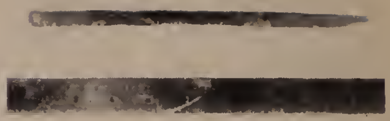


FIGURE 2. A knife, measuring 24 cm in length.

Blood and air were drained from the right pleural cavity by an intercostal tube and a laparotomy was then performed. The lower end of the knife was palpated in the stomach. A gastrotomy was performed and a 24-cm long knife was removed (Fig 2). The cardia was closed and a cervical esophagostomy was performed to provide total diversion of the damaged esophagus. An esophageal bypass was later performed by placing a segment of the colon presternally in the subcutaneous plane. The patient was eating normally at discharge and at subsequent follow-up.

Discussion: Children and mentally deranged adults commonly swallow foreign bodies causing serious injuries to the esophagus and adjacent structures. If perforation does not occur, foreign bodies in the esophagus are removed by esophagoscopy. However, surgical intervention is required if esophageal perforation does occur. If treated promptly, perforation can be repaired and adequate drainage provided. If treatment is delayed, however, the following must be undertaken: drainage of the mediastinum and the pleural cavity; decompression of the proximal esophagus and the stomach; and provision of adequate nutrition. Esophageal continuity can be restored later.

Bones, coins, pins, buttons, dental objects, and a food bolus are commonly swallowed objects found in the esophagus. A large number of unusual swallowed foreign bodies have, however, been reported in the literature: a 16.8-cm long knife[1]; a single 16.5-cm long dinner fork[2]; a straight handled razor[3]; 13-cm long spoon handles[4]; and multiple dinner forks[5].

As reported above, a schizophrenic young man swallowed a knife that perforated the esophagus and injured the lung, causing hemopneumothorax. He came to the hospital for treatment more than three days after the injury; therefore, primary repair of the esophagus was not attempted. The patient survived his injury, and food passage was restored later with the interposition of a colonic segment. The swallowed foreign body, a 24-cm long knife, is the largest reported in the literature to date.

V. K. KAPOOR, MS
A. K. KRIPLANI, MS
HEMANT SINGHAL, MBBS
M. C. MISHRA, MS
T. K. CHATTOPADHYAY, MS
L. K. SHARMA, MS, FICS
Department of Surgery
All India Institute of
Medical Sciences
New Delhi, 110029 India

1. Stough DF: Knife swallowing: report of a case. *J Okla Med Assoc* 1918; 11:331.
2. Lees KA: Dinner fork in stomach and duodenum. *Lancet* 1919; 1:298.
3. Carp L: Foreign bodies in gastrointestinal tracts of psychotic patients. *Arch Surg* 1950; 60:1055–1075.
4. Davidoff E, Towne JB: Ingested foreign bodies. *NY State J Med* 1975; 75:1003–1007.
5. Ghani A, Hashmi MS: Large foreign bodies of the gastrointestinal tract. *Int Surg* 1981; 66:271–272.

Glomus tumor of the clitoris

TO THE EDITOR: The first case of glomus tumor was described by Masson[1] in 1924. This lesion, most often located in the nailbeds and fingertips, has aroused much interest since reports of its occurrence in other sites such as the stomach, trachea, mediastinum, kidney, penis, uterus, vagina, bones, joints, skeletal muscle, and soft tissues.[1–6] The following is a report of what we believe to be the first case of a glomus tumor of the clitoris.

Case Report. A 27-year-old woman was admitted to the hospital with a history of dyspareunia for the past seven months. Her menstrual, obstetric, and medical histories were unremarkable.

Physical examination was negative. Examination of the perineum revealed a healed, second degree laceration. A pinkish-white ovoid mass measuring 1 × 0.5 cm was noted on the clitoris. The mass was tender to palpation. Removal of the mass with repair of the perineal laceration was performed.

The excised specimen consisted of a pinkish-white oval mass measuring 1 × 1 × 0.5 cm with a smooth surface. Cut section revealed a homogeneous pinkish-white slimy interior. Microscopic examination showed a well-circumscribed, nodular lesion comprised of small, uniform-appearing epithelioid cells. The cells were round to polyhedral and occasionally spindly, and the tumor contained slit-like vascular spaces lined by endothelial cells. There was no evidence of nuclear atypia or mitoses. Scattered areas of mucoid degeneration were seen in the stroma (Fig 1). Prominent nerve bundles were noticed in focal areas.

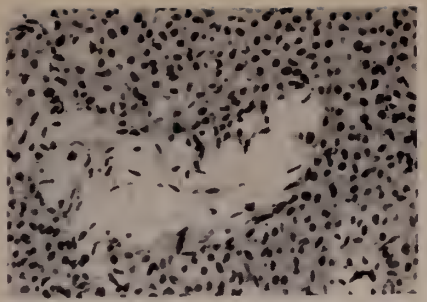


FIGURE 1. Uniform population of glomus cells with vascular spaces and a focus of myxoid degeneration. (hematoxylin and eosin stain, original magnification × 250)

The patient remained symptom free during follow up visits.

DISCUSSION

The glomus tumor or glomangioma is a neoplasm derived from the specialized cells of the glomus apparatus—an arteriovenous shunt concerned with local adjustments of vascular flow in response to certain mediators.[1–4] Current evidence on the basis of electron microscopy indicates that the characteristic epithelioid cells of the tumor are of smooth muscle origin and not the pericytes of Zimmermann as previously thought.[6] Some individuals believe that the glomus tumor represents a developmental disorder rather than a true neoplasm due to its morphology, long history, and absence of growth in most cases.[5,6]

The most common symptom of glomus tumors is pain with tenderness of varying degrees. In a series of 63 patients described by Tsuneyoshi and Enjoji,[6] 58 patients had either or both of these findings. The duration of symptoms at first presentation ranged from ten days to 30 years.[6] In our patient, the duration of symptoms lasted for several months, and the lesion presented as dyspareunia.

These tumors are found most often in the dermis and subcutaneous tissue. On gross examination the tumors measure approximately 0.5 to 2 cm and are round to ovoid with a smooth surface. On microscopic examination they are either encapsulated or well circumscribed, as in our case. An organoid pattern with numerous vascular channels is evident. The glomus cells are uniform in morphology and surround the vascular spaces. Mucinous degeneration and prominent nerve bundles in the vascular stroma, as noted in our case, may be found.

It is interesting to note that despite the rich vascularity of the clitoris, glomus tumors have not been described in this location before. Also, the tumor manifested as dyspareunia, which was cured with the excision of the lesion.

V. JAGADHA, MD
K. SRINIVASAN, MD
P. PANCHACHARAM, MD
Department of Pathology
New York Medical College
Lincoln Medical Center
234 East 149th St
Bronx, NY 10451

1. Masson P: Les glomus neuromyoarterial des regions tactiles et ses tumeurs. *Lyon Chir* 1924; 21:257–280.
2. Murray MR, Stout AP: Glomus tumor; investigation of its distribution and behavior, and identity of its "epithelioid" cell. *Am J Pathol* 1942; 18:183–203.
3. Stout AP: Tumors of the neuromyo arterial glomus. *Am J Cancer* 1935; 24:255–272.
4. Shugart RR, Soule EH, Johnson EW Jr: Glomus tumor. *Surg Gynec Obstet* 1963; 117:334–340.
5. Appleman HD, Helwig EB: Glomus tumors of the stomach. *Cancer* 1969; 23:203–213.
6. Tsuneyoshi M, Enjoji M: Glomus tumor: a clinicopathologic and electron microscopic study. *Cancer* 1982; 50:1601–1607.

Correction: Procainamide versus Procan SR®

TO THE EDITOR: In our letter that appeared in the May issue of the *New York State Journal of Medicine* (1985; 85:197) there has been a misprint. The last line states that "we no longer prescribe procainamide," when in fact what we no longer prescribe is Procan SR®. This is a critical difference in that this one particular product (Procan SR®) has been associated with neutropenia while procainamide does not have such a strong association. It is Procan SR® that we no longer prescribe, not procainamide.

LASZLO BOROS, MD
Co-Division Head
Oncology/Hematology Division
The Wasyl Pluta Medical Oncology Center
The Genesee Hospital
224 Alexander St
Rochester, NY 14607

ORIGINS AND EVOLUTION OF BEHAVIOR DISORDERS. FROM INFANCY TO EARLY ADULT LIFE

By Stella Chase, MD and Alexander Thomas, MD. 320 pp. New York, Brunner/Mazel Publishers, 1984. $30.00 (hardcover)

This book is perhaps not properly titled, since it is actually an attempt to develop a unified approach to the origins and predictability of psychiatric illness. It derives from conclusions drawn from a 25-year study of the behavior, psychiatric functioning, and well-being of children from 133 families. The authors argue that despite a broad knowledge base—including Freudian analytic concepts, behavioristic concepts and all their numerous offshoots, and information about genetic forces and biochemical disorders affecting behavior and personality—these theories are insufficient to explain, much less predict, the development and course of psychiatric illness. The central theme of the book is that only an integrated approach using all these concepts can be useful, and that even then there are many gaps in the current knowledge.

Drs Chase and Thomas are respected authorities in the field, making this remarkable contribution must reading for psychiatrists and workers in the mental health field. The book's approach is unique in many ways; it outlines what is known and what is not known about the entities classified as DSM-III disorders. Thus the book is an indispensable addition to any psychiatric library.

The book is based on the New York Longitudinal Study. This study included intensive efforts to follow up on participants, to define factors which seemed to be significant in terms of predictability of future psychiatric disturbance, and to offer statistical analyses to support the findings. The authors admit, however, that their findings require further study. The analyses are enriched by clinical vignettes of some of the participant-subjects, as well as by discussions of therapeutic interventions such as parental guidance, psychotherapy, and drug treatment.

The authors use two key terms, "good fit" and "poor fit," the first referring to a child whose innate temperament "fits" the demands and wishes of the parents and environment. The main criteria used have to do with whether or not a child is naturally flexible and able to cope with the degree of parental accord or discord and the demands made by parents and the surrounding society. Not surprisingly, when the child is flexible and the parents and environment "fit" the child's temperament, the outcome is expected to be good; if not, the opposite is said to occur. Yet, although this generalization was found to be true in most instances, there were some surprising results. In each case, explanations are sought and presented. It is emphasized, however, that it is not these factors alone that determine the onset or evolution of psychiatric disturbance. "It is rather the continuous interactional process among . . . influential factors which shape the development course . . . of children." And even then, this interaction is continuous throughout life, so that among many children in whom the development of psychiatric disorders might be expected, change remains possible and has been shown to occur.

The authors' observations are meticulously documented and statistically analyzed. I have not seen anything in the literature to compare with the exhaustiveness of the approach. The only possible criticisms might be that the terms "good fit" and "bad fit" are repeated too often, together with other concepts. As a result, the flow of the book suffers. Each chapter might make an independent paper in itself and a few have been used as such. But this in no way diminishes the refreshing lack of dogmatism, and the openness and willingness to say "We don't know." In fact, the authors know a great deal, communicate it well in the book, and open up a world of speculation to the reader, while adding to an understanding of the subject.

IRWIN J. BERKMAN, MD
Harlem Hospital
New York City 10037

DOPPLER ECHOCARDIOGRAPHY

By Stanley J. Goldberg, MD, Hugh D. Allen, MD, Gerald R. Marx, MD, Celia J. Flinn, MD. 178 pp, illustrated. Philadelphia, Lea & Febiger, 1985. $30.00 (hardcover)

Dr C. J. Doppler would have been pleased to see the growing scientific use for the principle he first described in 1842. We have all experienced what Doppler codified, namely the effect of relative motion on perceived frequency of a wave signal. Clinical use of this worthwhile observation is now flowering into full development.

This text is one of several dealing with the application of Doppler ultrasound to the examination of cardiac structures. The authors have written extensively in the field, focusing on the application of this method to pediatric cardiology. Their text covers a broad range of applications of Doppler echocardiography in both adults and children.

The book is written in a terse style. The structure of the text resembles a clinical handbook which makes the book practical for repeated use as a guide. On the other hand the choppy, frequent, and short paragraphs do not lend to a leisurely overview of this important subject. By comparison with other texts in the field such as the text, *Doppler Ultrasound in Cardiology* by Hatle and Angelsen, the format does seem rushed.

However, this style does not detract from the vast amount of information presented and the extensive references provided in the text.

The book itself begins (after an introduction and history which I found quite interesting) with a brief section on Doppler physics which makes the subsequent text easier to understand. A chapter on the technical aspects of performing a Doppler examination is both useful and practical. Subsequent chapters deal with the calculation of disturbed flow, outputs, and pressure gradients.

Each short section is immediately followed by a brief series of references, an interesting departure from the usual style of collecting references at the end of the chapter. Dispersing the references is quite helpful in focusing on each area. The discussions are very much to the point and therefore the book must be read carefully, probably more than once for a full appreciation of the correlation between text and illustration. A positive feature of the book is the juxtaposition of text with appropriate illustrations. On the whole, opinions are conservatively stated and the authors consistently provide references to substantiate the statements in the text.

In conclusion, I do not believe that this text represents any great advance over published material already available. The book is much too technical and detailed for any but the most motivated medical student or house officer but lacks the depth and breadth that a consultant cardiologist would like from a text covering this subject. It seems to be perfect for cardiology fellows in training who need a "hands-on" text which is brief, to the point, and full of practical applications, ably assisted by more than adequate references.

MARTIN M. KAY, MD
Booth Memorial Medical Center
Flushing, NY 11355

CHRONIC ULCERS OF THE SKIN

By Bok Y. Lee, MD. 225 pp. illustrated. New York, McGraw-Hill Book Co, 1985. $27.00 (hardcover)

This book is primarily concerned with decubitis ulcers or pressure sores. It is divided into three general sections on evaluation, prevention, and management. The 15 chapters are authored by various specialists, including biomedical engineers, nursing specialists, orthopedic surgeons, plastic surgeons, neurosurgeons, and general surgeons. The writing is clear, specific, and authoritative.

In gathering data on pressure sores, a specific evaluation or assessment form was used by nurses, resulting in a numerical score which indicated the probability of patients developing ulcers. Important factors were activity, mobility, and mental stability.

There is a review of the physiologic factors involving the different kinds of pressure and resultant ischemia and ulceration. Special management problems of various injuries, such as burns, which result in pressure sores are also discussed. Most of the book is concerned with cases involving burns or cases involving central nervous system injury. In order to arrive at the best plan of management, statistical analyses from various centers are detailed.

Surgical techniques are given extensive coverage. Among these, bone scrapings have proven to be superior to other techniques for the detection of osteomyelitis. The removal of bony protuberances for relief of pressure points and the superiority of myocutaneous flaps are discussed. The latter provides a cushion effect over the bone as well as a skin covering and is less likely to require further surgery as compared to other techniques of plastic surgery.

Included in this book is a short review of peripheral vascular disease, and arterial and venous stasis. There is, however, no discussion of dermatologic care of skin ulcers commonly associated with these problems.

There is a useful review of the different types of mattresses. The difficulties of prescribing the best type of wheelchair, proper cushions, their placement or shape, the proper height of arm rests, and correct positioning of the patient are all discussed as well. The laser Doppler technique for measuring microcirculation and plethysmographic techniques are described and are recommended for evaluating patients.

Pressure prevention clinics are now being encouraged in order to gather more material for this still difficult problem. It is hoped that the clinics will help to stimulate more interest in this problem. A collation of their statistical data will enable the improvement of existing techniques as well as increase the knowledge necessary to deal with this problem.

This book is valuable in that it reviews the latest techniques in surgical care and the utilization of microcirculatory measuring devices in dealing with chronic skin ulcers.

FREDERICK MEBEL, MD
Nassau County Medical Center
East Meadow, NY 11554

A CHILD DIES. A PORTRAIT OF FAMILY GRIEF

By Joan Hagan Arnold and Penelope Buschman Gemma. 160 pp, illustrated. Rockville, MD, Aspen Systems Corp, 1983. $21.50 (hardcover)

This volume is a simply stated and eloquent expression of the impact of children's death on parents. The two experienced and knowledgeable authors of the book, Joan Hagen Arnold and Penelope Buschman Gemma, both of whom are nurses, powerfully describe the anguish and suffering of parents. They acknowledge that words can never adequately communicate and convey the lasting devastation experienced by parents. Despite the difficulty in portraying this grief, the authors come as close as one can to conveying the enormity of the impact.

The book is comprised of nine chapters. The first three place the meaning of loss in context, including a family perspective and the mourning process. The second three chapters in the book cover the meaning to parents of the death of an infant, a young child, and an older child, respectively. Specific dynamics are influenced by each of these developmental stages. There are a number of pervasive themes regardless of age. The dead child is mourned forever. Efforts by others to deny the pain and emptiness seal off the parent's grief to be suffered alone and in isolation. Parental grief is without bounds, and the parents always remain parents of a dead child as well as of surviving children. Interwoven throughout the book are reproductions of paintings covering a span of history, each portraying the meaning of the death of children in the culture of the time. Also beautifully integrated into the substance of the book are passages from literature which enhance and bring to life the content of each chapter. No matter how often one might have read Frances Gunther's "A Word from Frances" in John Gunther's *Death Be Not Proud*, her description of grief continues to stir one's deepest emotions. Chapter seven offers a vivid portrayal of grief as it evolves in its many forms throughout parent's lives. The authors devote a chapter to sibling reaction and also offer compassionate attention to the impact on grandparents.

Finally, in chapters nine and ten, clear and explicit helping techniques are offered. There are no magical solutions and no ready ways of resolving emotional pain. There are instead ways of responding and caring which can lead in the direction of restoration and resolution. The helping professional's own response and use of self is well-addressed in this section. Professional caregivers often are in the unique role of continuously experiencing the grief of parents. Expectations are placed on them to be in tune with the emotions of the parents in order to be truly helpful. How to do this without developing distancing and self-protective measures becomes an ongoing dilemma and challenge. The authors offer life-enhancing techniques and values as an alternative to an avoidance of feelings and involvement.

This book is a must for all health care

professionals who face the death of children and the grief of parents. Unfortunately, the pain inherent in this topic may discourage some readers from seeking this greater understanding. It is a most helpful book for those who are struggling with how to deepen one's capacity to feel and to be truly responsive. The reward in allowing this universal and timeless sorrow to enter our full awareness is becoming stronger as more caring and sensitive professionals develop the skills of helping families cope with the death of a child.

ZELDA FOSTER, MSW
Brooklyn Veterans Administration
Medical Center
New York City 11209

TEXTBOOK OF NUCLEAR MEDICINE, VOLUME I: BASIC SCIENCE

Second edition. Edited by John Harbert, MD and Antonio Fernando Goncalves da Rocha, MD. 526 pp, illustrated. Philadelphia, Lea & Febiger, 1984. $80.00 (hardcover)

This volume constitutes the first of a two-volume set which is designed as a text for physicians, residents, and students of nuclear medicine. This first volume is devoted to the basic science aspects of nuclear medicine and therefore is also directed to laboratory scientists with an interest in nuclear counting and radioassay.

The current edition comes six years after the appearance of the first edition of this work by the same editors. With rapid advances in the technology of medicine it is not long before such works become outdated. The editors have managed to make this edition current by including new developments in instrumentation, radiopharmaceutical chemistry, and computers. New chapters on the complementary imaging methods, including magnetic resonance, digital radiography, computed tomography, and ultrasound have been added and provide a wider view of diagnostic imaging. These permit a better evaluation of radionuclide techniques and how they fit into the diagnostic algorithm.

The list of authors is comprised of recognized authorities in the field. The subjects are considered in depth and the range of material pertinent to the field make this a useful reference.

In addition to the usual information on the physics of nuclear medicine, instrumentation, counting statistics, and compartmental analysis, this volume also includes chapters on radiobiological effects, radiation safety, radiomicrobiology, labeled carbon breath analysis, and in vivo neutron activation analysis. There is also a chapter on perception parameters in medical images. The references are current.

The appendix contains a number of useful tables. These include a list of SI units, conversion factors, and physical constants. There are decay schemes of the most useful radionuclides, properties of the elements, a universal decay scheme, and a list of abbreviations and Greek letters.

Despite this being a multiauthored text the editors have imposed a uniformity of style and organization. The subjects are treated with the requisite depth and should provide a standard reference for workers in the nuclear medicine community. The print and tables are clear and the illustrations are surprisingly good in the non-glossy format. Typographical errors are rare.

This book must be compared to the recently published third edition of *Freeman and Johnson's Clinical Radionuclide Imaging*. The latter is concerned primarily with the nuclear radiology (imaging) aspects of nuclear medicine. Although the book does cover some aspects of physics, radiopharmaceutical production, and instrumentation, it does not provide in-depth coverage of non-imaging topics or of useful appendices included in the work under review by Harbert and Goncalves da Rocha. Residents training in nuclear medicine and physicians in departments that provide complete nuclear medicine services will find the Harbert-Goncalves da Rocha text to be of more value in this area.

The editors have achieved their objective of providing a comprehensive, up-to-date text which will be of value to all those working with radionuclides in medicine.

HAROLD L. ATKINS, MD
SUNY University Hospital at Stony Brook
Stony Brook, NY 11794

TEXTBOOK OF NUCLEAR MEDICINE, VOLUME II: CLINICAL APPLICATIONS

Second Edition. Edited by John Harbert, MD and Antonio Fernando Goncalves da Rocha, MD. 724 pp, illustrated. Philadelphia, Lea & Febiger, 1984. $95.00 (hardcover)

This companion volume to the *Textbook of Nuclear Medicine, Volume I: Basic Science* covers the clinical applications of radionuclide techniques. It must be compared directly to *Freeman and Johnson's Clinical Radionuclide Imaging*. Both cover similar material and are comparable in cost, although if one were interested only in the clinical applications one could purchase just volume II at considerably less cost.

The areas covered include the endocrine, central nervous, musculoskeletal, gastrointestinal, respiratory, cardiovascular, genitourinary, hematopoietic, and lymphatic systems, as well as tumors. All subjects are treated in depth and are current as of 1984. Included are discussions of positron and single photon emission tomography, monoclonal antibodies, and adrenal medullary imaging as an indication of the currency of the material.

This volume differs from the *Freeman and Johnson* text in that it covers several non-imaging topics such as ferrokinetics, vitamin B-12 absorption, radionuclide therapy of thyroid disease, and hematopoietic disorders, water and electrolytes, and red cell and plasma volume measurement. It goes into somewhat more detail concerning the eye, the peripheral vasculature, the salivary glands, and the hematopoietic system. There is also a useful section on guidelines for evaluating new tests. The sections have all been updated from the previous edition, the material is current, and references are recent.

The illustrations are adequate but are less effective than those in the *Freeman and Johnson* book. The text is written in an easy to read style that is consistent and the appendices contain useful information on absorbed dose from radiopharmaceuticals, common abbreviations, organ weights in children, a surface area nomogram, normal values for blood volume, and conversion factors for various units.

This volume is written by recognized authorities in the field and should serve as a useful reference for specialists and trainees in nuclear medicine. It should also be of value to other physicians who must often refer patients for radionuclide studies.

HAROLD L. ATKINS, MD
SUNY University Hospital at Stony Brook
Stony Brook, NY 11794

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Jona Allerhand, MD, Elmhurst. Died May 3, 1985; age 75. Dr Allerhand was a 1937 graduate of Deutsche Universität Medizinische Fakultät, Prague. He was a Fellow of the American College of Pathologists and a Diplomate of the American Board of Pathology, and a member of the American Society of Clinical Pathologists, the Medical Society of the County of Queens and the Medical Society of the State of New York.

Edgar Philip Bang, MD, Glen Cove. Died June 25, 1985; age 66. Dr Bang was a 1944 graduate of Johns Hopkins University School of Medicine. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Richard P. Bellaire, MD, Batavia. Died May 6, 1985; age 71. Dr Bellaire was a 1940 graduate of the Medical College of Virginia, Richmond. He was a Fellow of the American Academy of Family Practice, and a member of the Franklin County Medical Society and the Medical Society of the State of New York.

Hans Bergmann, MD, Forest Hills. Died April 18, 1985; age 90. He was a 1922 graduate of the Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin. Dr Bergmann was a member of the Queens County Medical Society and the Medical Society of the State of New York.

David Bloom, MD, New York. Died April 18, 1985; age 93. He was a 1919 alumnus of Universität Bern Medizinische Fakultät, Bern. Dr Bloom was a Diplomate of the American Board of Dermatology, Inc. His memberships included the Academy of Medicine, the New York State Dermatological Society, the American Academy of Dermatology, the Society for Investigative Dermatology, the New York County Medical Society and the Medical Society of the State of New York.

Alexander Borota, MD, New York. Died January 27, 1985; age 85. Dr Borota was a 1926 graduate of Orvosi Fakultas Pecsi Tudomanyegyetem, Pecs. He was a member of the American Academy of Compensation Medicine, Inc., the New York County Medical Society and the Medical Society of the State of New York.

Mark Carpati, MD, New York. Died June 2, 1985; age 85. Dr Carpati was a 1924 graduate of Universita Karlova Fakulta Lekarska, Praha. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Andrew James Charters, MD, Snyder. Died June 9, 1985; age 81. He was a 1932 graduate of the University of Buffalo School of Medicine. Dr Charters was a member of the Erie County Medical Society and the Medical Society of the State of New York.

Erwin Leslie Chusid, MD, New York. Died April 26, 1985; age 52. He was a 1958 alumnus of the University of Vermont College of Medicine. Dr Chusid was a Fellow of the American College of Physicians and the American College of Chest Physicians, and a Diplomate of the American Board of Internal Medicine. He was a member of the American Thoracic Society, the New York County Medical Society and the Medical Society of the State of New York.

Jay I. Evans, MD, Buffalo. Died April 29, 1985; age 89. Dr Evans was a 1929 graduate of the University of Buffalo School of Medicine. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

David Farber, MD, Great Neck. Died February 15, 1985; age 88. He was a 1919 graduate of New York University College of Medicine. Dr Farber was a Fellow of the American College of Surgeons, a Qualified Fellow of the International College of Surgeons, a Diplomate of the American Board of Surgery, and a member of the Kings County Medical Society and the Medical Society of the State of New York.

Albert Frohman, MD, Jamaica. Died April 25, 1985; age 88. He was a 1921 graduate of Friedrich Alexanders Universität Medizinische Fakultät, Erlangen. Dr Frohman was a Fellow of the American Academy of Family Physicians, and a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Percy Garson, MD, Peekskill. Died May 12, 1985; age 85. Dr Garson was a 1930 graduate of the St. Louis University School of Medicine. He was a member of the American Geriatrics Society, the Westchester County Medical Society and the Medical Society of the State of New York.

Fred Seymour Grunwald, MD, Clearwater, Florida. Died June 12, 1985; age 81. He was a 1932 graduate of New York Medical College. Dr Grunwald was a member of the Queens County Medical Society and the Medical Society of the State of New York.

Anthony James Guzzetta, MD, Rochester. Died June 24, 1985; age 93. Dr Guzzetta was a 1918 graduate of Tufts University School of Medicine, Boston. He was a Qualified Fellow of the International College of Surgeons and a member of the Academy of Medicine, the Monroe County Medical Society and the Medical Society of the State of New York.

Donald Davis Harman, MD, New York. Died April 4, 1985; age 80. Dr Harman was a 1932 alumnus of the State University of Iowa College of Medicine. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Joseph Charles Hart, MD, Poughkeepsie. Died June 30, 1985; age 70. Dr Hart was a 1941 graduate of the Georgetown University School of Medicine. He was a member of the Dutchess County Medical Society and the Medical Society of the State of New York.

Bernard John Hartnett, MD, Auburn. Died April 25, 1985; age 65. He was a 1944 graduate of the State University of New York Upstate Medical Center. Dr Hartnett was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. He was a member of the New York State Surgical Society, the Medical Society of the County of Cayuga and the Medical Society of the State of New York.

Alfred J. Hess, MD, Sarasota, Florida. Died October 12, 1984; age 90. Dr Hess was a 1920 graduate of Julius-Maximilians-Universität Medizinische Fakultät, Wurzburg. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Harry Kalenscher, MD, Bayside. Died June 19, 1985; age 82. Dr Kalenscher was a 1930 graduate of Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin. He was a member of the Queens County Medical Society and the Medical Society of the State of New York.

John E. Kuhlke, MD, Smithtown. Died May 16, 1985; age 68. Dr Kuhlke was a 1943 alumnus of the State University of New York Downstate Medical Center. His memberships included the Academy of Medicine, the American Academy of Family Physicians, the Suffolk County Medical Society and the Medical Society of the State of New York.

Vincent de Paul Larkin, MD, Brooklyn. Died May 21, 1985; age 71. He was a 1939 alumnus of Cornell University Medical College. Dr Larkin was a Diplomate of the American Board of Pediatrics, and a member of the Academy of Medicine, the American Academy of Pediatrics, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Vincent J. Panvini, MD, East Elmhurst. Died May 2, 1985; age 72. He was a 1941 graduate of Middlesex University School of Medicine. Dr Panvini was a Qualified Fellow of the International College of Surgeons, and a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Helene Papanek, MD, New York. Died May 21, 1985; age 84. She was a 1925 graduate of Medizinische Fakultät Universität Wien. Dr Papanek was a member of the American Psychiatric Association, the Association for Advancement of Psychotherapy, the New York County Medical Society and the Medical Society of the State of New York.

Robert J. Powalski, MD, Buffalo. Died May 15, 1985; age 57. He was a 1954 alumnus of the University of Buffalo School of Medicine. Dr Powalski was a Fellow of the American College of Obstetricians and Gynecologists and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Pan American Medical Association, the Medical Society of the County of Erie and the Medical Society of the State of New York.

Anna Earl Purdy, MD, Atlanta, Georgia. Died April 25, 1985; age 83. Dr Purdy was a 1927 alumna of Columbia University College of Physicians and Surgeons. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Paul Rapoport, MD, Brooklyn. Died February 2, 1985; age 73. Dr Rapoport was a 1941 graduate of the University of Kansas School of Medicine. He was a member of the New York State Society of Internal Medicine, the American Society of Internal Medicine, the Kings County Medical Society and the Medical Society of the State of New York.

Irvine M. Ratner, MD, South Salem. Died June 23, 1985; age 70. He was a 1947 Licentiate of the Royal College of Physicians of Edinburgh. Dr Ratner was a Fellow of the American College of Pathologists and Diplomate of the American Board of Pathology. His memberships included the Academy of Medicine, the New York State Society of Pathologists, the American Society of Clinical Pathologists, the Bronx County Medical Society and the Medical Society of the State of New York.

George Rosenow, MD, New York. Died January 9, 1985; age 99. He was a 1911 graduate of Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin. Dr Rosenow was a Fellow of the American College of Physicians, and a member of the Academy of Medicine, the International Society of Hematology, the New York County Medical Society and the Medical Society of the State of New York.

Nelson Hopson Rust, MD, Scotia. Died April 26, 1985; age 82. Dr Rust was a 1927 alumnus of Albany Medical College of Union University. He was a Fellow of the American Academy of Family Physicians, and a member of the Medical Society of the County of Schenectady and the Medical Society of the State of New York.

Henry Lupton Rutzler, MD, New York. Died July 8, 1984; age 76. He was a 1935 graduate of Columbia University College of Physicians and Surgeons. Dr Rutzler was a member of the Association for Advancement of Psychotherapy, the New York County Medical Society and the Medical Society of the State of New York.

Hans Schein, MD, West Palm Beach, Florida. Died May 13, 1985; age 76. Dr Schein was a 1935 alumnus of Schlesische-Friedrich-Wilhelms-Universität Medizinische Fakultät, Breslau. He was a member of the American School Health Association, the Pan American Medical Association, the American Geriatrics Society, the Medical Society of the County of Saratoga and the Medical Society of the State of New York.

Alfredo Schiano, MD, Rye. Died April 14, 1984; age 57. He was a 1950 alumnus of Università di Napoli Facoltà di Medicina e Chirurgia, Napoli. Dr Schiano was a Diplomate of the American Board of Radiology, and a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Joseph F. Sebesta, Jr., MD, Syracuse. Died June 24, 1985; age 54. Dr Sebesta was a 1960 graduate of the St. Louis University School of Medicine. He was a member of the Onondaga Medical Society and the Medical Society of the State of New York.

Robert E. Shea, MD, Buffalo. Died June 9, 1985; age 73. Dr Shea was a 1952 graduate of the University of Buffalo School of Medicine. He was a member of the Erie County Medical Society and the Medical Society of the State of New York.

James Stephenson, MD, New York. Died March 27, 1985; age 66. He was a 1951 graduate of Cornell University Medical College. Dr Stephenson was a member of the New York County Medical Society and the Medical Society of the State of New York.

Paul Sherlock, MD, New York. Died May 6, 1985; age 57. He was a 1954 graduate of Cornell University Medical College. Dr Sherlock was a Fellow of the American College of Physicians and the American College of Gastroenterology, and a Diplomate of the American Board of Internal Medicine. His memberships included the New York Gastroenterological Society, the Academy of Medicine, the American Gastro-Enterological Association, the American Society of Internal Medicine, the New York County Medical Society and the Medical Society of the State of New York.

Joseph Robert Sills, MD, Bronx. Died September 7, 1983; age 79. He was a 1930 graduate of New York University College of Medicine. Dr Sills was a Fellow of the American Academy of Family Physicians, and a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Otto Sternberg, MD, New York. Died March 31, 1985; age 86. Dr Sternberg was a 1924 graduate of Medizinische Fakultät Universität Wien. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Maurice Vaisberg, MD, Long Beach. Died April 19, 1985; age 80. Dr Vaisberg was a 1930 graduate of the State University of New York Downstate Medical Center. He was a member of the American Psychiatric Association, the Nassau County Medical Society and the Medical Society of the State of New York.

Ralph Reuben Zimet, MD, Scarsdale. Died April 26, 1985; age 77. Dr Zimet was a 1933 graduate of the State University of New York Downstate Medical Center. He was a member of the Westchester County Medical Society and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089–1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), an abbreviated title of no greater than 40 spaces for use as a running foot, and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear on the text. When there are six or fewer authors, all should be listed; where there are seven or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. McGuigan JE. Peptic ulcer. In: Petersdorf RG, Adams RD, Braunwald E, Isselbacher KJ, Martin JB, Wilson JD, eds. *Harrison's Principles of Internal Medicine.* New York, McGraw-Hill, 1983, pp 1697–1712.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Alan Blum, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

Essential reading for every physician

NEW YORK STATE
JOURNAL OF MEDICINE
JULY 1985 • Volume 85, Number 7
Table of Contents, page 279
THE WORLD CIGARETTE PANDEMIC
Part II
PRIMUM NON NOCERE?

The July issue of the **New York State Journal of Medicine** is the most comprehensive and disturbing ever published by a medical journal on the subject of smoking.

SEND A COPY TO COLLEAGUES AND MEDICAL STUDENTS in the US and abroad who might not normally receive the Journal. Or order an extra copy for your patients or yourself.

NOTE: the extra press run of The World Cigarette Pandemic Part 1 (December 1983 issue) sold out quickly. The issue has been updated and republished as *The Cigarette Underworld* ($9.95 plus $1.50 postage). Copies may be ordered from Lyle Stuart, Inc., 120 Enterprise Avenue, Secaucus, NJ 07094.

An extra press run has enabled the NEW YORK STATE JOURNAL OF MEDICINE to offer single or bulk copies of its recent issue on the world cigarette pandemic. As the official publication of the Medical Society of the State of New York since 1900, the *Journal* is one of the foremost independent peer-reviewed general medical journals in the country. Providing timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news, the *Journal* makes important reading—and a welcome gift—to physicians and students in the United States or abroad. A free copy of the July issue will be included with each subscription.

Quantity	Per Copy*
1	$7.50
2-9	$7.00
10-24	$6.50
25-49	$6.00
50-99	$5.50
100-149	$5.00
150-500	$4.00

ORDER FORM

Name______________________________

Address______________________________

City/State/Zip__________________________

Please send________copies of July 1985 issue of the Journal.
Enter my subscription to the Journal. ☐ $25.00 U.S. & Canada; ☐ $30.00 Foreign

Total amount enclosed $__________ ***Note:** Domestic postage included. Please add New York state & local sales tax where applicable, or sales tax exemption certificate. Foreign postage additional.

Mail to: Circulation Department, **New York State Journal of Medicine**
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

1986 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
New York Hilton, New York City

Maintaining the Quality of
Health Care into the 21st Century

FRIDAY, MARCH 7

10:00 a.m. - 1:00 p.m.	Cardiovascular Diseases
	Gastroenterology/Alcoholism
	Obstetrics/Gynecology
	Neurosurgery
	Physical Medicine & Rehabilitation
2:00 p.m. - 5:00 p.m.	Neurology
	Pediatrics/Alcoholism
	Chest Diseases
	Medical Legal & Workers Comp./Occupational Medicine
	Medical Liability Mutual Insurance Co.
	Realistic Uses of Computers in Medical Practice

SATURDAY, MARCH 8

10:00 a.m. - 1:00 p.m.	Ophthalmology (All Day)
	Internal Medicine (All Day)
	Hand Surgery/Anesthesiology (All Day)
	Urology/Dermatology
	Psychiatry (All Day)
	Realistic Uses of Computers in Medical Practice
2:00 p.m. - 5:00 p.m.	Ophthalmology
	Internal Medicine
	Otolaryngology
	Hand Surgery/Anesthesiology
	Psychiatry

SUNDAY, MARCH 9

9:00 a.m. - 1:00 p.m.	General Surgery/Colon & Rectal Surgery
10:00.a.m. - 1:00 p.m.	Allergy & Immunology
	Women's Medical Society of N.Y./Family Practice/Emergency Medicine
	Pathology

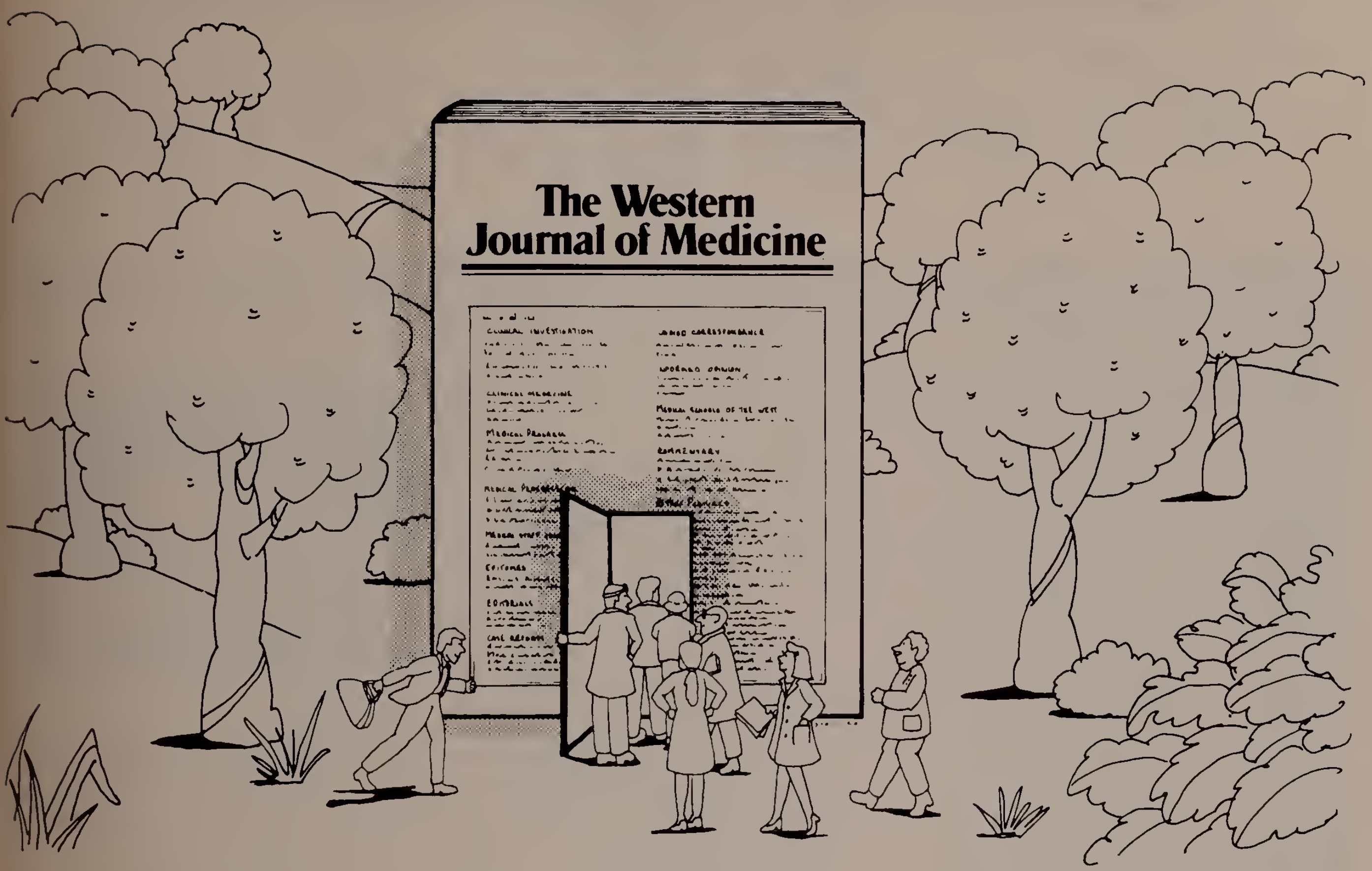
□

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis.

□

Technical Exhibits

Inside Information


The Western Journal of Medicine

Consider a few features of **The Western Journal of Medicine:**

- **Epitomes of Progress**—Each month this section highlights the major advances in a different specialty with 15 or 20 brief epitomes by experts in the field.
- **Clinical Investigation**—Five major clinical research societies coordinate important new research findings, emphasizing clinical value.
- **Socioeconomics**—WJM is often the first to predict, examine and evaluate socioeconomic changes, trends.
- **Yearly Special Issue**—Each December's issue is devoted to a topic vital to physicians: clinical nutrition (1979), cost of care (1980), geriatrics (1981), occupational/environmental medicine (1982) cross-cultural medicine (1983), personal health maintenance (1984), high-tech medicine (1985).

NYJ

The Western Journal of Medicine

Please mail this coupon with
enclosed payment to:

Circulation Department
44 Gough Street, San Francisco, California 94103

Subscriptions, 12 Issues Per Year
U.S. and Canada $20
All other countries $30

Enclosed is $________ to cover ________ subscriptions.

Subscription renewal ☐ Regular subscription ☐ Foreign subscription ☐

TO: ________________________________

____
Street

____
City State Zip

____

Specialty ________________________________

Gift Card to read "From ________________________________"

BACK COPIES NEW YORK STATE JOURNAL OF MEDICINE

For a limited time, back copies may be purchased at the following rates plus postage.

1971-1980 . . . $2.00
1981-1983 . . . 3.00

Exception: for the special issue of December 1983 which costs $5.00.

Send your order to:
Circulation Dept., New York State Journal of Medicine, 420 Lakeville Road, P.O. Box 5404, Lake Success, NY 11042.

Inquiries: (516) 488-6100, Circulation Dept.

MISCELLANEOUS—CONT'D

DISCIPLINARY LAW; SCIENCE LAW. BIRNBAUM & MANAKER, P.C., law firm actively engaged in science law and scientific litigation with emphasis on: defense and prosecution of medical malpractice actions; product liability suits involving medical and hospital devices and equipment; representation of physicians and other scientists in patent royalty negotiations and marketing of inventions; representation of physicians and other health care providers before state agencies, professional societies, review boards; litigation in anti-competition disputes concerning privileges, appointments and use of diagnostic facilities. Partners have extensive scientific, medical, and engineering law backgrounds. Senior partner, Irwin Birnbaum, has held law school adjunct teaching appointments, has lectured at medical schools and engineering colleges, and served as Chair of the Roscoe Pound Commission on Professional Responsibility. Partners' CV's available on request. BIRNBAUM & MANAKER, P.C., MONY Tower II, Syracuse, New York 13202, (315) 422-0246, (800) 358-8568.

MISCELLANEOUS—CONT'D

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuariat certification and employee statements of participation—partnership agreements and professional incorporation—No insurance required—references upon request—Jack Wachstock, PC, 3000 Marcus Ave., Lake Success, N.Y. 11530 (516) 437-4848.

MEDICAL EDITOR/WRITER: Seeks freelance assignments. Experienced in clinical/socio-economic subjects, MD manuscripts preparation, article research, presentations, graphics, copyediting, proofreading. Call (212) 861-7280.

ARCHITECTURAL DESIGN AND CONSTRUCTION: Designers, builders of custom professional offices. Consulting services available. Whether you're opening a new office, relocating, remodeling or expanding an existing office in New York City area and adjacent New Jersey counties. Donald Young, Inc. Tel: (718) 948-5191.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate, with no points, fees or charges of any kind. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

DOCTOR, YOU CAN'T BEAT the quality or the price! Holter Monitor Scanning Service. Physician owned, trained and supervised. No contracts to sign. We can arrange for lease/purchase of Holter equipment. New dual channel recorders with test cables $1,550. Now using UP Service for faster turn around time. DCG Interpretation (313) 879-8860.

ST. LUKE'S-ROOSEVELT HOSPITAL CENTER: CONTINUING EDUCATION INSTITUTE. Series of weekly seminars in general medicine for the general medical practitioner. Approved for Category 1 credit and submitted for AAFP approval. Total hours approximately 48–50. Beginning Thursday, September 26, 1985. Complimentary parking available. For information contact: Dr. Kenneth Weinstein, 262 Central Park West, New York, NY 10025. (212) 799-2627.

MISCELLANEOUS—CONT'D

PROFESSIONAL MISCONDUCT ATTORNEYS. WILLIAM L. WOOD, JR., formerly Executive Director of the New York State Office of Professional Discipline and ANTHONY Z. SCHER, formerly Director of Prosecutions, announce the opening of their law office, WOOD & SCHER. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct proceedings, malpractice, licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practitioners. WOOD & SCHER, Attorneys at Law, One Chase Road, Scarsdale, New York 10583, Telephone 914-723-3500.

Safe Deposit.

Most people look for a safe place to deposit their money. A place where they can keep an eye on it.

Well, buying U.S. Savings Bonds is a safe and easy way to a safe deposit. Because Bonds can be replaced if lost, stolen, or destroyed.

Plus, Bonds offer guarantees: a guaranteed way to save, through the Payroll Savings Plan; a guaranteed interest rate; and guaranteed tax benefits. All guaranteed by America.

Add it all up. U.S. Savings Bonds are the safest deposit.

Keep your eye on them *and* watch them grow.

Take stock in America.

UNITED STATES SAVINGS BONDS

When you put part of your savings into U.S. Savings Bonds you're helping to build a brighter future for your country and for yourself.

A public service of this publication and The Advertising Council.

Added Control for America's Coughs

Robitussin

Robitussin and codeine
Each 5 ml (1 teaspoonful) contains:

Guaifenesin, USP	100 mg
Codeine Phosphate, USP	10 mg

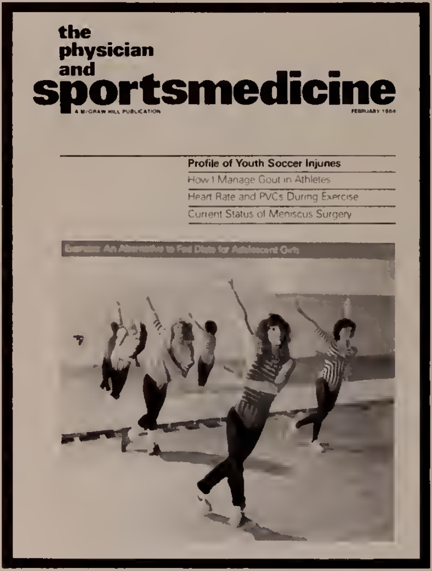
(Warning May be habit forming)
Alcohol 3.5 percent
In a palatable, aromatic syrup

A-C®Ⓥ

The expectorant action of guaifenesin plus the Advantage of Codeine!

A·H·ROBINS
Pharmaceutical Division, Richmond, Virginia 23261-6609
©A.H. Robins Company 1985

OVER 66,000 FAMILY PHYSICIANS READ THIS JOURNAL



the physician and sportsmedicine
A McGRAW HILL PUBLICATION

Profile of Youth Soccer Injuries
How I Manage Gout in Athletes
Heart Rate and PVCs During Exercise
Current Status of Meniscus Surgery

Practical information on the medical aspects of fitness and exercise.

Tennis elbow: Joint resolution by conservative treatment.

Hypertrophic cardiomyopathy and the athlete.

Effects of sunscreen use during exercise in the heat.

Overuse injuries to the knee in runners.

How I manage ingrown toenails.

McGraw Hill

Medical Society of the State of New York
ANNUAL CME ASSEMBLY
MARCH 7-9, 1986
The New York Hilton, New York City
Combined With The Annual Meeting of
The House of Delegates
MARCH 6-9

WEEKLY SEMINARS

Most major ski areas, Club Med, Disney World, Cruising aboard Sailboats in the Virgin Islands or a Mississippi Paddlewheeler.

Topic: Medical/Legal Issues
Accredited *Fee $175*

Current Concept Seminars, Inc. *(since 1980)*
3301 Johnson St., Hollywood, FL 33021
(800) 428-6069

Buy U.S. Savings Bonds

This Publication is available in Microform.

University Microfilms International

Please send additional information
for ______________
(name of publication)
Name______________
Institution______________
Street______________
City______________
State__________ Zip__________
300 North Zeeb Road, Dept. P.R., Ann Arbor, Mi 48106

Angina comes in many forms...

So does SORBITRATE®

(ISOSORBIDE DINITRATE)

Unsurpassed flexibility in nitrate therapy.

© 1985 ICI AMERICAS INC.

See following page for brief summary of prescribing information.

SORBITRATE

(ISOSORBIDE DINITRATE)

Please consult full prescribing information before use. A summary follows:

INDICATIONS AND USAGE: SORBITRATE (isosorbide dinitrate) is indicated for the treatment and prevention of angina pectoris All dosage forms of isosorbide dinitrate may be used prophylactically to decrease frequency and severity of anginal attacks and can be expected to decrease the need for sublingual nitroglycerin.

The sublingual and chewable forms of the drug are indicated for acute prophylaxis of angina pectoris when taken a few minutes before situations likely to provoke anginal attacks Because of a slower onset of effect, the oral forms of isosorbide dinitrate are not indicated for acute prophylaxis

CONTRAINDICATIONS: SORBITRATE is contraindicated in patients who have shown purported hypersensitivity or idiosyncrasy to it or other nitrates or nitrites. Epinephrine and related compounds are ineffective in reversing the severe hypotensive events associated with overdose and are contraindicated in this situation.

WARNINGS: The benefits of SORBITRATE during the early days of an acute myocardial infarction have not been established. If one elects to use organic nitrates in early infarction, hemodynamic monitoring and frequent clinical assessment should be used because of the potential deleterious effects of hypotension.

PRECAUTIONS: General: Severe hypotensive response, particularly with upright posture, may occur with even small doses of SORBITRATE The drug should therefore be used with caution in subjects who may have blood volume depletion from diuretic therapy or in subjects who have low systolic blood pressure (eg, below 90 mmHg). Paradoxical bradycardia and increased angina pectoris may accompany nitrate-induced hypotension. Nitrate therapy may aggravate the angina caused by hypertrophic cardiomyopathy

Marked symptomatic, orthostatic hypotension has been reported when calcium channel blockers and organic nitrates were used in combination. Dose adjustment of either class of agents may be necessary

Tolerance to this drug and cross-tolerance to other nitrates and nitrites may occur Tolerance to the vascular and antianginal effects of isosorbide dinitrate or nitroglycerin has been demonstrated in clinical trials, experience through occupational exposure, and in isolated tissue experiments in the laboratory The importance of tolerance to the appropriate use of isosorbide dinitrate in the management of patients with angina pectoris has not been determined However, one clinical trial using treadmill exercise tolerance (as an end point) found an 8-hour duration of action of oral isosorbide dinitrate following the first dose (after a 2-week placebo washout) and only a 2-hour duration of effect of the same dose after 1 week of repetitive dosing at conventional dosing intervals On the other hand, several trials have been able to differentiate isosorbide dinitrate from placebo after 4 weeks of therapy and, in open trials, an effect seems detectable for as long as several months.

Tolerance clearly occurs in industrial workers continuously exposed to nitroglycerin. Moreover, physical dependence also occurs since chest pain, acute myocardial infarction, and even sudden death have occurred during temporary withdrawal of nitroglycerin from the workers In clinical trials in angina patients, there are reports of anginal attacks being more easily provoked and of rebound in the hemodynamic effects soon after nitrate withdrawal. The relative importance of these observations to the routine, clinical use of isosorbide dinitrate is not known However, it seems prudent to gradually withdraw patients from isosorbide dinitrate when the therapy is being terminated, rather than stopping the drug abruptly.

Information for Patients: Headache may occur during initial therapy with SORBITRATE Headache is usually relieved by the use of standard headache remedies or by lowering the dose and tends to disappear after the first week or two of use.

Drug Interactions: Alcohol may enhance any marked sensitivity to the hypotensive effect of nitrates

Isosorbide dinitrate acts directly on vascular smooth muscle; therefore, any other agent that depends on vascular smooth muscle as the final common path can be expected to have decreased or increased effect depending on the agent.

Carcinogenesis, Mutagenesis, Impairment of Fertility: No long-term studies in animals have been performed to evaluate the carcinogenic potential of this drug A modified two-litter reproduction study in rats fed isosorbide dinitrate at 25 or 100 mg/kg/day did not reveal any effects on fertility or gestation or any remarkable gross pathology in any parent or offspring fed isosorbide dinitrate as compared with rats fed a basal-controlled diet

Pregnancy Category C: Isosorbide dinitrate has been shown to cause a dose-related increase in embryotoxicity (increase in mummified pups) in rabbits at oral doses 35 and 150 times the maximum recommended human daily dose There are no adequate and well-controlled studies in pregnant women SORBITRATE should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: It is not known whether this drug is excreted in human milk Because many drugs are excreted in human milk, caution should be exercised when SORBITRATE is administered to a nursing woman

Pediatric Use: The safety and effectiveness of SORBITRATE in children has not been established

ADVERSE REACTIONS: Adverse reactions, particularly headache and hypotension, are dose-related In clinical trials at various doses, the following have been observed

Headache is the most common (reported incidence varies widely, apparently being dose-related, with an average occurrence of about 25%) adverse reaction and may be severe and persistent Cutaneous vasodilation with flushing may occur Transient episodes of dizziness and weakness, as well as other signs of cerebral ischemia associated with postural hypotension, may occasionally develop (the incidence of reported symptomatic hypotension ranges from 2% to 36%) An occasional individual will exhibit marked sensitivity to the hypotensive effects of nitrates and severe responses (nausea, vomiting, weakness, restlessness, pallor, perspiration, and collapse) may occur even with the usual therapeutic dose Drug rash and/or exfoliative dermatitis may occasionally occur. Nausea and vomiting appear to be uncommon Case reports of clinically significant methemoglobinemia are rare at conventional doses of organic nitrates The formation of methemoglobin is dose-related and, in the case of genetic abnormalities of hemoglobin that favor methemoglobin formation, even conventional doses of organic nitrate could produce harmful concentrations of methemoglobin.

DOSAGE AND ADMINISTRATION: For the treatment of angina pectoris, the usual starting dose for sublingual SORBITRATE is 2 5 to 5 mg, for chewable tablets, 5 mg, for oral (swallowed) tablets, 5 to 20 mg, and for controlled-release forms, 40 mg

SORBITRATE should be titrated upward until angina is relieved or side effects limit the dose In ambulatory patients, the magnitude of the incremental dose increase should be guided by measurements of standing blood pressure.

The initial dosage of sublingual or chewable SORBITRATE for prophylactic therapy in angina pectoris patients is generally 5 or 10 mg every 2 to 3 hours. Adequate controlled clinical studies demonstrating the effectiveness of chronic maintenance therapy with these dosage forms have not been reported

SORBITRATE in oral doses of 10 to 40 mg given every 6 hours or in oral controlled-release doses of 40 to 80 mg given every 8 to 12 hours is generally recommended The extent to which development of tolerance should modify the dosage program has not been defined *The oral controlled-release forms of isosorbide dinitrate should not be chewed*

DOSAGE FORMS AVAILABLE: Sublingual Tablets (2 5, 5, 10 mg), Chewable Tablets (5, 10 mg); Oral Tablets (5, 10, 20, 30, 40 mg); Sustained Action Tablets (40 mg)

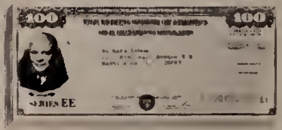
STUART PHARMACEUTICALS
Division of ICI Americas Inc.
Wilmington, DE 19897

STR-2282

SOME OF THE GREATEST THINGS IN AMERICA NEVER CHANGE.

Reprinted from *The Saturday Evening Post* 1943
The Curtis Publishing Co.

SOME DO.



Paying Over 9%.

In 1621 a small band of colonists gave thanks for their first harvest. Today, Thanksgiving is still a time to give thanks for all that we have.

Some things never change.

One great American tradition *has* changed—U.S. Savings Bonds. Today's Savings Bonds pay higher variable interest rates like money market accounts. Currently, Bonds are paying over 9%.

Just hold Savings Bonds for five years and you get the new variable interest rates. Plus, you get a guaranteed return. This means you can earn a lot more, but never less than 7½%!

You can purchase Bonds at almost any financial institution, or easier yet, through the Payroll Savings Plan where you work.

Buy U.S. Savings Bonds. Like Thanksgiving, they're well worth remembering. *To find out the current interest rate and more information, call toll-free 1-800-US-BONDS.*

U.S. SAVINGS BONDS
Paying Better Than Ever

Variable rates apply to Bonds purchased on and after 11-1-82 and held at least five years. Bonds purchased before 11-1-82 earn variable rates when held beyond 10-31-87. Bonds held less than five years earn lower interest

A public service of this publication

FOR PHYSICIANS AND SURGEONS WITH A PART-TIME EXPOSURE ONLY

Now offering Medical Malpractice Insurance on an ***occurrence basis*** at drastically reduced rates thru Frontier Insurance Co., Monticello, NY.

With the tail on claims made policies, you might lose your shirt.

Call or write for further information and application.

MEDICAL
MQ
QUADRANGLE

Medical Quadrangle, Inc.
P. O. Box 456
318 Bedford Road
Bedford Hills, New York 10507
914 666-0170
212 829-3654
"Specializing in Medical Liability Insurance"

Index to Advertisers

Ayerst Laboratories 2A,3A,4A,28A,29A,30A
Classified Advertising 7A,17A,18A,22A,24A,27A
Future Information Systems Computers .... 3rd Cover
HealthWays 11A
Knoll Pharmaceuticals 12A,13A,14A
Eli Lilly and Company .... 2nd Cover
Marion Laboratories 9A,10A
McGraw Hill Publications 24A
Medical Quadrangle 27A
A. H. Robins Company 8A,23A
Sandoz Company 15A,16A
Seabrook Island 17A
Stough Dermatology & Cutaneous Surgery Clinic 18A
Stuart Pharmaceuticals 25A,26A
Upjohn Company .... 4th Cover
Wyeth Laboratories 5A,6A

Need A Temporary Physician?
During: vacations, CME's, illness, recruiting.
Want Free Time While You Practice Medicine?
Join CompHealth's Locum Tenens Physician Group.
For further information, call 412-741-3310.

CompHealth A Physician Group
WILSON ROSS, Regional Administrator
114 Centennial Ave., Sewickley, PA 15143

NATIONAL EMERGENCY SERVICES, INC. (N.E.S.)

Directorships, full- and part-time emergency department opportunities are available throughout New York State and the country for primary care physicians with ER experience. Enjoy a variety of practice settings, competitive remuneration, paid malpractice insurance, and flexible scheduling. For information and application materials contact the recruitment department at (516) 365-4010, (Outside N.Y. State 1-800-645-4848) or send C.V. to N.E.S., One Hollow Lane, Suite 304, Lake Success, N.Y. 11042.

Right from the start in hypertension…

Once-daily INDERAL LA (propranolol HCl) for smooth blood pressure control without the potassium problems of diuretics

Once-daily INDERAL LA (propranolol HCl) avoids the risk of diuretic-induced ECG abnormalities due to hypokalemia.[1,2] In addition, INDERAL LA preserves potassium balance without additive agents or supplements while providing simple, well-tolerated therapy with broad cardiovascular benefits.

Once-daily INDERAL LA for the cardiovascular benefits of the world's leading beta blocker

Simply start with 80 mg once daily. Dosage may be increased to 120 mg to 160 mg once daily as needed to achieve additional control.

Like conventional INDERAL tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.

For beta-1/beta-2 blockade

Once-daily

INDERAL LA

(PROPRANOLOL HCl) [illegible]

80 mg 120 mg 160 mg

The appearance of these capsules is a registered trademark of Ayerst Laboratories.

Please see brief summary of prescribing information on the next page for further details.

For beta-1/beta-2 blockade Once-daily INDERAL® LA (PROPRANOLOL HCl) LONG ACTING CAPSULES

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period

The mechanism of the antihypertensive effect of INDERAL has not been established Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain. Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use Effects on plasma volume appear to be minor and somewhat variable INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential The significance of the membrane action in the treatment of arrhythmias is uncertain

The mechanism of the antimigraine effect of propranolol has not been established Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of symoathetic activity on conduction Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm

Propranolol is not significantly dialyzable

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

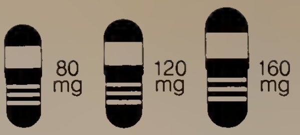
WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)— PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures



The appearance of these capsules
is a registered trademark
of Ayerst Laboratories

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g., dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes In these patients, it may be more difficult to adjust the dosage of insulin

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General* Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure

Clinical Laboratory Tests Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy Pregnancy Category C INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose

There are no adequate and well-controlled studies in pregnant women INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman

Pediatric Use Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular bradycardia, congestive heart failure, intensification of AV block hypotension; paresthesia of hands; thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type

Central Nervous System lightheadedness; mental depression manifested by insomnia lassitude, weakness, fatigue; reversible mental depression progressing to catatonia visua disturbances, hallucinations, an acute reversible syndrome characterized by disorientation fo time and place, short-term memory loss, emotional lability, slightly clouded sensorium anc decreased performance on neuropsychometrics

Gastrointestinal nausea, vomiting epigastric distress, abdominal cramping, diarrhea constipation, mesenteric arterial thrombosis, ischemic colitis

Allergic pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory bronchospasm

Hematologic agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol have not been associated with propranolol

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute fo INDERAL. INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION—*Dosage must be individualized* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic The dosage may be increased to 120 mg once daily or higher until adequate blood pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris the value anc safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis If a satisfactory response is no obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period o several weeks

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are toc limited to permit adequate directions for use

REFERENCES

1. Holland OB, Nixon JV, Kuhnert L: Diuretic-induced ventricular ectopic activity. *Am J Med* 1981,70:762-768 **2.** Holme I, Helgeland A, Hjermann I, et al. Treatment of mild hypertension with diuretics. The importance of ECG abnormalities in the Oslo study and in MRFIT. *JAMA* 1984,251.1298-1299

Ayerst® AYERST LABORATORIES
New York, N Y. 10017

9411/1184

Copyright © 1984 AYERST LABORATORIES
Division of AMERICAN HOME PRODUCTS CORPORATION

THE PHYSICIAN'S ATVANTAGE

FUTURE transforms the IBM PC AT into a nine-user system to manage your practice.

Now FUTURE Information Systems offers the ATvantage that lets you practice medicine instead of accounting: Computone's powerful combination of Medical Office Management System software and AT expansion hardware. The total system that provides everything you need to automate your office from one of the country's fastest-growing computer dealers.

You can build a single IBM PC AT into a nine-user multi-tasking system with the AT ATvantage board, using any IBM PC, PCjr or inexpensive stand-alone terminals you may already own.

You can reduce paperwork with easy-to-use Medical Office Management System software, designed to let your staff enter or access information and simultaneously perform up to five related functions. Many accounting and management control operations are automatically updated with patient entry and posting.

And you can do it all with FUTURE beside you every step of the way, providing in-house or on-site training, complete hardware and software maintenance and continuous support.

Get the Physician's ATvantage working for you. Contact FUTURE Information Systems today.

Features of the Physician's ATvantage:

☐ Instant access to patient balance, insurance submission and insurance payment information
☐ Open-item patient accounting
☐ Patient statements and "Super-bill"
☐ Third-party insurance processing
☐ Patient/guarantor relationships
☐ Insurance rebilling or write-off
☐ Cash flow control
☐ Patient history
☐ Problem accounts
☐ Accounts receivable reductions of 20-50%
☐ Patient referrals
☐ Appointment scheduling
☐ Automatic audit trails
☐ Single doctor office, or several
☐ Multi-office practices

FUTURE℠
INFORMATION SYSTEMS

A Computone company

MOVING YOU UP IN THE WORLD℠

Business Market Sales Centers:
Downtown Manhattan: 95 Trinity Place • (212) 732-3905
Albany: 1492 Central Ave. • (518) 456-8162

Midtown Manhattan: 52 East 53rd St • (212) 751-7888
Schenectady: Woodlawn Plaza, 1594 State St. • (518) 382-1182
Glens Falls: 28 Ridge St. • (518) 798-1446
Kingston: King's Mall, Rt 9-W • (914) 336-4155

IBM PC, IBM PCjr and IBM PC AT are trademarks of International Business Machines Corp ATvantage is a trademark of Computone Systems Inc

New Motrin® 800 TABLETS mg

ibuprofen

Extra strength

Convenience

Economy

Upjohn

The Upjohn Company
Kalamazoo, Michigan 49001 USA

© 1985 The Upjohn Company

J-5491 June 1985

STACKS

NEW YORK STATE JOURNAL OF MEDICINE

NOVEMBER 1985 • Volume 85, Number 11

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA

DEC 12 1985

M. D. S.

Contents

HEALTH CARE IN THE 21st CENTURY

This section consists of papers presented at a symposium entitled, ***U.S. Health Care Systems into the 21st Century, Demand for Health Care, Changing Incidence of Injuries, Illnesses and Diseases***

sponsored by the Institute for Economic Analysis, the Health Policy and Management Program in the Graduate School of Public Administration, New York University, and the New York County Health Services Review Organization.

Reflections on health care at the coming turn of the century 619
LOWELL E. BELLIN, MD

Future patterns of chronic disease incidence, disability, and mortality among the elderly: Implications for the demand for acute and long-term health care 623
KENNETH G. MANTON, PhD

Implications of new medical technology 634
ROGER C. HERDMAN, MD

The roles of new medical technology and health care delivery systems in shaping medicine into the 21st century 637
PASCAL JAMES IMPERATO, MD

Implications of changes in payment practices for health care: The insurer's perspective 641
MICHAEL R. McGARVEY, MD
SHARON B. KING, JD, MPA

Health care payment reform: projections for the next 15 years 644
GERARD F. ANDERSON, PhD
CATHERINE R. RAPOZA

THE CPC

A 74-year-old man with a lung mass, arthritis, and difficulty walking 647

CASE REPORTS

Pregnancy obtained by in vitro fertilization in an ambulatory-care surgical facility 654
ANDREW Y. SILVERMAN, MD, PhD, STEPHEN RENZIN, MD, OTTO RATZ, MD, FLORENCE BACK, STEVEN G. KAALI, MD, ROBERT LANDESMAN, MD

Ultrasound diagnosis of interstitial pregnancy 655
DEBORAH J. COADY, MD, JON R. SNYDER, MD, STEVEN R. GOLDSTEIN, MD, BALA R. SUBRAMANYAN, MD

Type I herpes simplex esophagitis with candidal esophagitis in an immunocompetent host 656
JOHN V. YACONO, MD

Farmer's lung in a 10-year-old boy 658
ALLAN S. CUNNINGHAM, MD, JOHN J. MAY, MD, JORDAN N. FINK, MD

Cocaine-induced myocardial infarction 660
LAWRENCE GOULD, MD, CHITRA GOPALASWAMY, MD, CHANDRAKANT PATEL, MD, ROBERT BETZU, MD

LETTERS TO THE EDITOR

Spontaneous complete remission in acute promyelocytic leukemia 662
ROBERT E. ENCK, MD

The physician/participant in war 663
RONALD E. ROSENTHAL, MD

Placental pseudotumor of the uterus: An iatrogenic disease? 663
MARIAN WAXMAN, MD

Breast self-examination 664
NATALIE SHAINESS, MD,
KEVIN M. O'GORMAN, MD

AIDS and the alleged Haitian connection 664
JEAN-ROBERT LEONIDAS, MD

Disseminated histoplasmosis in a man with AIDS 665
SELWYN J. BAPTIST, MD, JOHN B. MONTANA, MD, SHELDON B. ARDEN, PhD, LUCINA LEON, MARIE DUTEL

MSSNY MEDICAL LIABILITY REFORM FUND HONOR ROLL 666

BOOK REVIEWS 671

OBITUARIES 675

MEDICAL SOCIETY AFFAIRS 678

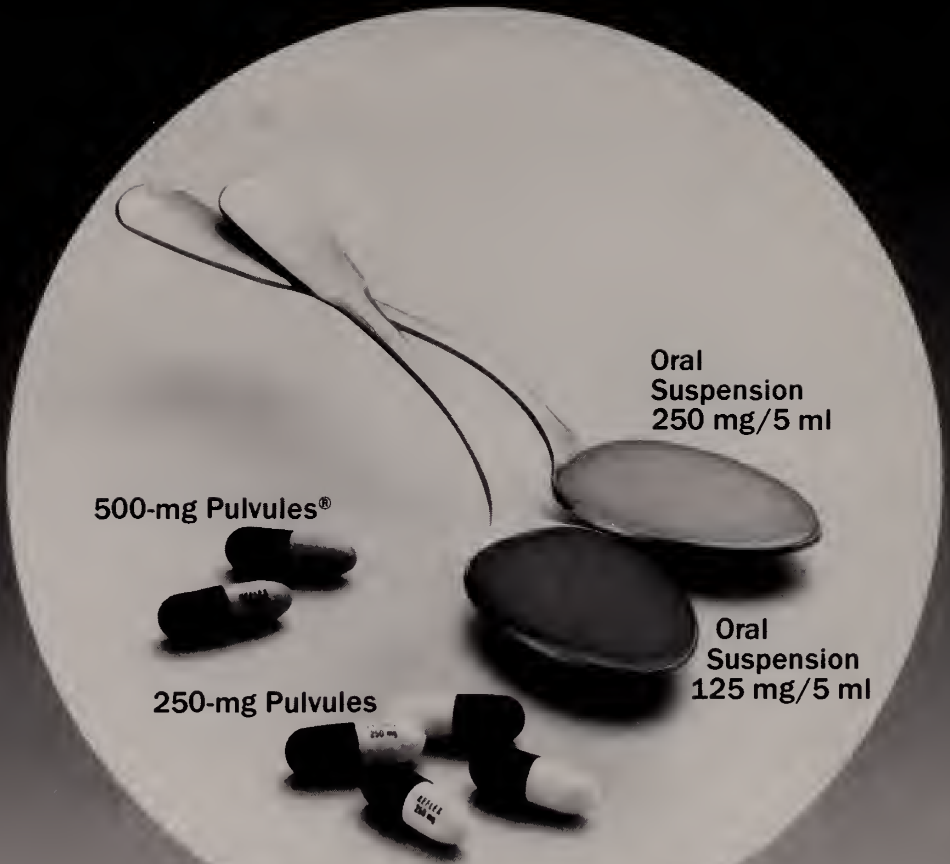
GUIDELINES FOR AUTHORS 678a

MEDICAL MEETINGS AND LECTURES 8A

Annual CME Assemb
(See

Library of the College Physicians
of Philadelphia Dec. 85
19 S. 22nd St.
Phila, PA 19103

Easy To Take



Keflex®
cephalexin

Additional information
available to the profession
on request.

DISTA
420113

Dista Products Company
Division of Eli Lilly and Company
Indianapolis, Indiana 46285
Mfd. by Eli Lilly Industries, Inc.
Carolina, Puerto Rico 00630

NEW YORK STATE
JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD	FRANK C. MAXON, JR, MD
ELIZA H. CALDWELL, MD	RICHARD B. NOLAN, MD
URSULA A. MATULONIS (Medical Student)	JOHN T. PRIOR, MD

Editor	ALAN BLUM, MD
Deputy Editor and Book Review Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Production Manager	KATHLEEN WALSH TULLEY
Librarian and Administrative Assistant	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS
Editorial Assistant	MILDRED ARFMANN
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1985

RANDALL BLOOMFIELD, MD	ROBERT LANDESMAN, MD
ROBERT D. BRANDSTETTER, MD	JAMES M. MORRISSEY, ESQ
JOHN S. DAVIS, MD	STEPHEN NORDLICHT, MD
MARK C. HOMONOFF, MD	JOSEPH SCHLUGER, MD
ALFRED P. INGEGNO, MD	BJORN THORBJARNARSON, MD
FLORENCE KAVALER, MD	RODRIGO E. URIZAR, MD

NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. Synergistic Media Sales, 50 East 42nd St., Suite 504, New York, N.Y. 10017. Telephone: (212) 682-2200.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

DANIEL F. O'KEEFFE, MD,	*President*
ALLISON B. LANDOLT, MD,	*Past-President*
JOHN A. FINKBEINER, MD,	*President-Elect*
SAMUEL M. GELFAND, MD,	*Vice-President*
JOHN H. CARTER, MD,	*Secretary*
GEORGE LIM, MD,	*Assistant Secretary*
CHARLES D. SHERMAN, JR, , MD,	*Treasurer*
MORTON KURTZ, MD,	*Assistant Treasurer*
CHARLES N. ASWAD, MD,	*Speaker*
SEYMOUR R. STALL, MD,	*Vice-Speaker*

Councilors

Term Expires 1986	ROBERT A. MAYERS, MD, Westchester THOMAS D. PEMRICK, MD, Rensselaer RALPH E. SCHLOSSMAN, MD, Queens STANLEY A. STECKLER, MD, Suffolk ROBERT J. NEWBORN, Albany *(Councilor representing the medical student membership)*
Term Expires 1987	DAVID M. BENFORD, MD, New York DUANE M. CADY, MD, Onondaga JAMES M. FLANAGAN, MD, Wayne M. THEODORE TANENHAUS, MD, Brooklyn
Term Expires 1988	JAMES H. COSGRIFF, JR, MD, Erie RICHARD A. HUGHES, MD, Warren ANTONIO F. LASORTE, MD, Broome SIDNEY MISHKIN, MD, Nassau

Trustees

G. REHMI DENTON, MD, Albany, *Chairman*
RALPH M. SCHWARTZ, MD, New York
EDGAR P. BERRY, MD, New York
RICHARD D. EBERLE, MD, Onondaga
JOSEPH F. SHANAPHY, MD, Richmond
VICTOR J. TOFANY, MD, Monroe
BERNARD J. PISANI, MD, New York

Executive Vice-President	GEORGE J. LAWRENCE, JR, MD
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Associate	IRMA A. ERICKSON
Director, Division of Scientific Publications	ALAN BLUM, MD
Director, Division of Management Services	GEORGE FORREST, JR
Assistant Director, Division of Governmental Affairs	JERRY S. HOFFMAN, ESQ
Director, Division of Communications	EDWARD HYNES
Director, Division of Insurance and Membership Benefits	BERNARD M. JACKSON
Director, Division of Membership	EUNICE SKELLY
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Scientific Activities	PETER B. FARNSWORTH, MD
Director, Division of Advertising-Sales and Meeting Services	ROBERT MILLER
General Counsel	DONALD R. MOY, JD
Director, Division of Computer Services	IVAN H. NAJMAN
Acting Director, Division of Medical Services	MARIO MENGHINI
Director, Division of Long-Range Planning	DEBORAH R. SIEGEL
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. Editorial material published in the *Journal* is covered by copyright. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.

Right from the start in hypertension...

Once-daily INDERAL LA (propranolol HCl) for smooth blood pressure control without the potassium problems of diuretics

Once-daily INDERAL LA (propranolol HCl) avoids the risk of diuretic-induced ECG abnormalities due to hypokalemia.[1-2] In addition, INDERAL LA preserves potassium balance without additive agents or supplements while providing simple, well-tolerated therapy with broad cardiovascular benefits.

Once-daily INDERAL LA for the cardiovascular benefits of the world's leading beta blocker

Simply start with 80 mg once daily. Dosage may be increased to 120 mg to 160 mg once daily as needed to achieve additional control.

Like conventional INDERAL tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, heart block greater than first degree, and bronchial asthma.

For beta-1/beta-2 blockade

Once-daily

INDERAL LA

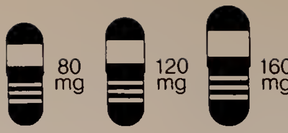
(PROPRANOLOL HCl) [illegible]TING [illegible]

80 mg 120 mg 160 mg

The appearance of these capsules is a registered trademark of Ayerst Laboratories.

Please see brief summary of prescribing information on the next page for further details

Once-daily
For beta-1/beta-2 blockade
INDERAL® LA
(PROPRANOLOL HCl) LONG ACTING CAPSULES



The appearance of these capsules is a registered trademark of Ayerst Laboratories.

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*
INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

The mechanism of the antihypertensive effect of INDERAL has not been established. Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain. Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use Effects on plasma volume appear to be minor and somewhat variable. INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients.

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction. Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential. The significance of the membrane action in the treatment of arrhythmias is uncertain.

The mechanism of the antimigraine effect of propranolol has not been established Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital. For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction. Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

Propranolol is not significantly dialyzable

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)— PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e g , dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA: Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes In these patients, it may be more difficult to adjust the dosage of insulin.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General:* Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies.

Beta adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure

Clinical Laboratory Tests Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS. Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy. Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman.

Pediatric Use Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular bradycardia, congestive heart failure; intensification of AV block, hypotension; paresthesia of hands, thrombocytopenic purpura; arterial insufficiency, usually of the Raynaud type

Central Nervous System lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue; reversible mental depression progressing to catatonia, visual disturbances; hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics

Gastrointestinal: nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory. bronchospasm

Hematologic agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous: alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic The dosage may be increased to 120 mg once daily or higher until adequate blood pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily. In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

REFERENCES

1. Holland OB, Nixon JV, Kuhnert L: Diuretic-induced ventricular ectopic activity. *Am J Med* 1981;70:762-768. **2.** Holme I, Helgeland A, Hjermann I, et al: Treatment of mild hypertension with diuretics. The importance of ECG abnormalities in the Oslo study and in MRFIT. *JAMA* 1984;251:1298-1299

9411/1184

Ayerst® AYERST LABORATORIES
New York, N Y 10017

Copyright © 1984 AYERST LABORATORIES
Division of AMERICAN HOME PRODUCTS CORPORATION

Classified Advertising

PHYSICIANS WANTED

PHYSICIAN-NY, Metropolitan areas. Air National Guard Units are seeking qualified physicians. Flexible active duty time, rewarding work, training opportunities for flying, travel, conferences, CME credits. Retirement benefits and insurance. Serve your state, country and community. For more information call 106th USAF Clinic, Suffolk Cty Air National Guard Base, Westhampton Bch., NY 11978. (516) 288-4200 Ext. 247.

N.Y.C. HOLISTIC HEALTH CENTER with heavy patient load looking for physician (G.P. or Internal Medicine) to refer our patients to. Office available full or part-time, flexible hours. Please send CV and resume to: Shari Lieberman, M.A., R.D., Nutritionist, 60 East 8th Street, Apt. 14C, NY, NY 10003.

ARIZONA BASED PHYSICIAN RECRUITMENT firm has opportunites Coast to Coast. "Professionals working with Professionals". Over 13 years experience. Call (602) 795-7474; or send CV to: Mitchell & Associates, Inc., 2761 N. Country Club Rd., Suite 202, Tucson, AZ 85716.

DESPERATELY SEEKING ASSOCIATE to share some expenses and gradually (3–5 yrs.) take over active internal medicine practice in prime location, White Plains, NY. Prefer creative, industrious, entrepreneurial individual with some capital. Send cv to Dept. 393 c/o NYSJM.

INTERNAL MEDICINE, large practice outside Buffalo, New York for sale. For more information send C.V. to: Health Care Personnel Consulting, Inc., 400 GSB Building, One Belmont Avenue, Bala Cynwyd, PA 19004 (215) 667-8630.

SURGERY: GENERAL PRACTICE AVAILABLE—this GP/GS combination practice in upstate New York is a unique opportunity for the physician interested in a broad practice. Send C.V. to: Health Care Personnel Consulting, Inc., 400 GSB Building, One Belmont Avenue, Bala Cynwyd, PA 19004 (215) 667-8630.

EASTERN LONG ISLAND, NEW YORK. Internist wanted to form a partnership in a busy primary care practice. Position immediately available. Reply Dept. 396 c/o NYSJM.

GASTROENTEROLOGIST: Two man gastroenterology group in Central New York near Syracuse looking for third M.D. One hundred percent gastroenterology practice. Excellent opportunity. Many recreational facilities. Must be proficient in all phases of invasive gastroenterology, endoscopy, and nutrition. Please send curriculum vitae with three references to Dept. 397 c/o NYSJM.

PHYSICIANS WANTED—CONT'D

FAMILY PRACTITIONER—Established medical group needs a family practitioner. Located in the sun-belt and no state tax. Guaranteed salary and early partnership for the conscientious person. Contact clinic administrator 10737 Gateway West, El Paso, Texas 79935 or call (915) 592-0201.

PRACTICES AVAILABLE

SELLING PRACTICES IS OUR ONLY BUSINESS—Want to maximize the return on your investment? Before you buy or sell, call for a consultation to assure the best financial and transfer terms. We guide you through the entire sales process from initial meeting to closing. Serving 100 mile radius of New York City. Countrywide Business Brokerage, Inc., 319 East 24th Street, Suite 23-G, New York, NY 10010 (212) 686-7902.

MEDICAL PRACTICE SALES and Appraisals. We specialize in the valuation and selling of medical practices. If interested in buying or selling a medical practice, contact our Brokerage Division at: The Health Care Group, 400 GSB Building, Bala Cynwyd, PA 19004. (215) 667-8630.

INTERNAL MEDICINE—Active, long-established solo practice for sale. Nassau county; home-office fully equipped for 1 or 2 physicians, within minutes of several hospitals. Phone (516) 747-3072.

APPRAISALS-SALES-RELOCATION
Medical Practices—All Specialties

MEDICAL PRACTICES TRANSFER
Frederick Katz, Ph.D. & Assoc.

42-27 Union Street, Flushing, N.Y. 11355
Tel. (718) 445-9797 (any time)

POSITIONS WANTED

PULMONARY INTERNIST, 30, ABIM certified, completing fellowship June 1986. Fully trained in all aspects of pulmonary medicine. Seeks hospital-based or subspecialty group practice affiliated with a teaching institution. Reply Harlan Weinberg, M.D., 337 N. Palm Drive, Beverly Hills, CA. 90210, Apt. 8.

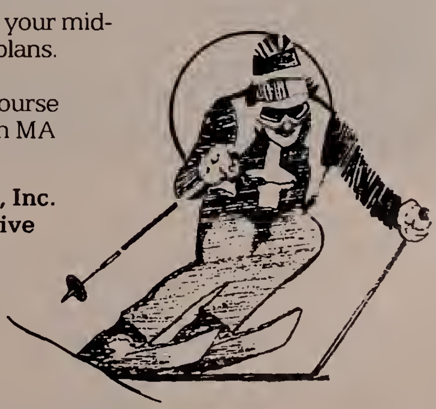
GASTROENTEROLOGIST-ABIM, University-trained in all endoscopic procedures, ERCP, PEG, peritoneoscopy and consultative gastroenterology/hepatology. Seeking private practice associateship leading to partnership in the NYC/LI area. Available after completion of fellowship in July 1986. Reply Dept. 399 c/o NYSJM.

Tax-deductible Seminars At Five Vermont Ski Areas

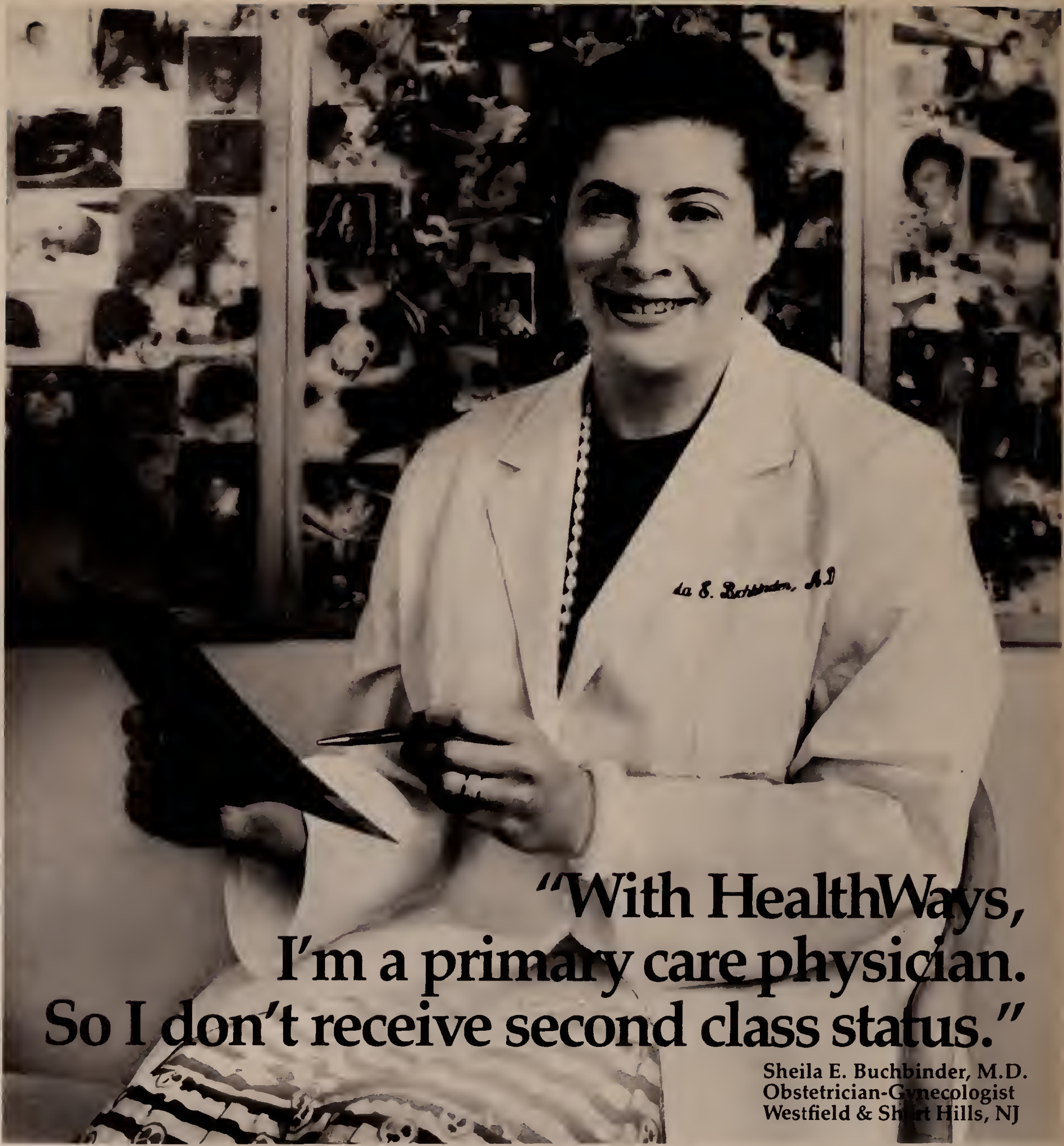
Register now for **4-session course on medical malpractice** now offered throughout the season at Mt. Snow, Stratton-Bromley, Killington, Sugarbush and Stowe.

Flexible scheduling adjusts to your midweek and/or weekend vacation plans. Tuition only $140.

For brochure with complete course description call 800/447-1175 (in MA 413/525-4785).



Eastern Educational Programs, Inc.
Benton Building, 15 Benton Drive
East Longmeadow, MA 01028



"With HealthWays, I'm a primary care physician. So I don't receive second class status."

Sheila E. Buchbinder, M.D.
Obstetrician-Gynecologist
Westfield & Short Hills, NJ

"It's difficult to comprehend, but many health care plans do not consider Obstetrician-Gynecologists such as myself to be a primary care physician. With those plans, I can't be freely chosen by patients, but require a referral. That's one of the many reasons why I participate in HealthWays.

With HealthWays, Obstetrician-Gynecologists are primary care physicians, which means my HealthWays patients still see me directly for all care—both routine and specialized—with no referral necessary. HealthWays doesn't ignore what primary care is for a woman.

And with HealthWays, I'm not forced to accept a big reduction in my fee. Instead, I am compensated with traditional fee-for-service.

As far as I'm concerned, HealthWays is not only first class for my patients, it's first class for me."

To start HealthWays working for you and your patients, call HealthWays' IPA Department at 1-800-624-0720 in New Jersey or 201-636-6200 out-of-state.

HealthWays
THE WAY TO BETTER HEALTH CARE

© 1985 HealthWays, Inc.

Added Control for America's Coughs

Robitussin A-C®℗

Robitussin and codeine
Each 5 ml (1 teaspoonful) contains:
Guaifenesin, USP . . . . . . 100 mg
Codeine Phosphate, USP . . 10 mg
(Warning: May be habit forming)
Alcohol 3.5 percent
In a palatable, aromatic syrup

The expectorant action of guaifenesin plus the **A**dvantage of **C**odeine!

A·H·ROBINS
Pharmaceutical Division, Richmond, Virginia 23261-6609

©A.H. Robins Company 1985

FIGHT LUNG DISEASE WITH CHRISTMAS SEALS.®

Christmas Seals do so much more than make your gifts and cards sparkle. They help fight lung disease, smoking and air pollution. So everyone benefits. Can you think of a more meaningful message to send to your friends and family at holiday time! Show them that you really care by using Christmas Seals from the American Lung Association.

TAKE CARE OF YOUR LUNGS. THEY'RE ONLY HUMAN.

AMERICAN LUNG ASSOCIATION
The Christmas Seal People®

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meetings and lectures unless the information is submitted at least three months prior to the event.*

DECEMBER 1985

AROUND THE STATE

MANHATTAN

Dec 5. **Depression in the Elderly.** *Dec 12.* **A Study of Families of Some Chronically Ill Patients.** *Dec 19.* **Hypnosis in the '80s: Uses and Misuses.** Info: Seymour Gers, MD, Director, Medical Education, Manhattan Psychiatric Center, Ward's Island, NY 11035. Tel: (212) 369-0500.

Dec 5–6. **Current Topics in Gastroenterology.** 16 Cat 1 Credits. Info: Dr Elizabeth C. Gerst, Assistant Dean for Continuing Education, 630 West 168 St, New York, NY 10032. Tel: (212) 305-3682.

Dec 6–7. **Implications of Molecular Neurobiology for Neurology and Psychiatry.** Info: Bernard Cohen, MD, Mount Sinai School of Medicine, One Gustave L. Levy Pl, Room 21–74, New York, NY 10029. Tel: (212) 348-8133.

Dec 7. **Stress and Medical Illness.** 6 Cat 1 Credits. *Dec 9–11.* **Managing Clinical Problems in the Elderly.** 20 Cat 1 Credits. *Dec 9–12.* **Neurosurgery NYC/1985.** 22 Cat 1 Credits. *Dec 13–14.* **Ovarian Cancer: Therapeutic Results and Exciting New Leads.** 7 1/2 Cat 1 Credits. *Dec 16–21.* **Computed Tomography Head to Toe.** 39 Cat 1 Credits. Info: NYU Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Dec 7. **Ocular Pharmacology.** Info: Martha B. Klapp, Administrator, Department of Ophthalmology, Manhattan Eye, Ear & Throat Hospital, 210 East 64 St, New York, NY 10021. Tel: (212) 605-3762.

Dec 14. **Voice Restoration.** Info: Kristen M. Johnson, Administrator, Department of Otolaryngology, Manhattan Eye, Ear & Throat Hospital, 210 East 64 St, New York, NY 10021. Tel: (212) 838-9200.

BROOKLYN

Dec 4. **Environmental Carcinogenesis.** *Dec 18.* **Polycythemia Vera: Diagnosis and Treatment.** Info: Division of Hematology/Oncology, Interfaith Medical Center, 555 Prospect Pl, Brooklyn, NY 11238. Tel: (718) 240-1211.

Dec 5–6. **The Application of the CO_2 Laser in Otolaryngology Head and Neck Surgery.** 16 Cat 1 Credits. Info: Yosef P. Krespi, MD, Long Island College Hospital, Department of Otolaryngology, Room 460, Brooklyn, NY 11201. Tel: (718) 780-1281.

QUEENS

Dec 3. **Programmed Ventricular Stimulation.** *Dec 10.* **What's New in Hepatitis B.** *Dec 17.* **Symposium: DRG's.** Info: Ms. Clo Vezza, Coordinator, Continuing Medical Education Committee, Booth Memorial Medical Center, 56–45 Main St, Flushing, NY 11355. Tel: (718) 340-1419.

NASSAU

Dec 6. **NMR Imaging.** *Dec 20.* **Pre-operative Evaluation and Preparation of ASHD Patients.** Info: Medical Staff Office, Long Beach Memorial Hospital, 466 East Bay Dr, Long Beach, NY 11561. Tel: (516) 432-8000, ext 2275.

SUFFOLK

Dec 4. **Current Controversies in Cardiology.** Info: American Heart Association, 600 Johnson Ave, Bohemia, NY 11716. Tel: (516) 567-7900.

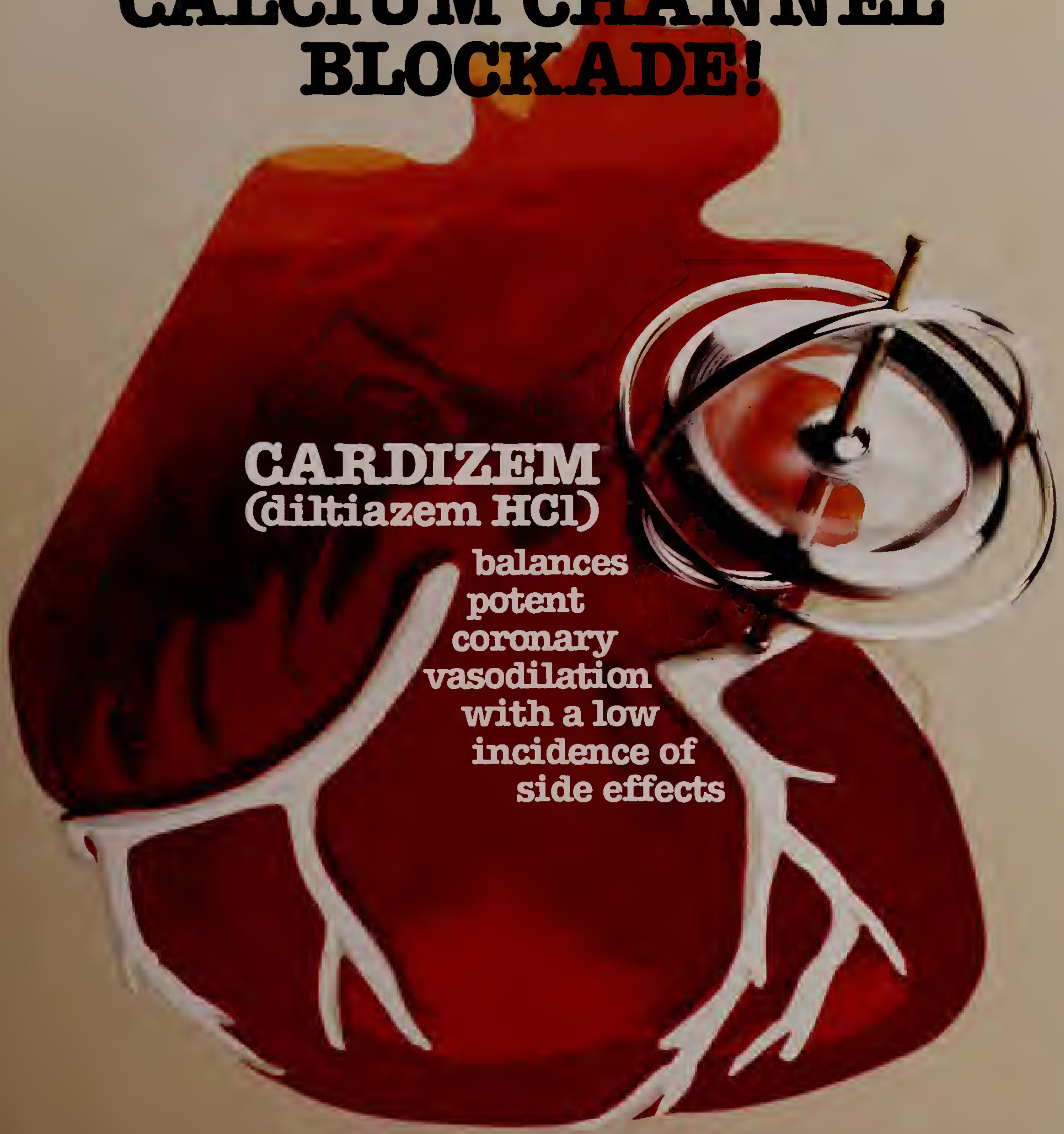
Dec 9. **Thrombolysis and Acute Myocardial Infarction.** 6 Cat 1 Credits. Info: Donna C. Joslin, CME Secretary, Southampton Hospital, Medical Records, 240 Meeting House Lane, Southampton, NY 11968. (516) 283-2600, ext 505.

ROCKLAND

Dec 3. **Hypertension: Emphasis on Compliance and Management of Sexual Dysfunction in Hypertensive Patients.** 1 Cat 1 Credit. Info: Mrs. Myrna Hahn, Secretary, Department of Internal Medicine, Nyack Hospital, North Midland Ave, Nyack, NY 10960. (914) 358-6200, ext 2509.

(*Continued on p 22A*)

BALANCED CALCIUM CHANNEL BLOCKADE!


CARDIZEM
(diltiazem HCl)
balances potent coronary vasodilation with a low incidence of side effects

Low incidence of side effects
CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

Reduces angina attack frequency*
42% to 46% decrease reported in multicenter study.[1]

Increases exercise tolerance*
In Bruce exercise test,[2] control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes ($P<.005$).

CARDIZEM®
(diltiazem HCl)

THE BALANCED CALCIUM CHANNEL BLOCKER

Please see full prescribing information on following page. 2/84

---

*Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

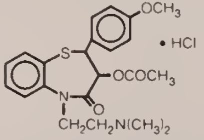
References:
1. Strauss WE, McIntyre KM, Parisi AF, et al: Safety and efficacy of diltiazem hydrochloride for the treatment of stable angina pectoris: Report of a cooperative clinical trial. Am J Cardiol 49:560-566, 1982.
2. Pool PE, Seagren SC, Bonanno JA, et al: The treatment of exercise-inducible chronic stable angina with diltiazem: Effect on treadmill exercise. Chest 78 (July suppl):234-238, 1980.

PROFESSIONAL USE INFORMATION

cardizem®
(diltiazem HCl)
30 mg and 60 mg tablets

DESCRIPTION

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one,3-(acetyloxy)-5-[2-(dimethylamino)ethyl]-2,3-dihydro-2-(4-methoxyphenyl)-, monohydrochloride,(+)-cis-. The chemical structure is:



Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform. It has a molecular weight of 450.98. Each tablet of CARDIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration.

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth muscle.

Mechanisms of Action. Although precise mechanisms of its antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways:

1. Angina Due to Coronary Artery Spasm: CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial and subendocardial. Spontaneous and ergonovine-induced coronary artery spasm are inhibited by CARDIZEM.
2. Exertional Angina: CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal and maximal exercise work loads.

In animal models, diltiazem interferes with the slow inward (depolarizing) current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Diltiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance.

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricular conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load. Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect; cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of diltiazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by diltiazem.

Intravenous diltiazem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Diltiazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, diltiazem significantly prolongs sinus cycle length (up to 50% in some cases).

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Diltiazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40%. CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 60 mg are given; a 120-mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem.

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm.** CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina).** CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS).

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia.
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing.
Cardiac Failure	Administer inotropic agents (isoproterenol, dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

The oral/LD_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort acute anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy**—CARDIZEM may be safely coadministered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

Cardizem 30-mg tablets are supplied in bottles of 100 (NDC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1771-49). Each green tablet is engraved with MARION on one side and 1771 engraved on the other. CARDIZEM 60-mg scored tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1772-49). Each yellow tablet is engraved with MARION on one side and 1772 on the other.

Issued 4/1/84

Another patient benefit product from
PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MISSOURI 64137

Medical Society of the State of New York

ANNUAL CME ASSEMBLY

Maintaining the Quality of
Health Care into the 21st Century.

March 7–9, 1986

and the

Annual Meeting of the House of Delegates

March 6–9, 1986

The New York Hilton
New York City

COURTESY N.Y. CONVENTION & VISITORS BUREAU

HIGHLIGHTS . . .

- 22 Sessions
- Panel Discussions
- Technical Exhibits
- President's Dinner Dance

CLIP & MAIL

Director of Front Office Operations
The New York Hilton
1335 Avenue of the Americas
New York, N.Y. 10019

Phone: 212-586-7000

for hotel use only

NAME ______________ TITLE ______________

FIRM ______________ PHONE ______________

STREET ______________

CITY/STATE/ZIP ______________

REMARKS ______________

(please print)

NOTE: Reservations must be received no later than February 13, 1986 and will be held only until 6 p.m. on day of arrival unless later hour is specified.

please print

ARRIVAL DATE ______________

☐ 2-4 p.m. ☐ 4-6 p.m. ☐ 6-8 p.m. ☐ after 8 p.m.

DEPARTURE DATE ______________

(checkout time is 1 p.m.)

HAVE A CREDIT CARD? You can register at our QUICK CHECK desk. If you wish to take advantage of this credit card PAYMENT GUARANTEED reservation, please complete the information below.

☐ AMERICAN EXPRESS
☐ DINERS CLUB
☐ CARTE BLANCHE

Expiration Date ______________

____
Credit Card No.

Signature ______________

MEDICAL SOCIETY OF THE STATE OF NEW YORK
Annual Meeting of the House of Delegates
March 6-9, 1986
Annual CME Assembly
March 7-9, 1986

Circle preferred rate:

Singles: $125 $135 $145
Double/Twin: $150 $160 $170
Executive Tower: $200, Single/Double
Suites: (P&1): $275 $340 $375
Suites: (P&2): $450 $485

Rates subject to 8¼% N.Y. State sales tax and $2.00 per room per night N.Y.C. occupancy tax. (If room at the rate requested is unavailable, one at the nearest available rate will be reserved.)

GENERAL INFORMATION

The Medical Society of the State of New York invites you to attend its 1986 Annual CME Assembly to be held March 7–9, at the New York Hilton. The Continuing Medical Education Sessions and Technical Exhibits are open to all physicians, their guests and the allied professions. The two and a half days of meetings will provide the individual physician with a possible 18 credits out of a total of 93 Category 1, CME credit hours being offered. Over 120 outstanding speakers, including a number from out-of-state, will present papers at 22 sessions.

The theme of the CME Sessions will be "Maintaining The Quality of Health Care Into The 21st Century." A few of the subjects to be presented are, Alcoholism and Gastroenterology; Realistic Uses of Computers in Medical Practice; Evaluation and Management of Temporal Mandibular Joint Disorders; Laser Therapy in Urogenital Disorders; and An Overview of Geriatric Psychiatry. For details, see the Preliminary Program which appears in the December 1985 issue of the *New York State Journal of Medicine*. Our program will offer the clinician new approaches in diagnosis and treatment of diseases in a wide range of medical specialties.

COURTESY NEW YORK CONVENTION AND VISITORS BUREAU

MUSEUM OF MODERN ART SCULPTURE GARDEN

Comments from attendees at the 1985 CME Assembly were: 'The Speakers are excellent and well-informed'; 'The presentation was timely, and the material was well illustrated'; 'My time was well spent'. Don't miss this opportunity to gain Category 1 credits over a weekend when you are free from office hours. Why not bring your family. There is no registration fee for MSSNY members. Professional representatives from more than 50 companies will be in the Rhinelander Gallery Exhibit Hall to discuss their products and services. The Technical Exhibits will include drug products, clinical laboratories, office equipment, computer services, educational, insurance protection, and financial services, career opportunities and more.

SPECIAL EVENTS

Thursday, March 6

The Annual Dinner Dance will be held in the Trianon Ballroom of the New York Hilton, in honor of Daniel F. O'Keeffe, M.D., President, preceded by a reception in the Mercury Ballroom at 7:00 p.m. All attendees are welcome. Tickets are $75.00 per person and should be ordered in advance from the Medical Society.

Friday, March 7 and Saturday, March 8

A reception for physicians, their guests, the allied professions and exhibitors will be held on both days in the Exhibit Hall between 5:00 p.m. and 6:00 p.m., compliments of the Medical Society of the State of New York. A free drawing for a color TV set will take place at both receptions.

HOTEL RESERVATIONS

Physicians must make their own reservations with the New York Hilton no later than February 13, 1986. After that date, rooms are on an availability basis only. If making reservations by telephone, please mention that you are attending the Medical Society Convention for billing at the special rate.

REGISTRATION

CME Registration will take place in the Rhinelander Gallery Exhibit Hall, located on the 2nd floor which is accessible by escalators from the lobby. Friday, March 7 and Saturday, March 8 from 8:30 a.m. to 5:00 p.m.; Sunday, March 9 from 8:30 a.m. to 12:30 p.m.

House of Delegates Registration will take place on the 2nd and 3rd floor promenades at the following hours: Wednesday, March 5, from 12 Noon to 7:30 p.m.; Thursday, March 6, Friday, March 7 and Saturday, March 8, from 8:30 a.m. to 5:30 p.m. and Sunday, March 9 from 8:30 a.m. to 12 Noon. All MSSNY members are welcome.

CME MEETING HOURS

Friday, March 7	—10 a.m.–1 p.m. 2 p.m.–5 p.m.
Saturday, March 8	—10 a.m.–1 p.m. 2 p.m.–5 p.m.
Sunday, March 9	—10 a.m.–1 p.m.

EXHIBIT HOURS

Friday, March 7	—9 a.m.–11 a.m. 1 p.m.–6 p.m.
Saturday, March 8	—9 a.m.–11 a.m. 1 p.m.–6 p.m.
Sunday, March 9	—9 a.m.–1 p.m.

1986 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
New York Hilton, New York City

Maintaining the Quality of
Health Care into the 21st Century

FRIDAY, MARCH 7

10:00 a.m. - 1:00 p.m.	Cardiovascular Diseases Gastroenterology/Alcoholism Obstetrics/Gynecology Neurosurgery Physical Medicine & Rehabilitation
2:00 p.m. - 5:00 p.m.	Neurology Pediatrics/Alcoholism Chest Diseases Occupational & Environmental Health/Workers Comp. Medical Liability Mutual Insurance Co.

SATURDAY, MARCH 8

10:00 a.m. - 1:00 p.m.	Ophthalmology (All Day) Internal Medicine (All Day) Hand Surgery/Anesthesiology Urology/Dermatology Psychiatry
2:00 p.m. - 5:00 p.m.	Ophthalmology Internal Medicine Otolaryngology Hand Surgery/Anesthesiology Psychiatry

SUNDAY, MARCH 9

10:00 a.m. - 1:00 p.m.	Allergy & Immunology General Surgery Women's Medical Society of N.Y./Family Practice Pathology

◆

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis.

◆

Technical Exhibits

Central Park Lake

Washington Square Arch

COURTESY N Y. CONVENTION & VISITORS BUREAU

Advance Registration Form

1986 Annual CME Assembly, March 7-9
The New York Hilton, New York City

Complete and return this form to the Medical Society address as indicated below.

Name____
PLEASE PRINT LAST FIRST

Address____

City/State/Zip____________________________________

Medical Specialty__________________________________

REGISTRATION FEE *Please check categories*

☐ Non-Member Physician	$50.00	☐ Nurse	☐ Lab Technician
☐ Allied Profession	$10.00	☐ Physician Assist.	☐ PT/OT
		☐ Dentist	☐ Other__________ Specify

NO FEE *Please check category*

- ☐ MSSNY Active Member
- ☐ MSSNY Life Member
- ☐ Resident/Intern
- ☐ Medical Student
- ☐ Medical Assist.
- ☐ Speaker
- ☐ Spouse/Family
- ☐ News Media/Medical Writer

REMITTANCE ENCLOSED $______________

Please make check payable to:

Medical Society of the State of New York, and return this form with remittance to: Division of Meeting Services/MSSNY, 420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

Important: Your ID Badge will be held at the MSSNY Registration Desk located in the Exhibit Hall, on the second floor of the New York Hilton.

NEW YORK STATE
JOURNAL OF MEDICINE

November 1985 • Volume 85, Number 11

HEALTH CARE IN THE 21st CENTURY

Reflections on health care at the coming turn of the century

LOWELL E. BELLIN, MD

It is but 15 years to the turn of the century. In the year 2000 what may be anticipated with respect to the delivery of health care? In this discussion some of the forces in play 15 years hence shall be considered: the shifting roles of health care practitioners; the advent of health planning with consumer participation; the effect of the Professional Standards Review Organizations; the innovation of private review in industry; and the continuity versus accessibility of care.

ROLE OF THE HEALTH CARE PRACTITIONER

Limited licensure refers to health care professionals who are legally permitted to provide health care services within anatomic or physiologic limitations. For example, the dentist is restricted to diagnosis and treatment of illness afflicting the teeth and gums. The dentist is permitted to prescribe local and systemic pharmacologic agents. Other limited licensed health care practitioners include nurses, optometrists, and podiatrists—the latter two are permitted in many states to prescribe systemic medications.

In contrast, the MD degree and a medical license bestow on the physician the legal right to provide comprehensive non-surgical and surgical services. In theory any credentialed physician is permitted, if he or she is so inclined, to do anything in the field of therapeutics—from treating complex cardiac arrhythmias to excising brain tumors. In actuality this freedom given to the physician is untrue. The physician who wishes to obtain and retain his or her hospital staff privileges functions as a "limited" licensed practitioner; limited within the conventional bounds of a specific specialty. The state public agency responsible for the professional licensure of health care practitioners actually carries out little enforcement of regulatory provisions with respect to what a physician may or may not do. It is the hospital, with its policies of which physician is admitted and not admitted to the hospital's attending staff, that has come to fulfill this quasi-public function.

Whatever predominant method of financing and payment for health care services exists in the 1990s, functional roles will have to be sorted out and assigned to health care professionals, paraprofessionals, and technicians. An individual bearing relevant credentials of education and experience will still have to perform the services of diagnosis and therapy. Who will do what, for whom, and to whom? These are not trivial questions. It is desirable to spend some time on this subject because without qualified and responsible health care practitioners there can be no health care delivery program at all.

A representative list of historic and contemporary conflicts over professional domain points to unresolved quar-

---

Address correspondence to Dr Bellin, Professor of Public Health, Columbia University, School of Public Health, 600 West 168 St, New York, NY 10032.

Based on a presentation at the symposium entitled, *The U.S. Health Care System into the 21st Century*, January 21, 1985, New York University, New York City.

rels over jurisdiction. Therefore, the scope of professional licensure is *not* altogether stable. Long established distinctions of turf boundaries grow blurry and there are ambiguities of roles. The following nonexhaustive list of dyads in conflict over domain should be considered, as well as the jurisdictional conflicts between physician and nonphysician and between physician and physician that exist today.

- Nurse practitioner versus Physician
- Optometrist versus Ophthalmologist
- Clinical psychologist versus Psychiatric social worker versus Psychiatrist
- Podiatrist versus Orthopedic surgeon
- Otolaryngologist versus Plastic surgeon
- Oral surgeon (DDS) versus Oral surgeon (MD)
- Internist versus Subspecialist in Cardiology
- Internist versus Subspecialist in Gastroenterology
- Internist versus Subspecialist in Rheumatology
- Internist versus Emergency physician
- Nurse anesthetist versus Anesthesiologist
- General surgeon versus Gynecologic surgeon
- Physical therapist versus Physiatrist

A few of these representative dyads are considered in greater detail.

Nurse practitioner versus Physician (MD). During the late 1950s I was one of two National Institutes of Health (NIH) fellows in cardiology at a hospital in Boston. One of the duties as a cardiology fellow was to act as a consultant on-call to hospital interns and residents who might be having difficulty interpreting the more complex electrocardiographic tracings. Naturally, only physicians were thought intellectually capable of assuming so grave a responsibility. The suggestion that any other health care practitioner may some day share that responsibility with the physician would have been scorned.

Today, at this same hospital in Boston, as at many American hospitals, the policy has changed considerably. On a minute-to-minute basis, the coronary care nurse now makes the crucial differential diagnoses. It is also the coronary care nurse who injects the appropriate medication in a timely fashion in order to abort the dangerous arrhythmias, often before the physician gets there. This is an example of transfer and reassignment of responsibility from one professional to another as a consequence of high technology. Nurses at the turn of the century will continue to assume responsibilities once within the exclusive domain of physicians.

Optometrist versus Ophthalmologist. This age-old conflict most recently has encompassed, among other things, the fitting and application of soft contact lenses. Ten years ago a legal challenge was issued by ophthalmologists in a western state contesting the rights and qualifications of optometrists to fit and apply soft lenses. The ophthalmologists argued that soft lenses were, in effect, drugs, and that optometrists, at that time, were forbidden to dispense drugs. Therefore, by logical extension optometrists should not be permitted to dispense lenses. The argument was original and ingenious, but the court was not persuaded by the analogy and ruled in favor of the optometrists.

The two groups were also in conflict over the right of the optometrist to use diagnostic eye drops to enhance the funduscopic examination for retinal and vascular disease. Traditional optometry had been a drugless profession. As a result of successful lobbying on the part of organized optometry, optometrists may now dispense diagnostic eye drops legally in over 40 states. In West Virginia, moreover, optometrists have lobbied effectively for the right to use therapeutic as well as diagnostic eye drops (eg, in treating conjunctivitis). The use of eyedrops for therapeutic purposes may well be the next area of nationwide contention.

In short, optometrists, like nurses, can serve as a model for expanding the scope of licensure of health care practitioners. Optometrists have thereby strengthened their professional legitimacy. As part of the grand strategy, colleges of optometry throughout the country have now included in their curricula courses in pharmacology. By the year 2000 optometrists may be performing all the functions of the ophthalmologist with the one exception of surgery.

Psychologists versus Psychiatric social worker versus Psychiatrists. Psychiatrists customarily argue that by virtue of their being physicians, only they are qualified to diagnose and treat mental disorders. During medical school and hospital training, psychiatrists receive the pertinent formal education in physical diagnosis and internal medicine to recognize organic disease as a possible etiology of mental health problems. Thyroid disease, brain tumor, or electrolyte imbalance may all mimic functional mental illness. Psychologists and psychiatric social workers argue that all patients under their care are examined periodically by the appropriate physician as a precautionary measure to exclude organic disease. The remainder of the patient's illness can then be handled quite proficiently by the clinical psychologist or the psychiatric social worker. It is often argued that there is no support in the professional literature behind the claim that more effective treatment of mental illness is provided by the psychiatrist over the MS or PHD therapist.

Podiatrist versus Orthopedic surgeon. Podiatrists today are no longer the chiropodists of yore who worked in shoestores with barely enough education to care for calluses and corns. The podiatrist today has completed a minimum of two years of pre-professional college followed by four years of professional education at a college of podiatry. Through training and experience the podiatrist can apply his surgical and medical skills to the bones, joints, fascia, muscle, and vascular tissue of the foot. Like dentists, podiatrists can now write prescriptions for drugs with systemic pharmaceutical effects without the supervision of a physician.

Otolaryngologist versus Plastic surgeon. In the past mastoidectomy was a common procedure of the otolaryngologist—as well as a source of considerable income. However, the advent of antibiotics made the mastoidectomy a rarity among surgical procedures, and, incidentally, had a devastating impact on the income of otolaryngologists. These physicians were therefore compelled to look to nearby anatomical areas to transfer their professional attention. Not surprisingly the plastic surgeons have resented this incursion. They have sternly opposed such an extension of otolaryngologic activity to nasal plastic surgery and to bronchoscopy.

All of the above conflicts have certain features in common:

- ambition of the health care practitioner to expand his or her professional domain for increased income and prestige;

- denigration by each group of the professional qualifications or uniqueness of the other group;
- and the claim that the challengers can do the professional work more cheaply.

The desacralization and demystification of the physician practice are common today. The current atmosphere of skepticism has permitted and perhaps accelerated the shift of MD responsibilities to non-MD practitioners who have successfully challenged the traditional exclusivity of the non-MD or MD health care practitioner.

It is unlikely that the professional responsibilities of any health care practitioner, having once been expanded, will ever shrink again. No group willingly surrenders privileges that have been gained by such an extraordinary effort and this expansion will continue to blur the distinctive boundaries of professional domain. The practitioner of the 21st century may still be called "Doctor" or "Nurse" but the titles will suggest something different. In any case, the titles will probably be modified or abandoned long before the recently acquired rights and responsibilities will be surrendered.

HEALTH PLANNING

On the government level, planning in this country has been suspect, hinting at socialism and perhaps worse. Planning has been viewed as antithetical to the spontaneity that has been deemed somehow more American. Until recently the American virtues of spontaneity and nonplanning seemed to be good enough, and apparently better than good enough. The programs derived from this system had worked seemingly well over the years. Unlike other countries, the United States could afford spontaneity even at the price of duplication and overlapping of certain programs. Each institution, hospital, or nursing home focused exclusively on what it perceived to be its own institutional needs. What was good for General Hospital A was deemed automatically good for the community that was supporting Hospital A, and incidentally supporting Hospitals B and C.

Horror stories have been told about overbuilding and "overbedding." In one town there may be two children's hospitals when one would have sufficed—two open heart surgery units, two home care programs, two oncology units—or, in some superfluous cases, more than two. The counter-argument to rational planning was that the rivalry of scholars increases wisdom, and continuing competition is indispensible. Unfortunately, formal planning may become stifling and anticompetitive. Those health care institutions already in the community try to prevent the competition from getting in. Planning "grandfathers" the old and can be used perversely to grant refuge to the obsolete.

In the year 2000 some agency, perhaps the current Health System Agencies (HSA), will be handling community-wide health planning in order to optimize the use of scarce resources. An important portion of HSA experience should be kept in mind: It has been assumed over the years that health care consumers, more than health care providers, would be motivated to do a more objective job and make more rational decisions on behalf of resource allocations to the general community. This assumption has yet to be proven. For no less than the provider, the consumer demands his or her share of the most recent technologic advances. The history of computed tomographic scanners and nuclear magnetic resonance imagers has shown that the consumer is no less zealous than the provider in lobbying on behalf of his constituency to the detriment of community-wide planning. If so, it is perhaps better political strategy to shift the center of planning decision-making to the state capital; more remote from local squabbles and pressures that subvert planning. In any case formal health planning will continue into the 21st century, and the political process that is part of this planning will likewise continue.

PROFESSIONAL STANDARDS REVIEW ORGANIZATIONS AND QUALITY ASSURANCE

Physicians in private practice have opposed the proliferation of publicly subsidized health care delivery programs. They feel that these programs pose an economic threat to their practices and an unwarranted incursion into health care delivery. The most strident opposition on the part of physicians has thus far been reserved for government imposition, enforcement, and promulgation of quality control standards—as in the Handicapped Children's Program, in the obstetrical program for wives of service men during World War II, and, more recently, in the activities of the Utilization Review (UR) committees in hospitals reimbursed by Medicaid and Medicare.

During the mid 1960s any hospital wishing to participate in Medicare was obliged to subject it's in-hospital care to the UR committee. Hospitals depended on publicly funded health care delivery services for day-to-day survival and to put off bankruptcy. UR committees were charged with the responsibility of examining the reasons put forth by the physician for admission and continued hospitalization of patients. In the absence of adequate justification the hospital would be held liable and therefore receive no payment for hospital care. The ultimate threat of sanction and separation of both the hospital and physician from the federal program was to be reserved for the most aberrant abuse.

This program, however, did not prove to be successful. Some UR committees met for a few hours each month, which was clearly an inadequate amount of time to carry out a serious review. Moreover, some members of the UR committees were simultaneously on the staffs of the hospitals they were reviewing. How could there be objectivity in chart review when doctors from Hospital A reviewed other doctors from Hospital A within the same consultative and social network? Errant hospitals and health care practitioners in such UR territories faced little more than a mild reprimand.

The assessment by Congress of the worth and effectiveness of the UR committees was reflected in the intensity of support given to the formation of the Professional Standards Review Organization (PSRO), the successor to the UR committees. With respect to cost and quality control the PSROs constitute a mosaic.[1] Some PSROs identified only the most flagrant abuses while others were more stringent. However, quality control and cost control of health care delivery services have become nonexpendable. There is neither a political nor administrative alternative. Any government that would spend billions of dollars annually for health care service is obliged to create a system to monitor the cost and quality of these services.

Since its inception the PSRO has faced the scrutiny and criticism of federal agencies. Today that same organization has been restructured into a more austere form—the Professional Review Organization (PRO)—because the formal review of professional work performed has become a permanent managerial feature of health care.

This phenomenon will not necessarily be limited to publicly funded health care services such as Medicaid or Medicare. PSROs or PROs have moved into the field of private review. Contracts are being drawn up by PSROs or PROs and private industries for the peer review organization to evaluate the quality and cost of care. This is to determine whether the industry is getting its money's worth from the health care insurance benefits that the industry and its employees have supported. It is no surprise that preliminary findings suggest that poor quality and overutilization already recognized in the public sector have their functional counterparts in the private sector as well.

CONTINUITY VERSUS ACCESSIBILITY OF CARE

To the chagrin of public health professionals, patients do not appear to be as interested in continuity of care as much as public health practitioners would like them to be. When given a choice between accessible care that is episodic or less accessible care that provides continuity, the patient will most often opt for episodic care.

Just as diagnostic and treatment domains of responsibility are shifting between competing health care practitioners, there is continuing geographic shifting of the setting of professional services. Hospital inpatient treatment is now being offered at the surgi-center and the physician's office, and emergency room treatment at the emergi-center. We can look forward to more fragmented care as these various settings compete with each other in providing health care service to the sometimes bewildered patient.

The geographic shifting of health care services has been nurtured and accelerated by fiscal incentives. There is a powerful and competitive counterforce to produce multihospital complexes in order to provide more service and to take advantage of alleged economies of scale.

NATIONAL HEALTH INSURANCE

When I was a medical student during the late 1940s and early 1950s, I can recall, at the most, two lectures on narcotics and narcotic addiction, plus one reading assignment on the subject in E. S. Goodman and A. Gilman, *The Pharmacological Basis of Therapeutics*. As a junior intern in internal medicine during my senior year, I encountered a single case of adolescent narcotics addiction. This was the sum total of my formal exposure to the subject before my graduation from medical school. This curricular neglect is reminiscent of the ancient philosophic conundrum: If a tree falls in the forest devoid of animals and humans (that is, devoid of creatures who can hear), does the tree in fact make the sound of a tree falling?

Analogously some thousands of people become addicted to narcotics each month. Is there a sound emitted only when white people are afflicted? During the 1940s and 1950s, as long as the narcotics problem was perceived as afflicting primarily Blacks and Hispanics, and not afflicting whites, there was no operative public health problem to address. Accordingly it has become almost a *sine qua non* that the white middle class must become afflicted with a particular problem in order for that problem to become recognized. Therefore if a substantial portion of the middle class defaults on payment of health care bills, then clearly something will have to be done; the government will have to intervene.

Each year for the past 15 years, students at American schools of public health have been reassuringly told that the passage of National Health Insurance (NHI) is imminent. To date, however, NHI is yet to be enacted. At the risk of straining credibility further, I would reiterate that NHI will yet be enacted within the next 15 years. The immediate impetus for NHI enactment will be the agonizing financial suffering of the hitherto comfortable white middle class.

So much for predicting.

REFERENCES

1. Davis K: Cost containment and health care in the United States, in Schnall DJ, Figliola CL (eds): *Contemporary Issues in Health Care*, New York, Praeger, 1984, pp 37-59.

2. Bellin LE: The politics of ambulatory care, in Pascarelli EF (ed): *Hospital-Based Ambulatory Care*, Norwalk, Conn, Appleton-Century-Crofts, 1982, pp 95-109.

Future patterns of chronic disease incidence, disability, and mortality among the elderly

Implications for the demand for acute and long-term health care

KENNETH G. MANTON, PhD

Projecting changes in health status into the 21st century is a difficult task. This is a historically unique period since the risks of certain chronic degenerative diseases have declined significantly, producing life expectancy increases at advanced ages. From 1968 to 1978 the number of deaths caused by stroke fell 32%—and 44% by 1984—while heart disease mortality dropped 23%.[1] From 1959 to 1982 white male life expectancy at age 65 increased 13% (from 12.97[2] to 14.60 years[3]) while for white women the increase above age 65 was 19% (from 15.9 to 18.9 years). Not only were mortality declines at advanced ages not anticipated, but many theorists argued persuasively why such declines should not occur. For example, Omran's[4] theory of the epidemiologic transition does not envision significant improvements in mortality due to chronic disease. Some authors argue that the essential nature of modern society elevates the risks of these conditions.[4]

Mortality rates for the extreme elderly (those aged 85 and over) have also declined. White male life expectancy at age 85 increased 0.88 years (20%) to 5.22 years from 1959-61 to 1982; white female life expectancy increased 1.93 years (41%) to 6.59 years. Such increases in life expectancy at extreme ages have necessitated a reassessment of the biologic limits of life expectancy and consideration of the feasibility of intervention in human physiologic aging. Analyses conducted by the Social Security Actuaries[5] suggest that the effective life span in the United States (ie, the age to which one in 100,000 people from a birth cohort can expect to survive) has increased approximately eight years from 1900 to 1980. Analyses of the age-at-death distribution show little truncation in the standard deviation of ages at death among the oldest 25 percent of deaths from 1962 to 1979.[6] Analyses of the age trajectory of mortality generally show a slower rate of increase beyond age 80—perhaps since individuals who live to that age may be highly selected for survival characteristics; those who have chronic diseases or who smoke cigarettes may die before age 80.[7,8]

Life expectancy in the United States is far from being the highest in the world. The life expectancy at birth for females in Iceland in 1982 was 80.5,[9] and the average life expectancy for Japanese females was 80.2 years.[10] In one prefecture in Japan female life expectancy in 1980 was 81.7 years.[11] At present, nine parts of the world have reported female life expectancies at birth in excess of 79 years (ie, greater than the current estimate of 78.7 years for white females in the United States).[10]

The projected dramatic growth of the population aged 85 and over to 5.2 million by the year 2000 and to 13.1 million by the year 2040[12] may create problems due to the high per capita service needs of this group. Recent increases in life expectancy have increased survival rates at age 85 by 18% for men and 38% for women in 1980[5]; this guarantees large future increases in the demand for health care because of the increasing size of more recent birth cohorts. For example, Rice[13] estimates that the nursing home population will increase 112% by the year 2003 if mortality rates continue to decrease as observed from 1967 to 1976. Even with no further mortality rate decreases, the nursing home population would increase by 57 percent.

Health care projections are also difficult to make because of qualitative changes in the health status of the elderly. Considerable medical progress has been made in controlling chronic disease processes such as the control of osteoporosis and decreasing the risk of hip fracture, although it is too recent to accurately gauge its full effect.[14] Qualitative changes in individual health suggest the need to evaluate health status changes at advanced ages, as well as the effects of medical innovations and lifestyle factors.

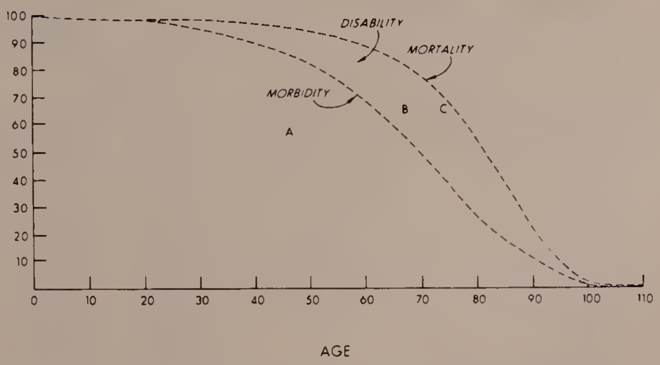


FIGURE 1. Graph illustrating the risk of three age-controlled health outcomes: how the possibility of surviving (**vertical axis**) without one of three health events (disease, disability, death) changes as a function of age.

Address correspondence to Dr Manton, Research Professor, Duke University, Center for Demographic Studies, 2117 Campus Dr, Durham, NC 27706.

Based on a presentation at the symposium entitled, *The U.S. Health Care System into the 21st Century*, January 21, 1985, New York University, New York City.

A model that rationalizes the interaction of such changes was developed by a recent World Health Organization[15] scientific advisory group (Fig 1). This graph can be used to both clarify the complex nature of health changes at advanced ages,[16] and organize information from multiple sources about health processes at advanced ages.

Increases in survival have been observed at later ages that have profound implications for health service demand in the absence of underlying morbidity and disability changes. The growth of the noninstitutionalized disabled, elderly population from 1980 to 2000 would be 2.29 million (52 percent) if mortality changed as projected by the Social Security Administration (SSA) and age-specific disability rates were fixed at the level observed in the 1982 National Long Term Care Survey (NLTCS).[17] From 2000 to 2040 the increase is projected to be 6.43 million (96%). If improvements in health paralleled reductions in mortality—age-specific disability rates decreasing along with mortality rates—then the increase in the disabled elderly population from 1980 to 2000 may be less than 1 million (23%), and from 2000 to 2040 only 3.75 million (69%). Thus in order to assess the future demand for health services, it is necessary to ascertain how morbidity and disability changes are linked to mortality reductions. Recent trends and possible future changes in morbidity from certain chronic degenerative conditions will be considered.

TABLE I. **Mean Age at Death Expected from Chronic Degenerative Diseases: Underlying Cause for White Men and Women—1968–1980**

	Average age in years			
	White men		White women	
Disease	1968	1980	1968	1980
Septicemia	56.86	73.57	62.32	78.84
Cancer	68.45	70.62	69.28	71.22
Leukemia	62.47	67.46	66.12	71.07
Diabetes	69.83	72.38	74.32	76.58
Multiple sclerosis	55.29	57.04	55.10	59.06
Heart disease	71.70	74.32	79.75	82.14
Stroke	76.15	78.35	80.83	83.15
Influenza and pneumonia	70.45	79.29	77.88	84.61
Chronic obstructive pulmonary disease	70.63	74.67	70.49	74.42
Cirrhosis	58.67	60.38	59.36	62.48
Nephritis and nephrosis	66.55	76.78	71.71	79.97

MORBIDITY AND DISABILITY AT ADVANCED AGES

The prevalence of multiple chronic conditions tends to increase with age, and the complexity and possible interaction of these conditions raises several issues. One issue is whether explicit pathologic changes decrease in significance as causes of death at advanced ages—mortality as a result of biological senescence. Scientific evidence on the existence of natural death is inconclusive with data showing the existence of extensive chronic disease at advanced ages. In an autopsy series of 90- and 100-year-old decedents many distinct pathologic changes are noted.[18] It often appears that resistance to mortality from explicit pathologic changes decreases with age, resulting in a decrease of the "lethal threshold" for a specific disease. However, any reduction in threshold may be due to the interaction and cumulative impact of multiple chronic conditions. For example, in an autopsy series of extreme elderly patients, extensive circulatory degeneration is often noted. If such degeneration were reduced, the body's capacity to preserve homeostasis might be improved. Recent evidence on circulatory function among the extreme elderly has found that healthy 80-year-old individuals maintained cardiac output at the level of much younger subjects.[19] Thus, degeneration in circulatory function may be related to explicit pathologic changes.

If age-related functional decline is due to pathologic processes of the type observed at younger ages, there may be considerable potential for intervention at younger ages. Recent evidence suggests that certain risk factors change in their relation to disease risk at advanced age.[20] However, this change may once again be due to the interaction of multiple disease processes at advanced ages. It has also been suggested that the boundary between premature death (due to disease or accident) and natural death (due to senescent frailty) may be artificial as the nature of age-related physiologic change is investigated.[21]

TABLE II. **Proportion of Deaths Expected from Chronic Degenerative Diseases: Underlying Cause for White Men and Women—1968 and 1980**

	White men						White women					
	All Ages		Age 65+		Age 85+		All Ages		Age 65+		Age 85+	
Disease	1968	1980	1968	1980	1968	1980	1968	1980	1968	1980	1968	1980
Septicemia	0.12	0.14	0.10	0.45	0.11	0.52	0.12	0.48	0.09	0.49	0.07	0.49
Cancer	14.62	19.00	14.27	18.55	7.86	11.01	13.87	16.60	11.05	13.59	5.77	7.13
Leukemia	1.65	1.88	1.37	1.70	0.75	1.12	1.46	1.73	1.15	1.49	0.49	0.78
Diabetes	1.48	1.32	1.56	1.38	1.13	1.12	2.42	1.96	2.45	1.96	1.29	1.37
Multiple sclerosis	0.06	0.05	0.02	0.02	0	0	0.09	0.08	0.03	0.03	0	0
Heart disease	42.42	41.14	45.92	44.41	46.61	47.09	42.00	42.59	46.51	46.65	48.29	50.38
Stroke	9.64	7.28	12.40	8.94	16.49	11.71	15.18	12.16	17.18	13.55	19.43	15.71
Influenza and pneumonia	3.64	2.96	4.12	3.63	6.46	6.28	4.10	3.58	4.31	3.99	5.96	5.55
Chronic obstructive pulmonary disease	2.65	4.04	2.96	4.74	1.37	3.11	0.74	1.83	0.63	1.75	0.40	0.91
Cirrhosis	1.55	1.53	0.71	0.79	0.14	0.17	0.88	0.85	0.36	0.44	0.09	0.09
Nephritis and nephrosis	0.41	0.81	0.37	0.96	0.40	1.30	0.38	0.81	0.34	0.85	0.31	0.86

TABLE III. **Number and Percent of Persons with Condition, With and Without Limitation, and Mean Medical Charges ($)**

Age group	Number of persons	% Persons with condition	Total persons with condition	Persons with condition and limited		Persons with condition and not limited	
				Number	$	Number	$
Cancer							
Birth–44	155,826	0.39	608	95	*	513	1,715
45-54	22,443	1.67	338	*	*	338	1,847
55-64	21,135	4.32	913	196	*	717	3,305
65-74	15,165	5.63	854	432	9,117	422	3,438
75+	8,305	7.50	623	381	7,039	242	4,285
Stroke							
Birth–44	155,826	0.16	252	101	*	151	*
45-54	22,443	0.67	105	105	*	*	*
55-64	21,135	2.03	535	303	3,688	127	*
65-74	15,165	4.39	666	502	3,012	164	*
75+	8,305	8.02	666	481	7,258	185	*
Heart disease							
Birth–44	155,826	0.19	248	*	*	248	*
45-54	22,443	1.92	432	97	*	335	2,234
55-64	21,135	5.04	1,064	471	4,873	593	2,841
65-74	15,165	6.01	911	524	4,319	387	1,805
75+	8,305	8.36	694	457	6,755	237	*
Upper respiratory tract diseases							
Birth–44	155,826	32.46	50,583	1,901	1,588	48,681	465
45-54	22,443	23.20	5,206	304	1,258	4,902	785
55-64	21,135	21.90	4,629	675	2,128	3,954	802
65-74	15,165	20.05	3,040	944	2,386	2,096	1,107
75+	8,305	17.57	1,459	695	3,249	764	880
Blood disease							
Birth–44	155,826	1.10	1,712	79	*	1,633	623
45-54	22,443	1.64	323	*	*	323	1,749
55-64	21,135	1.51	319	95	*	224	*
65-74	15,165	2.40	365	181	*	184	*
75+	8,305	5.35	444	279	6,044	165	*
Hypertensive diseases							
Birth–44	155,826	2.12	3,309	357	3,354	2,952	795
45-54	22,443	17.37	3,897	400	1,842	3,498	833
55-64	21,135	27.85	5,888	1,230	2,582	4,658	964
65-74	15,165	36.30	5,505	2,197	2,109	3,308	1,147
75+	8,305	37.70	3,131	1,523	3,609	1,608	1,277

* Unstable estimate suppressed.
Reproduced from the *National Medical Care Utilization and Expenditure Survey* 1980; and the Research Triangle Institute, Center for Survey Statistics, 1984.

An empirical analysis of these concepts is difficult to make since nationally representative data on health trends at advanced ages are relatively rare. One extensive data source is the National Cause of Death statistics. In 1980 there were approximately 350,000 deaths over age 85 in the United States.

The Cause Elimination Life table examines mortality trends at advanced ages by providing estimates of the gain in life expectancy from the elimination of a specific cause of death. This gain can be divided into two components: the mean age at death and the proportion of deaths expected due to a certain condition. Mean ages at death for white men and women between 1968 and 1980 for certain chronic degenerative diseases are contained in Table I. The mean age at death for all conditions increased significantly from 1968 to 1980. Thus while the relative significance of different chronic conditions changed, an upward shift of two or more years in the mean age at death from each disease occurred.[22]

The second component of mortality trends—the proportion of deaths expected due to the condition—is contained in Table II. Of the diseases studied, leukemia, cancer, septicemia, chronic obstructive lung disease, and nephritis and nephrosis showed increases in the proportion of deaths they caused. Stroke, influenza and pneumonia, and diabetes declined. Heart disease, despite significant declines in mortality from 1968 to 1980, exhibits either little change or a slight increase at later ages in the proportion of deaths affected.

A comparison of this measure over age indicates whether a condition is concentrated at later ages. For certain chronic diseases such as diabetes, the disease declined in significance with increasing age. Individuals with these conditions have a lower life expectancy than the general population and usually die by these extreme ages. This is consistent with the age-specific prevalence of diabetes that also declines at advanced ages.[23] Other diseases (eg, stroke, hip fracture) increase with age in the proportion of deaths affected.

Mortality statistics are useful because they are based on large numbers and are available for a long-time series. These statistics take on added significance since recent evidence shows Medicare expenditures to be heavily concentrated in the final year of life[24] and vary by disease. However, to understand the nature of the linkage of health outcomes in Figure 2 and their implications for health expenditures, data from the National Medical Care Utilization and Expenditure Survey (NMCUES) shown in Table III should be considered.

The prevalence of each specific condition increases with age as does the proportion of persons with the condition who report limitations (except for upper respiratory diseases). In addition, expenditures are higher when the individual reports limitations. Thus intervention should be directed at two levels. First, although prevention of the onset of chronic disease is desirable it may not be as feasible as delaying the age at onset of the condition, especially for chronic diseases strongly associated with age-related physiologic changes. Secondly, we are aware that some interventions may increase the prevalence of chronic conditions. Individuals with certain chronic diseases may survive longer, thereby increasing the number of person-years spent in a morbid state. By living to ages with higher incidence rates, the risk of acquiring a chronic condition increases.[25] For example, the prevalence of diabetes increased 24% from 1973 to 1980; this was perhaps a result of reduced stroke mortality risks since diabetes is a risk factor associated with stroke.[26] Therefore, an increasingly important aspect of future health care will be the reduction of disability resulting from the progression of chronic degenerative processes. Functional assessment and the provision of care to maintain and improve individual functioning is a primary goal of geriatric medicine.[27]

The implications of intervention in the natural history

TABLE IV. Projections of the Noninstitutionalized Long-Term Care and Institutionalized Populations by Age, Sex, and Marital Status Assuming Constant and Decreasing Disability Rates for 1980, 2000, and 2040

	Constant disability rate				Decreasing disability rate			
	Noninstitutionalized	Institutionalized	Noninstitutionalized	Institutionalized	Noninstitutionalized	Institutionalized	Noninstitutionalized	Institutionalized
Year	Married men		Married women		Married men		Married women	
Age 65–74								
1980	600	22	472	20	600	22	472	20
2000	708	25	579	25	598	21	481	21
2040	1,103	40	995	41	817	29	682	29
Age 75–84								
1980	385	39	248	29	385	39	248	29
2000	666	67	491	58	569	57	373	44
2040	1,309	131	1,031	120	986	98	657	734
Age 85+								
1980	118	24	46	14	118	24	46	14
2000	253	51	82	24	207	41	56	16
2040	671	137	230	68	485	95	131	38
Age 65+*								
1980	1,102	84	767	63	1,102	84	767	63
2000	1,627	143	1,152	107	1,374	119	910	81
2040	3,093	307	2,198	229	2,288	223	1,471	144
Year	Unmarried men		Unmarried women		Unmarried men		Unmarried women	
Age 65–74								
1980	165	65	653	118	165	65	653	118
2000	219	86	696	126	186	73	579	105
2040	425	167	1,037	188	319	124	746	134
Age 75–84								
1980	164	90	922	339	164	90	922	339
2000	254	139	1,355	498	219	119	895	376
2040	602	331	2,458	903	463	248	1,636	579
Age 85+								
1980	124	77	530	407	124	77	530	407
2000	211	132	1,203	924	178	106	928	626
2040	557	347	2,779	2,133	597	242	1,840	1,187
Age 65+*								
1980	453	232	2,105	864	453	232	2,105	864
2000	683	357	3,255	1,547	584	298	2,558	1,107
2040	1,583	845	6,275	3,223	1,200	613	4,221	1,901

* Totals may reflect rounding error.
Reproduced from the National Long-Term Care Survey.

TABLE V. Projections of Total Excess Hours Per Week Spent In Providing Informal Care for Disabled Elderly by Sex and Marital Status Assuming Constant and Decreasing Disability Rates for 1980, 2000, and 2040

	Constant rate				Decreasing rate			
	Married men	Married women	Unmarried men	Unmarried women	Married men	Married women	Unmarried men	Unmarried women
Spouse is helper								
1980	25,588	14,529	—	—	25,588	14,529	—	—
2000	37,649	21,694	—	—	31,791	17,205	—	—
2040	71,435	40,975	—	—	52,844	27,548	—	—
Offspring is helper								
1980	4,321	3,565	4,015	23,665	4,321	3,565	4,015	23,665
2000	6,586	5,686	6,256	38,885	5,540	4,395	5,347	30,399
2040	13,133	11,665	15,056	77,518	9,651	7,565	11,402	51,912
Relative is helper								
1980	1,425	2,113	4,933	16,573	1,425	2,113	4,933	16,573
2000	2,179	3,249	7,380	25,605	1,833	2,577	6,314	20,116
2040	4,364	6,581	16,919	49,306	3,206	4,291	12,826	33,170
Nonrelative is helper								
1980	1,489	1,621	1,703	6,433	1,489	1,621	1,703	6,433
2000	2,327	2,565	2,593	10,562	1,962	2,001	2,214	8,268
2040	4,645	5,088	6,078	21,088	3,427	3,348	4,596	14,137

Reproduced from the National Long-Term Care and NORC surveys.

of a chronic disease process to delay the onset of limitations are shown in Table IV.[16] The 1982 NLTCS and the 1977 National Nursing Home Survey were used to project the long-term care population in 2000 and 2040. Population projections made by the SSA were multiplied by age, sex, marital status (an important factor in determining the availability of informal care and nursing home utilization), and disability specific rates from NLTCS after adjusting the base population for age, sex, and marital status specific institutionalization rates. Individuals aged 65 to 84 with serious disability (ie, three or more limitations in daily activity[28]) or in nursing homes grew by 64% from 1980 to 2000 and by 106% from 2000 to 2040. Even more dramatic is the 140% increase of those aged 85+ from 2000 to 2040; the interval during which those of the post-World War II "baby boom" reach age 85. The growth in the nursing home population was 2.2 percent per year over that period.

The effects of reducing disability rates as fast as mortality rates are also illustrated in Table IV. This intervention effectively reduces the annual nursing home population growth rate by 37% (from 2.2 to 1.4%) and the total number of noninstitutionalized seriously disabled persons by 19.4 percent in 2000 (30.5% in 2040). Projections of the amount of long-term care needed by the disabled elderly community requires the translation of the number of disabled persons into a measure of service. This was achieved by using data from an NORC survey on informal caregivers to estimate the number of excess hours of care delivered per day by different types of caregivers, and applying those estimates to the projections in Table IV. These estimates and projections of the number of excess hours spent providing informal care services are seen in Table V.[16] These estimates were generated by multiplying the number of hours of care provided by informal caregivers to persons of different age, sex, and marital status groups. The effect of assuming that disability rates would decline as fast as mortality rates is also illustrated. The total number of hours of care needed by the seriously disabled elderly, noninstitutionalized population—though approximately 110 million hours per week in 1980—can be reduced significantly by 19.2% in the year 2000 and 30.2% in the year 2040 if the disability can be delayed.

FACTORS AFFECTING FUTURE HEALTH TRENDS

In order to project future morbidity and disability prevalence rates, the epidemiologic and technologic factors shaping future changes in the health of the elderly population should be evaluated. Several chronic conditions that have significant effects on either acute or chronic health care are discussed here.

Stroke. Contrary to expectations that modern lifestyles would enhance the risk of chronic diseases, stroke mortality has declined 44% from 1968 to 1984. Despite its decline, stroke is still a major factor in mortality at advanced age; it is associated with more than 25% of white female mortality past age 85 (Table II). Thus it is important to understand the cause of past changes in stroke mortality to determine if further improvements are likely.

Between 1968 and 1980, the effect of stroke on life expectancy dropped 23% for white men (from 1.55 to 1.19 years) and 22% for white women (from 2.46 to 1.92 years). Considerable debate exists over the factors causing this decline; control of hypertension appears to be an important aspect.[29] However, 80% of the clinical management of hypertension has been instituted since 1974. Since national programs to control hypertension were only initiated in 1972,[30] it has been argued that reductions in stroke mortality prior to 1972 were caused by better management of stroke patients.[31] If true, this would imply an increasing prevalence of stroke-disabled, chronic care patients through 1972. After 1972, the control of hypertension may have resulted in reduced stroke incidence.[32]

Future reductions in stroke incidence may become difficult with the initiation of more effective control of hypertension. There are also signs that further reductions are possible. For example, although the risk of stroke is only

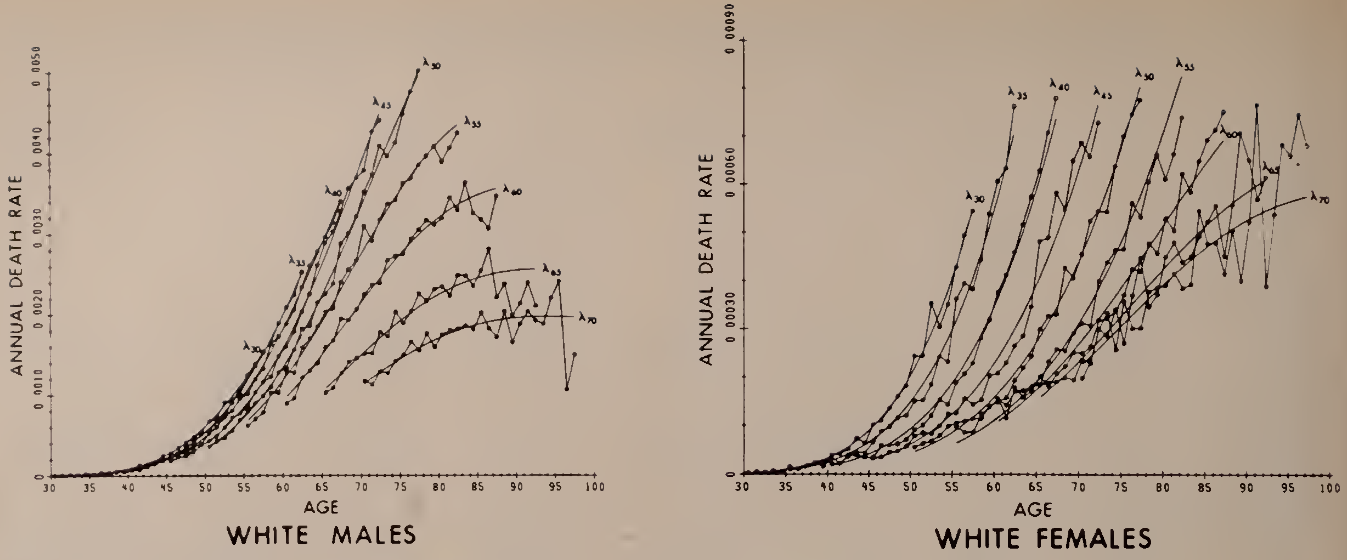


FIGURE 2. Graphs projecting lung cancer mortality and morbidity to year 2000 for men **(left)** and women **(right)**.

moderately elevated with borderline hypertension, the large size of the population with borderline hypertension produces a large number of strokes. Hence, though clinical treatment of borderline hypertension may not justify an aggressive clinical approach, control of this condition through nutritional and lifestyle modification may significantly reduce stroke mortality. A second intervention currently under study is the control of isolated systolic hypertension among the elderly. If such intervention is successful, significant improvement in life expectancy at advanced ages may result.

Two factors seldom addressed when forecasting future health trends are the differential risk of disease for cohorts, and the high prevalence of multiple chronic conditions at advanced ages. Strong cohort differentials in risk exist for a number of major chronic diseases (eg, cancer, stroke, heart disease) in the United States and several other countries.[16] Therefore, early life experiences in cohorts appear to be important determinants of the level of cohort risks. The interdependence of chronic conditions at advanced ages results because of either the interaction of the disease processes, or their mutual dependence on common risk factors. One reason for the decline of stroke mortality is that certain lethal events—such as chronic heart disease and pneumonia—associated with stroke morbidity have decreased.[33] Another source of dependency is the association of stroke with diabetes. The decline in stroke mortality is thought to be responsible for the 24% increase in the prevalence of diabetes after 1973.[1] The control of hypertension will also cause simultaneous reductions in heart disease and stroke mortality. Projection of future health status trends will require consideration of the interdependence of multiple chronic conditions.

If stroke morbidity and mortality continue to decline, the prevalence of stroke-related disability and its contribution to the institutional care population will also decline. At present 8% of nursing home days are spent on persons with a primary diagnosis of stroke.[34] With this decline increases in the prevalence of clinically controlled hypertension, diabetes, and disability associated with those conditions can be expected. This may change the relative share of different types of medical expenditures, such as higher expenses on ambulatory care and lower expenses on hospital and nursing home care.

Heart disease. Heart disease has many of the same risk factors as stroke and also exhibits large declines in mortality. This condition is often lethal, so its prevalence as a condition with associated disability is relatively small as compared to hypertension (Table III). Heart disease is associated with severe disability less often than stroke. Consequently, the decline in stroke mortality is most likely related to declines in disability.

Heart disease, like stroke, manifests strong cohort trends[16,35] and tends to increase in significance as a cause of death at later ages (Table II). Its role as an associated cause of death at advanced ages has increased 21% between 1968 to 1980, suggesting that cardiovascular morbidity is a major contributor to mortality from many other causes at advanced ages.

Cancer. This is a heterogeneous group of diseases with different underlying trends. Stomach cancer mortality is rapidly declining while lung cancer mortality has increased dramatically for both men and women—most likely influenced by the early smoking habits at ages 18 to 30.[36] Breast cancer risk appears to be increasing apparently in part from declining fertility and because of the negative association of breast cancer with the age at first pregnancy.[37] The net impact of these differing trends has been an increase in overall cancer risk.

Cancer represents an instructive group of diseases to examine in detail because a biologically motivated model of human carcinogenesis provides an adequate description of age-specific incidence[38] and detailed cancer mortality data dating from 1950 permit the examination of cohort patterns. Such analyses make investigation of the relationship between disease incidence, progression, and mortality possible.

Manton[39] projected lung cancer morbidity and mortal-

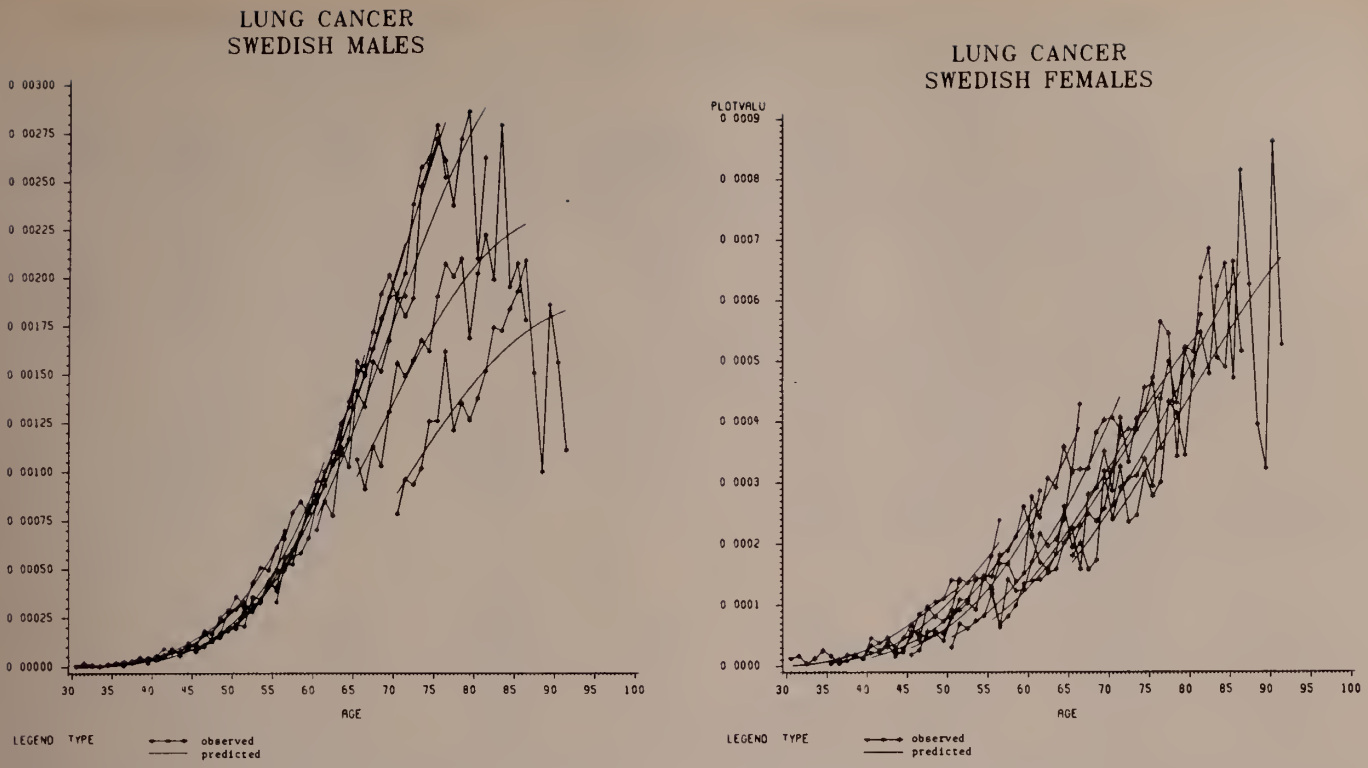


FIGURE 3. Swedish male (**left**) and female (**right**) cohort risks for lung cancer as compared to cigarette consumption.

ity to the year 2000 based on the cohort mortality from 1950 to 1977. These projections are presented in Figure 2.

Although cohorts born in 1910 or earlier showed increasing mortality risks, younger male cohorts appear to have stabilized at very high levels. This reflects the fact that cohorts with the highest level of tobacco consumption are now at the age for peak lung cancer mortality risks.[40] For women, there are large increases in the risk of lung cancer in younger cohorts. This pattern reflects the fact that female cigarette consumption started to decline 20 years later than for males. These patterns may be compared with Swedish male and female cohort risks seen in Figure 3.[41] The Swedish patterns follow very different male and female cohort patterns in tobacco consumption.[42] The peak levels of male lung cancer risk are much lower in Sweden than in the United States.

Three insights help to explain the cohort differences in lung cancer risk. The relation of the level of lung cancer risk in specific cohorts to the proportion of regular smokers in those cohorts was examined.[43] Approximately 90% of the increase in cohort lung cancer risk may be attributed to the increase in smokers over cohorts. Per capita smoking rates were then computed by state for each year from 1950 to 1977, as the ratio of total annual pack sales in the state to the total population ten years or older. The relationship of the state cohort lung cancer mortality rates (for nine cohorts for 49 states for 28 years) with state per capita smoking rates (for 49 states for 28 years) was also examined. The correlation over time was consistent with the hypothesis that the state-specific cohort risks were strongly associated with state-specific smoking levels. The implications for reducing lung cancer risk by reducing smoking levels to the lower levels observed for older cohorts was studied.[44] Approximately 80% of disease costs could be eliminated in such simulations.

The results of the projections for US males are illustrated in Table VI. These projections provide quantitative evidence of chronic disease morbidity and mortality risks at advanced ages. First, although approximately 60,000 persons died from lung cancer in 1977, 66,000 persons were estimated to die of another condition, but with tumor growth. Thus there is considerable latent disease that could be made manifest with a significant reduction in mortality from other causes. Secondly, the future growth in disease prevalence due to demographic factors can be isolated from changes in disease risk. Given the 1977 population structure, a 25% decline in lung cancer mortality may occur by the year 2000 because the most recent male cohorts appear to have lower lung cancer risks that reflect recent declines in smoking.[40] The decline may only be partially apparent because the very early lung cancer risks of the most recent birth cohorts are high, and because the decline is based on the smaller number of cases observed in the youngest cohort. It is primarily at the later ages, where mortality has removed the heaviest smokers, that there is evidence of stability or decline in risk. Projections for women, however, show large increases in mortality.

Above age 85 a large increase in lung cancer morbidity and mortality is seen (Table VI). This occurs because lung cancer risk appears to peak for the 1910 cohort. Introduction of population aging in the projections produces an increase in lung cancer mortality and prevalence due to the large size of the high-risk cohorts in the year 2000. The lower panel in Table VI shows the net balance of declining cohort risk and an aging population. Even with success in controlling smoking, there may be large in-

TABLE VI. Estimates and Projections of Male Lung Cancer Morbidity and Mortality Derived Assuming Independent Competing Risks

					---------Predicted Dead---------			
Age group	Observed population	Disease-free population	Annual morbidity incidence	Prevalence of morbid population	From other cause and no cancer	From other cause with cancer	From cancer	Observed dead from cancer
Observed and predicted cancer morbidity and mortality conditions in 1977								
Total	93806538	91757465	141508	2049073	876851	66359	59784	60773
0-44	65794126	65584483	29416	209643	174335	624	1124	1369
45-64	19303180	18367798	63002	935383	217478	13127	23669	24498
65-84	8118528	7253858	47258	864670	388207	45946	32969	33075
85-97	590704	551326	1833	39377	96831	6661	2021	1831
Cancer morbidity and mortality conditions in 2000 given the 1977 population structure								
Total	93806538	92230234	108163	1576304	880946	48887	51092	—
0-44	65794126	65584483	29416	209643	174335	624	1124	—
45-64	19303180	18482002	53616	821178	216257	10536	22409	—
65-84	8118528	7640080	22155	478448	400886	26050	23900	—
85-97	590704	523669	2976	67035	89468	11677	3658	—
Predicted cancer morbidity and mortality conditions in 2000								
	Projected population							
Total	99378460	97375177	139904	2003283	1115242	66397	63519	—
0-44	64321733	64007504	41488	314229	184565	991	1876	—
45-64	23807045	22832609	65660	974436	249764	11765	25145	—
65-84	10348346	9736699	28188	611646	541748	35313	30875	—
85-97	901337	798365	4568	102972	139164	18328	5623	—

creases in lung cancer mortality because of the larger size of more recent cohorts.

For stroke and heart disease, the implication of dependent competing risks was discussed. The effect of dependent competing risks in the lung cancer projections was examined since smoking is a risk factor both for lung cancer and for many other diseases. Thus the mortality for other diseases will be correlated with lung cancer mortality. This was simulated by assuming that most people with lung cancer were smokers, and that heavy smokers had twice the mortality risk of other conditions. The primary effect is to increase the estimated and projected numbers of latent lung cancer cases.

Cancer trends primarily affect acute health services though recently there has been a development of specialized terminal and hospice care. Chronic respiratory diseases have been rapidly increasing in prevalence for men, parallel to the increase in lung cancer. Because of the long survival time for these conditions, they may contribute significantly to the demand for long-term care.

Diabetes and hypertension. More than five million persons have diabetes, and more than 22 million persons have hypertension according to the NMCUES survey. These chronic conditions are important risk factors for stroke and heart disease, and effective medical treatments are available for both. One effect of these treatments may be to raise the prevalence of these conditions in the population by reducing mortality from associated lethal conditions. In order to make this assumption, two factors must be considered: the quality of life of individuals with these conditions; and preventing individuals with these conditions from developing a serious disability.

Hip fracture. This condition is common at advanced ages, and especially in women. Mortality associated with hip fracture is, however, declining, most likely due to better management (Table II). This condition generated 7% of nursing home bed-days in 1977.[45] Brody[14] estimated that approximately 200,000 persons had hip fractures in 1980. Because of the growth in the population of individuals age 85 or older, this number is expected to increase rapidly—reaching 330,600 in the year 2000, and 650,000 in 2050. Therefore, as circulatory disease mortality declines, hip fracture is likely to become an even greater factor in the demand for acute care and long-term care services. The projected growth of the incidence of hip fracture is seen in Figure 4.[14]

Progress has recently been made in developing treatments for osteoporosis—the degenerative process underlying the risk of hip fracture at later ages. Since the risk of hip fracture rises exponentially with age, even a five-year delay in the age at onset could lead to a significant reduction in its occurrence.[14] Because the risk of many chronic diseases increases sharply with age, the effects of even small delays in the age at onset may have considerable impact on disease-specific medical expenditures.

Infectious diseases. With the advent of immunization and antibiotic therapy, infectious diseases are considered well controlled in the United States. However, morbidity due to infections such as pneumonia and septicemia are common among infants, the extreme elderly, and debilitated patients. Currently, the combined mortality from influenza and pneumonia, and septicemia has not increased significantly, even among the extreme elderly. With the rapid growth of the extreme elderly population and often uncontrolled use of antibiotics, this situation may change. The recent increases in septicemia mortality, however, are disturbing.[46]

Alzheimer's disease and senile dementia. The greatest future health problem in the United States may be caused by senile dementia and Alzheimer's disease since these

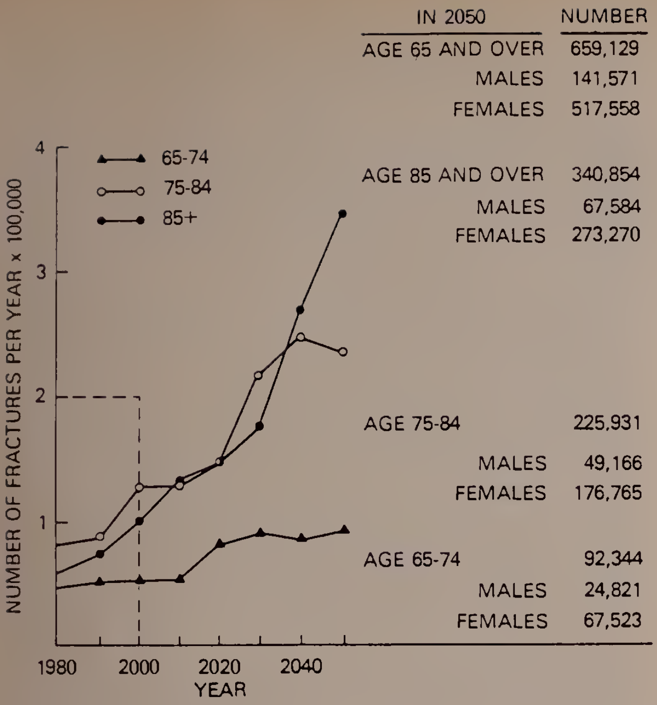


FIGURE 4. Projected growth of the incidence of hip fracture. (Reproduced from [14] with permission.)

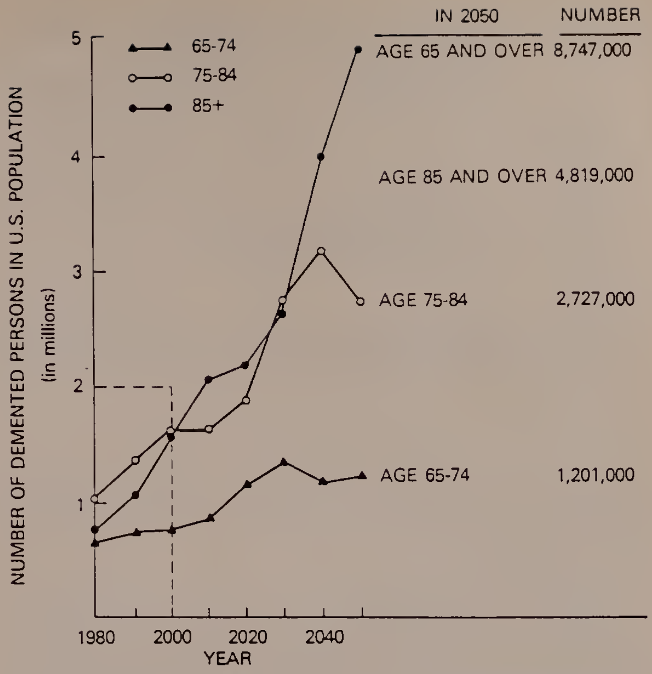


FIGURE 5. Projected prevalence of Alzheimer's disease. (Reproduced from [14] with permission.)

conditions are profoundly debilitating, currently not treatable, and have a high rate of occurrence among the extreme elderly (ie, 3% increase per year past 80 years of age[47]). The magnitude of the problem has not been noticed since Alzheimer's disease is not often recorded as a cause of death. Katzman[48] estimated that 90,000 deaths in 1970 were associated with Alzheimer's disease. Brody[14] projected the prevalence of Alzheimer's disease to be 2 million in 1980 and 3.8 million in the year 2000 (Fig 5). Dementia requires chronic care management both in the institutional setting (20.4% of nursing home residents have mental disorders and senility[34]) and in the community.[16]

In order to project the future impact of senile dementia, the effect of intervention must be addressed. Up to one third of senile dementia cases are estimated to be due to treatable causes (eg, microcerebral infarcts). Consequently, more detailed projections by dementia type are needed.

Renal dysfunction. Renal conditions have received much attention because of the federal funding of dialysis. Projections of the costs of End-Stage Renal Disease (ESRD) programs have been previously underestimated. Recent projections by Eggers et al[49] are provided in Table VII. The number of individuals with ESRD is projected to grow far more rapidly than the general population (ie, 180% versus 35%). This is not a result of population aging since the most rapid growth is at younger ages.

Two issues to be considered when projecting the demand of ESRD on future health services are the availability of organs for transplant, and the ability of the individual to cope with organ rejection. The development of the

TABLE VII. Projected Medicare ESRD Program Enrollment by Age, Sex, and Race—1980 to 2030

	1980	1990	2000	2010	2020	2030	Percent change in ESRD population 1980–2030	Percent change in US population
All persons	57,800	94,400	117,200	136,700	152,800	162,100	180	35
Average annual percent increase	—	50	2.2	1.6	1.1	0.6	—	—
Age								
0–24	4,300	10,800	13,100	14,900	15,700	16,300	279	11
25–44	15,600	34,100	44,400	46,500	47,800	50,600	224	24
45–64	22,900	34,700	43,300	57,700	66,100	65,200	185	53
65+	15,000	14,800	16,400	17,600	23,200	30,000	100	121
Sex								
Male	31,800	51,800	64,600	75,000	83,100	87,700	176	34
Female	26,000	42,600	52,600	61,700	69,700	74,400	186	37
Race								
White	40,600	62,100	72,900	80,800	86,500	88,600	118	28
All other	17,200	32,300	44,300	55,900	66,300	73,500	327	83

Reproduced from Eggers PW et al: The Medicare experience with end-stage renal disease: trends in incidence, prevalence, and survival. In press.

TABLE VIII. Life Expectancies at Ages 30 and 45 Years for Men Derived from Stochastic Process Models Applied to Data from the Framingham Study

Total mortality	Age 30	Age 45
Total	45.33	32.64
Nonsmokers	47.84	34.86
Smokers	43.45	31.04
All men with cardiovascular disease mortality eliminated under a dependent competing risk model	48.24	35.57

Reproduced with permission from Manton KG, Stallard E, Woodbury MA: Chronic disease evolution and human aging: a general model for assessing the impact of chronic disease in human populations. *International Journal of Mathematical Modeling*, in press.

drug cyclosporine has created a whole range of transplant possibilities. Thus future projections of health changes must take into account the increasing ability to control tissue rejection.

Dependent competing risks. In order to assess the effect of many chronic diseases at advanced ages, a satisfactory model of disease interactions must be created. One model, as seen in the case of lung cancer, describes disease dependency as an elevated relative risk for other causes of death. A second model describes disease interdependence through an individual's risk factor profile. The effects of health interventions can be represented in the second model by adjusting the risk factor distribution of the population. In Table VIII the mortality risk of heart disease, and the effects of smoking on total mortality are described by a time series model of risk factor changes adjusted for mortality selection.[50]

Table VIII contains the age-specific (ages 30 to 45) life expectancies estimated for men in the Framingham study under certain assumptions. The calculations for the elimination of heart disease are based on the assumption that diseases are independent only for persons with the same risk factor levels. Thus, for example, the effects of smoking that include increased risk of cardiovascular disease are assumed to exist for other diseases, even when cardiovascular disease is eliminated. Total mortality estimates are also separated by smoking status. For the study population, a large difference in life expectancy may be seen between persons who smoke and those who do not. A comparison of all men with nonsmoking men shows a potential for health improvement with smoking cessation.

THE FUTURE COSTS OF DISEASE AND DISABILITY

Forecasts of the growth of American health care expenditures often do not involve assumptions about future changes in the risks of specific diseases or disabilities. For example, Freeland and Schendler[51] projected health care expenditures to 1990 by assuming that disease rates would not change. With the introduction of Prospective Payment in Medicare and Diagnosis Related Groups (DRGs), future health care costs will be directly related to the prevalence of specific diseases. Thus, to project future health care costs, future changes in disease risks must be anticipated.

Projecting health care costs at advanced ages is difficult because of both the multiplicity of chronic conditions affecting elderly persons, and the impact of serious disability on long-term care costs. Such factors are not well described within the current DRG structure. Even more difficult is the development of a price system for long-term care. While acute care costs may be logically organized with regard to medical diagnosis, long-term care involves many social and economic factors. This suggests the need for an alternate mechanism to the discrete categories of DRGs in order to organize the price structure.[52] One approach is known as the Grade of Membership model.[53] This is a system that categorizes cases using both discrete and continuous elements. The continuous elements describe individual differences in costs as a set of "blending" weights while the discrete elements represent the basic dimensions over which price varies.[54]

Future health care expenditures will be modified by the change in prevalence of specific diseases and disability. Better models are necessary to project disability and morbidity. These projections must describe not only the risks of changes in health status, but also a complex multidimensional health profile for very elderly persons. With such models, information on disease risks and projections from multiple data sources can be organized and integrated into economic forecasts of health expenditures.

Acknowledgment. The research reported herein was supported by NIA grant no. AG-01159 and HCFA grant no. 18-P-97710/4-0452.

REFERENCES

1. Advance Report, Final Mortality Statistics, 1981, *Monthly Vital Statistics Report* DHHS Publication No. (PHS) 84-1120. Hyattsville, MD, National Center for Health Statistics, 1984.
2. Vital Statistics of the United States, DHHS Publication No. (PHS) 84-1104. National Center for Health Statistics, 1984.
3. Current Population Reports, *Projections of the Population of the United States by Age, Sex, and Race: 1983 to 2080* Series P-25, No. 952. United States Bureau of the Census, 1984.
4. Omran AR: The epidemiological transition: a theory of the epidemiology of population change. *Milbank Mem Fund Q* 1971; 49:509–538.
5. *Life Tables for the United States: 1900–2050* SSA Publication No. 11-11536, Social Security Administration Office of the Actuary, 1983.
6. Myers GC, Manton KG: Compression of mortality: myth or reality? *Gerontol* 1984; 24:346–353.
7. Manton KG, Stallard E, Vaupel JW: Alternative estimates of the heterogeneity of mortality risks among the aged. *J Am Stat Assoc*, in press.
8. Wilkin JC: Recent trends in the mortality of the aged. *Trans Soc Act 1981* 1982; 33:11–62.
9. World Health Organization: *World Health Statistics Annual 1983*. Geneva, Switzerland, 1983.
10. World Health Organization: *World Health Statistics Annual 1984*. Geneva, Switzerland, 1984.
11. Kuroda T: Comment on IUSSP-NIRA seminar on Biological and Social Correlates of Mortality, Nov 24–27, 1984.
12. *Social Security area population projections, 1984* SSA Publication No. 11-11539, Social Security Administration Office of the Actuary, 1984.
13. Rice DP: Impact and implications of the changing age structure on health status and use of health care. Presented at the Federal Statistics Users' Conference, Washington, DC, May 12, 1980.
14. Brody JA: Decreasing mortality and increasing morbidity in the 20th and 21st centuries, in *Philosophy and Medicine Series*, D. Reidel Publishing Co, 1985, in press.
15. World Health Organization: The uses of epidemiology in the study of the elderly. Technical Report Series 706. Geneva, Switzerland, WHO, 1984.
16. Manton KG, Soldo BJ: Dynamics of health changes in the extreme elderly: new perspectives and evidence. *Milbank Mem Fund Q* 1985; 63:206–285.
17. Manton KG, Liu K: The future growth of the long-term care population: projections based on the 1977 NNHS and the 1982 Long Term Care Survey. Presented at the Hillhaven Foundation Third National Leadership Conference, "Future World of Long Term Care," Washington, DC, Mar 7–9, 1984.
18. Howell TH: Multiple pathology in nonagenarians. *Geriatrics* 1963; 18:899–902.
19. Lakatta EG: Health, disease and cardiovascular aging. Presented at the National Academy of Sciences Institute of Medicine, Committee on Aging, Society, Health and Disease. Washington, DC, July 18–20, 1983.
20. Manton KG, Woodbury MA: A mathematical model of the physiological

dynamics of aging and correlated mortality selection: II. Application to the Duke Longitudinal Study. *J Gerontol* 1983; 38:406–413.

21. Fries JF: The compression of morbidity. *Milbank Mem Fund Q* 1983; 61:397–419.

22. Manton KG, Tolley HD, Poss SS: Life table techniques for multiple cause mortality. *Demography* 1976; 13:541–564.

23. Scott G: Prevalence of chronic conditions of the genitourinary, nervous, endocrine, metabolic, and blood and blood-forming systems and of other selected chronic conditions, United States, 1973, DHEW Publication No. (HRA) 77-1536. Rockville, MD, National Center for Health Statistics, 1977.

24. Lubitz J, Prihoda R: Use and costs of Medicare services in the last years of life, in *Health, United States, 1983.* DHHS Publication No. (PHS) 84-1232. USGPO, 1984.

25. Golini A, Egidi V: Effect of morbidity changes on mortality and population size and structure, in Vallin J, Pollard JH, Heligman L (eds): *Methodologies for the Collection and Analysis of Mortality Data* Leige, Belgium, IUSSP 1984, chap 17.

26. Baum HM, Manton KG: Cerebrovascular disease mortality: the relationship of underlying and associated causes of death. 1985, unpublished data.

27. Minaker KL, Rowe JW: Health and disease among the oldest old: a clinical perspective. *Milbank Mem Fund Q* 1985; 63:324–349.

28. Katz S, Akpom CA, Papsidero JA, Weiss ST: Measuring health status of populations, in Gerg RL (ed): *Health Status Indices.* Chicago, Hospital Research and Educational Trust, 1973, pp 39–52.

29. Whisnant JP: The decline of stroke. *Stroke* 1984; 15:160–168.

30. Golden PM, Wilson RW, Kavet J: Prevention profile, in *Health: United States, 1983.* DHHS Publication No. (PHS) 84-1232, USGPO, 1983.

31. Sherwin RW: Disability due to hypertension: is it preventable? Presented at the Fifth Annual Invitational Symposium on the Elderly and Their Health, Dept. Epidemiology and Preventive Medicine, University of Maryland School of Medicine, Oct 9, 1984.

32. Robins M, Baum HM: The national survey of stroke: incidence. *Stroke* 1981; 12:45–57.

33. Hachinski V: Decreased incidence and mortality of stroke. *Stroke* 1984; 15:376–378.

34. Characteristics of nursing home residents, health status, and care received, *National Nursing Home Survey, US, May–December, 1977.* DHHS Publication No. (PHS) 81-1712, Hyattsville, MD, National Center for Health Statistics, 1981.

35. Patrick CH, Palesch YY, Feinleib M, Brody JA: Sex differences in declining cohort death rates from heart disease. Am J Public Health 1982; 72:161–166.

36. Klebba AJ: Mortality from diseases associated with smoking. DHHS Publication No. (PHS) 82-1854, 1982.

37. MacMahon B, Cole P, Lin TM, et al: Age at first birth and breast cancer. *Bull WHO* 1970; 43:209–221.

38. Armitage P, Doll R: Stochastic models for carcinogenesis, in Neyman J (ed): *Proceedings of the Fourth Berkeley Symposium on Mathematical Statistics and Probability, Biology and Problems of Health.* Berkeley, University of California Press, 1961, vol 4, p 19–38.

39. Manton KG: Forecasting health status changes in an aging US population: assessment of the current status and some proposals. Presented at the Conference on Forecasting in the Social and Natural Sciences sponsored by the National Research Council, Boulder, CO, June 10–13, 1984, and forthcoming in *Proceedings.*

40. Harris JE: Cigarette smoking among successive birth cohorts of men and women in the United States during 1900–80. *JNCI* 1983; 71:473–479.

41. Malker B, Malker H, Manton KG: An analysis of cohort risks for lung cancer: Swedish males and females. 1985, unpublished data.

42. Carstensen J, Wallgren A: Cancerincidens och dödlighet: Lungcancertrend och rökning. Lakartidningen 1983; 80:4980–4981.

43. Manton KG, Stallard E, Riggan WB: Strategies for analyzing ecological health data: methods for the biological risk of individuals. *Statistics in Medicine* 1982; 1:163–181.

44. Tolley HD, Manton KG, Stallard E: Compartment model methods in estimating cancer costs. *Trans Soc Act 1983,* 1984; 35:399–413.

45. Liu K, Manton KG: The characteristics and utilization patterns for admission cohort of nursing home patients. *Gerontol* 1983; 23:92–98.

46. Manton K: Cause specific mortality patterns among the oldest old: multiple cause of death trends 1968–80. *J Gerontol* 1985.

47. Hagnell O, Lanke J, Rorsman B, Ojesjö L: Does the incidence of age psychosis decrease? A prospective, longitudinal study of a complete population investigated during the 25-year period 1947–1972: the Lundby Study. *Neuropsychobiology* 1981; 7:201–211.

48. Katzman R: The prevalence and malignancy of Alzheimer's disease. *Arch Neurol* 1976; 33:217–218.

49. Eggers PW, Connerton R, McMullan M: The Medicare experience with end-stage renal disease: trends in incidence, prevalence, and survival. 1983, unpublished data.

50. Manton KG, Stallard E, Woodbury MA: Chronic disease evolution and human aging: A general model for assessing the impact of chronic disease in human populations. *International Journal of Mathematical Modeling* 1985, in press.

51. Freeland MS, Schendler CE: National health expenditure growth in the 1980's: an aging population, new technologies and increasing competition. *Health Care Financing Review* 1983; 4:1–58.

52. Manton KG, Liu K: A multivariate strategy for developing case mix measures for LTC services: an illustration with data from the 1982 National LTC Survey. 1985, unpublished data.

53. Woodbury MA, Manton KG: A new procedure for analysis of medical classification. *Methods Inf Med* 1982; 21:210–220.

54. Vertrees J, Manton KG: The generation of hospital groups for the adjustment of PPS reimbursement: the Pennsylvania Medicare experience, 1982–1983. 1985, forthcoming in *Medical Care.*

Implications of new medical technology

Roger C. Herdman, MD

In order to anticipate what the coming century may hold for advances in technology, one must first look to the recent past. Biomedical science and the financial, organizational, and social systems that support the resources for prevention, diagnosis, and treatment of disease should be considered. However, a full analysis of that sort could be quite lengthy. A brief reflection is important in deciding whether a major milestone has been reached that would lead to something new, and provide a basis for insights and predictions for the next century. Milestones that initiated major reorientations and expectations in the past have included the development of sanitation, anesthesia, the radiograph, antibiotic agents, and the concept of third party payments.

Another trend became increasingly evident at about the time of the introduction of Medicare and Medicaid—the use of technology in American medicine. The Office of Technology Assessment, an analytic support agency of Congress, uses a broad definition of technology—the drugs, devices, and medical and surgical procedures used for health care, and the organizations and supporting systems within which such care is provided. Data to describe the magnitude, changes, and importance of technology in health care in the recent past are available from analyses of costs of the health system, from studies of physician behavior, and from reports by suppliers of technology. Third party financing, most recently the Medicare and Medicaid programs, has supported the growth of technology. Physician and institutional providers have responded to that growth, and the number of suppliers has increased as a result.

The two major components of the $1 billion per day health cost are hospitals (42%) and physicians (19%). Annual changes in hospital costs may be divided into population or enrollment changes, overall wage and price inflation, inflation in the cost of medical care greater than that of the general inflation rate, and changes in service intensity (the quantity of components that produce a given unit of health care). Depending on whether hospital inflation is measured per patient day, per admission, or per capita, costs have been increasing by 10% to 17% per year and changes in service intensity account for 20% to 50% of this increase. These costs have consistently been greater than general inflation rate and population changes.

During the past decade there have been sporadic efforts to control hospital inflation; with few exceptions these efforts have been unsuccessful. Now, however, a number of changes such as new statutory payment methods for the major payor (Medicare), involvement of major corporate buyers, and competition are affecting the hospital sector. Occupancy, length of stay, and admissions have either leveled off or decreased, and payments are estimated to be at or below the general inflation rate. It may be too early to predict major changes, but financial pressures are causing hospitals to analyze the need and cost implications of improvements, and to focus on fewer types of new technology and more cost-conscious purchasing. Reports suggest that hospitals under tight financial control limit the purchase and use of technology and favor cost-reducing improvements.[1]

This trend might mean fewer facilities as well as less money and may also cause physicians whose costs are less strictly regulated to increase their use of technology. Medicare data for physician costs may be broken down to annual increases caused by fee changes and annual increases due to residual factors. Over the past decade, the residual factors have grown from less than 20% to more than 50% of total annual increases. A large part of this is due to more expensive procedures—presumably technology-driven. Furthermore, between 1975 and 1982, the number of primary care physicians who are less technology-oriented dropped from 44% to 39% of total physicians, procedural codes have tripled, surgical income is 50% greater than primary care income, and surgical rates have increased by 22%. Thus it seems reasonable to assert that for both hospitals and physicians at least one third and perhaps more of annual increases in costs are due to technologic advances.

For the past ten years, the medical device industry has increased the number of new devices patented each year by 5% (2,142 in 1979), has annually increased its work force by approximately 5% (217,500 in 1982), and has grown from $4 to $18 billion annually since 1972.[2] Not only does the health system pay this direct cost bill each year, but the cost of use over time will probably amount to more than the cost of acquisition.

Increasing emphasis is being placed on the application of technology in health care, and the implications of technology for the future depend not so much on the continued reliance on existing technology, but the continued trend of development and the use of more and better technology. Although forced economies may slow or divert improvements to other sites of use, both the public and competitive providers will require improvements in health care.

It is worthwhile to consider the potential effects of technology in the future. Such effects may be on cost, quality, outcome, access, demographics, employment, and struc-

---

Address correspondence to Dr Herdman, Assistant Director, Office of Technology Assessment, Congress of the United States, Washington, DC 20510.

ture of the health care system. Some of these effects may accelerate debate in the areas of morality and medical ethics (eg, gene therapy, life-sustaining treatment for the terminally ill) while others may be more directly related to health benefits and costs. The important roles of other influences, population changes, the general economy, corporate behavior, changes in statutes and regulation, and provider supply should not be overlooked.

A number of scientific developments, such as DNA technology (especially as applied to human gene therapy), implantable infusion pumps, monoclonal antibodies, magnetic resonance imaging (MRI) and spectroscopy, transplantation, and computers in medicine have come about as a result of advances in medical technology.

The study of DNA has become an important aspect of medical progress. Genetic products will be increasingly available and will have substantial implications either because of low cost, higher quality or safety, or availability for the first time. Many natural substances currently difficult to produce may result in changes in approaches to diseases. Their effects on costs, however, are hard to judge.

It seems likely that gene therapy will become a reality within the next decade. However, its immediate effect will probably be slight since initial attempts will be for rare recessive diseases (eg, adenosine deaminase deficiency, Lesch-Nyhan syndrome). The ability to detect gene abnormalities could have significant social implications for family planning or employment and the detection and quantification of new mutations might have great consequences for environmental and occupational health. However, public concern may surface if human mutations are detected as a result of occupational or environmental exposure to mutagens. Such technology therefore requires new detection and product capabilities. Cheaper products may mean savings, but the more likely implications are for greater advances in health care, some of which may generate considerable social concern.

Implantable infusion pumps are presently in use for the treatment of cancer, diabetes, anticoagulation, and pain control. Future projections for their use run as high as 120,000 per year. A computerized pump that could monitor its own effects and respond appropriately is believed to be an achievable objective. Such a development may not only control diabetes but also the complications of the disease. Artificial organs sensitive to biofeedback would have major effects on economic and societal costs and quality of life.

Monoclonal antibodies show great promise for the future. Because these antibodies are specific, pure, and reproducible, unlike conventional antibodies, they may be used for very precise diagnoses. Monoclonal antibodies can detect minute tumors easily and accurately, and categorize the tumors more precisely. These reagents are already in use with the approval of the Food and Drug Administration for the diagnosis of certain human diseases.

There are approximately 170 MRI devices in use in US institutions today that offer improved soft tissue imaging. However, it is unlikely that this development will replace other imaging methods sufficiently to justify its cost on that basis alone, and there is debate about the value of the information gained. But this new system offers more than imaging. It should lead to noninvasive biochemical tissue analysis in the future, although the clinical value of such analysis is not yet defined. The equipment costs up to $2 million to purchase and approximately $400,000 per year to operate. More hospitals will gradually acquire this machinery and use it while it continues to be improved, and third-party payors will cover the cost of this care.

Continued improvement in controlling the body's immune system may be expected to stimulate advances in transplantation procedures. In the case of kidney transplantation, since this procedure decreases the number of patients with active end-stage renal disease (ESRD), it can be considered cost reducing. In fact, analyses of more transplantation procedures and better graft survival rates suggest potential savings of greater than $500 million annually. For other types of transplantations additional costs appear likely given the absence of structured end stage programs to be relieved of patients. Thorough analyses are not available and the technology is much less standardized than is the case for kidney transplantation. The problems to be overcome in transplantation are organ supply and rejection; both of these are being addressed and have already improved. Thus the number of kidney transplants could possibly double or triple to 10,000 to 15,000 per year. The potential for other organ transplantation is less certain but probably also impressive. Transplant programs could consume a significant portion of resources at tertiary care centers if limited to certain centers as they are today and this may affect the structure, financing, and even stability of such centers. If organ transplants were confined to one diagnosis-related group in the next decade then that institution would be among the highest in economic value.

The use of computers in health care includes digitalized imaging; computerized medical records, patient, and financial management systems; low cost access to medical literature data bases; and computer-aided diagnostic and treatment decision support systems. These uses are not only effective in the health care setting but also in the home and office. The availability of hardware and medical information to patients, individual providers, and institutions increases the dissemination of health information, the capacity for participation by many former bystanders in health care, and the effectiveness of all health care providers. Since health care is often delivered at different sites, this development alleviates the problem of keeping track of patient information. There are therefore many opportunities to improve the quality, efficiency, employment, and structure of the health care system. Quality may not always be enhanced, however, given the potential hazard of the distribution of misleading information, especially into the home.

Along with computers there has been an increase in the development of procedures for use at home not only for pregnancy, blood sugar, and occult blood but also for other tests, such as those that monitor electrolytes in hypertension. In addition, in line with existing trends for fail-safe equipment for the office, the development of procedures for the full array of laboratory diagnoses should continue. In sum, not only will information advances provide data, but other developments will add new sources of data for consumers and noninstitutional providers to reinforce the existing American penchant for self help and the financial incentives for out-of-hospital office diagnosis and treatment. The "consumerization" of health care is perhaps another trend developing today that will influence the relationship between health cae advances and the health care delivery system.

It is difficult to predict the effect of technology on health care in the future even when it has been carefully investigated. Much of the medical advances occurring today have been reviewed by the Office of Technology Assessment, yet there are few cost effectiveness analyses available. There have also been few detailed analyses conducted of the implications of these advances, or any analyses beyond circumscribed safety and efficacy testing. Without these analyses it is not known which of these developments will make a major difference, even though it seems safe to say that many have that potential.

Current and projected developments in health care advances will continue to shape the health care delivery system. There will continue to be fewer patients in hospitals for shorter stays. A more technical capacity to influence disease and better control human biology will enable more patients to be treated at home. Hospital costs for more seriously ill patients should increase provided that hospital financing continues. With the same proviso, better care and declining morbidity and mortality rates should persist. In response to new developments and cost containment, fewer but more highly trained and flexible personnel will be working in high-technology care settings. Trends to longer and healthier life should continue considering the availability of social, retirement, and medical support systems.

Fewer but more costly hospitals, more expensive long-term care facilities, greater attention to the elderly, more expensive machinery in institutions and in outpatient settings, and more highly trained and highly paid personnel to use the new machinery will cause major increases in health care expenditures. As technological changes such as ambulatory surgery, infusion pumps, ambulatory dialysis, outpatient chemotherapy, and parenteral nutrition increase outpatient services at the expense of inpatient services, hospitals may stabilize or even shrink. However, these changes in the health system should not be ascribed to any one given factor. The changes in structure, quality, employment, and even technology itself are dependent on major influences such as financing, personnel supply, life style, and statutory and regulatory decisions as noted earlier. This is the difficulty in analyzing a system of such complexity, and in knowing how such a system should be managed.

REFERENCES

1. Romeo A, Wagner J, Lee R: Prospective reimbursement and the diffusion of new technologies in hospitals. *J Health Econ* 1984; 3:1–24.
2. *Federal Policies and the Medical Devices Industry.* OTA-H-230, US Congress, Office of Technology Assessment, US Government Printing Office, 1984.
3. *Medical Technology and Costs of the Medicare Program.* OTA-H-227, US Congress, Office of Technology Assessment, US Government Printing Office, 1984.
4. Wagner JL, Krieger MJ, Lee RH, et al: A study of the impact of reimbursement strategies on the diffusion of medical technologies, Vol. 1: Summary and findings. Prepared for the Urban Institute, 1982.
5. *Medicare: Paying the Physician—History, Issues, and Options.* Information Paper, 98th Congress, 2nd Session, US Senate, Special Committee on Aging, US Government Printing Office, 1984.
6. *Nuclear Magnetic Resonance Imaging Technology: A Clinical, Industry, and Policy Analysis.* OTA-HCS-27, US Congress, Office of Technology Assessment, US Government Printing Office, 1984.

FROM THE LIBRARY

SOCIAL INSURANCE

In this, the greatest period of social unrest which the world has ever experienced, every individual and every group of individuals has to face problems of more or less far-reaching importance. Were they given opportunity by their leaders, usually self-appointed and having as many motives as there are varieties of character, the great mass of the people would settle down into regular modes of life. But stimulated imaginations do not make for peace, and we can feel no assurance that the comparative sanity of 1915 will be recovered immediately.

We physicians have had our problem before us for several years. A set of radical measures which compel all employees in certain groups and their employers to pay sums of money into funds which are to provide to employees, without further cost, medical, surgical, and dental care, medical and surgical supplies, nursing, hospitals, and sanitarium care, case indemnities for time lost through illness, and funeral expenses. These measures are known as Social Insurance, or Health Insurance, and comprise the most radical scheme for social legislation which has ever been presented in the United States.

There is no other radical measure which has so nearly succeeded in being put upon the statute books in New York State, and there is no other measure which will be more actively pushed at the next session of the New York State Legislature.

This is our problem because the public welfare and the public health are involved. It is our duty to push ourselves forward and solve it. The problem is an acute one. We must get after our solution of it without delay, and when we decide what we will do with it, let us stick to our decisions.

HENRY LYLE WINTER, MD
(*NY State J Med* 1920; 20:15)

The roles of new medical technology and health care delivery systems in shaping medicine into the 21st century

PASCAL JAMES IMPERATO, MD

In view of current trends of health care delivery and in medical technology, it is reasonable to say that the provision of health care at the dawn of the next century will be markedly different than it is today. Predicting details about this new medical environment is obviously hazardous because so many new variables may intervene during the 15 years between now and the year 2000. One has only to compare the profile of our health care system today with that of 15 years ago, in 1970, to realize that the probability of the improbable taking place is quite high. However, unlike the time span of 1970 to 1985, the next 15 years are likely to witness the incorporation of technologies that will have a profound effect not only in modifying the diagnosis and treatment of disease but also in altering the structure of medical practice.

New technological advances will work in concert with a host of other factors, including choices made through the political process, that will profoundly change the environment in which medical care is delivered. The adoption and acceptance of these new technologic advances will be accompanied by old polemics, namely the debates about the appropriate loci of governance for technology assessment (private versus public), and arguments between economists, whose assessment of medical technology is based on cost-accounting evaluation tools, and physicians, who are trained to provide medical care using the best available technology no matter what the cost. The fact that providing optimal medical care with the best type of technology does not necessarily lead to better health will prominently figure in this debate.

A great challenge to both economists and physicians during the next several years will be the ability to reach a common ground that balances the often-conflicting objectives of cost containment and quality medical care.

If the immediate past is any predictor of the future, then there may be changes in the loci of technology assessment over time, with an ever-increasing role being played by government. For example, the National Center for Health Care Technology was created within the US Department of Health and Human Services in 1978 and was disbanded in 1981.[1] In 1984, Congress passed and President Reagan signed legislation that placed medical technology assessment in the National Center for Health Services Research, now renamed to include the phrase "and Health Care Technology Assessment." The new center will give greater scrutiny than its predecessor to technology now in use and that being proposed, particularly in terms of economic impact. Under the law, the center is directed to monitor the safety and effectiveness of technological advances and their cost effectiveness as well as medical appropriateness. Importantly, the center is to advise the Secretary of the US Department of Health and Human Services about which technologic advances should be paid for under programs such as Medicare.[2]

The 1984 legislation calls for the establishment of a National Advisory Council on Health Care Technology Assessment whose members are to consist of government representatives and individuals from outside the government prominent in law, ethics, medicine, engineering, management, and economics. The new law, as a concession to the private sector, calls for federal assistance to the National Academy of Sciences through the Institute of Medicine. Not surprisingly, this new legislation was strongly opposed by the American Medical Association. This opposition has its roots in AMA concerns about increased governmental involvement in medical technology assessment and its fiscal reimbursement ramifications and the fact that the AMA has itself been increasing its role in medical technology assessment through its Diagnostic and Therapeutic Technology Assessment Program.[3]

These recent events presage the future escalating conflict between physicians, whose training orients them towards the use of efficacious but expensive newer technology, and government, which wants to contain the costs generated by such use. Patient-consumers will not be mere spectators to this conflict because they have come to expect as necessary every potentially beneficial technology. Physicians, it should be noted, have likewise come to justify the use of any technology that provides even a slight return over zero. Scarred by malpractice experience and fears, most physicians tend to use new technologic advances to the fullest.

QUALITY OF LIFE

Ethical, moral, and legal issues, spawned by new technology that prolongs life, have already assumed enormous

---

From the Department of Preventive Medicine and Community Health, State University of New York, Downstate Medical Center, New York, NY.

Address correspondence to Dr Imperato, Department of Preventive Medicine and Community Health, State University of New York, Downstate Medical Center, 450 Clarkson Ave, Brooklyn, NY 11203.

Based on a presentation at the symposium entitled, *The U.S. Health Care System into the 21st Century,* January 21, 1985, New York University, New York City.

importance. These issues are currently being addressed in a variety of ways in the courts and state legislatures. However, it will be some time before comprehensive legislation is enacted and court decisions handed down that will cover all possible contingencies.

There are numerous medical treatment circumstances in which these issues arise. Premature babies or babies with a variety of congenital defects who heretofore died can now be kept alive, often indefinitely. However, their lives are often scarred by serious physical and mental disabilities. The quality of their lives is judged to be poor by some and not so poor by others. Sharp debates rage in our society about whether or not these babies should be kept alive and who has the right to make that decision. Baby Doe cases are now common on the court dockets. Thus far, there is no societal consensus, nor are there legal or legislative actions that can be viewed as setting guidelines to be followed in all circumstances. Parents of infants severely damaged by congenital defects cogently defend decisions to allow their children to die. Others can muster equally convincing arguments in favor of helping them to live. The sharpest conflicts have arisen when others, either physicians, public-interest groups, or government, step in and in the view of many usurp what is seen as a parental right. Parents in such circumstances who have opted for their children to die can and do present convincing arguments to support their decision. So do their opponents.

Reaching a consensus on this issue will be a protracted process because it involves the reconciliation of sharply divergent moral, ethical, and religious points of view. Yet eventually, a societal consensus must be reached and expressed through both legislative action and judicial decisions.

In the case of adults who are terminally ill, the issues are no less charged with conflict. Numerous individuals and public-interest groups have long campaigned for self-determination for the patient concerning continuation of treatment or dying with dignity. Twenty-two states and the District of Columbia have now adopted Natural Death Acts (NDAs), also known as living will laws, that permit mentally competent adults to declare, before they become ill, that they do not want their lives prolonged artificially.[4] Thirty-five states now have "brain death" laws that allow ventilators to be withdrawn when a patient no longer shows any sign of brain activity.

There is much variation in the wording of all of these laws from one state to another. In many laws, the term "life-prolonging" when applied to procedures or treatments is qualified in that death must be imminent or likely to occur whether or not the procedure or treatment is used. Definitions of this kind effectively exclude many comatose, vegetative, terminally ill, and technically dead people.

In many cases currently decided in the courts, some judges are understandably reluctant to hand down decisions. Clearly the existence of a living will law makes some decisions easier. Still, what about a lingering comatose patient who has no such will or one who changes his mind? Sometimes next of kin are allowed to decide. Sometimes relatives disagree. The fate of those without relatives frequently is decided by the courts. Recently, the Minnesota Supreme Court solicited the advice of three hospital ethics committees in reviewing a dying loner's case.[5] Increasingly, hospital ethics committees are playing an important role in decisions about suspending life-prolonging interventions and in the ultimate issuance of "do not resuscitate" orders.

Physicians and hospitals have moved very cautiously in this area, for both ethical and legal reasons. Some fear being indicted for homicide, and with good reason. In 1982, two California physicians were indicted for murder when they removed a comatose patient from a life-sustaining ventilator at the family's request. The indictments were later dismissed. Physicians worry about being sued for malpractice for stopping treatment. Yet in May 1984, an Ohio appeals court ordered a trial in a lawsuit filed by the survivors of a vegetative patient whom doctors refused to disconnect from life-support systems, even after the family obtained a court order. The family is accusing the physicians of battery.[5]

The number of cases coming into the courts is increasing. This is likely to continue. On the one hand there are those who argue that life-sustaining technology is being applied because of adherence to technological imperatives rather than in response to consideration of patient wishes, quality of life, and futility of treatment. On the other hand are those who argue that every possible technology must be applied to sustain every life. The cost implications of these different views is enormous.

According to a study sponsored by the federal Health Care Financing Administration, 28% of the annual Medicare budget is spent on maintaining those over 65 years in their last year of life. Most of this sum is spent on individuals in the last month of life. This means that some $20 billion is annually being spent on dying elderly patients, equivalent to what was spent in 1984 by the federal government on assistance to the unemployed.[6] The recitation of such statistics gives cause for concern to many who fear that cost considerations might enter into the decision-making process about who lives and who dies. Whether or not cost considerations will be part of these decisions in the future is an open question at this time.

What is clearer is that the courts will progressively establish guidelines covering an enormous range of situations by issuing opinions, particularly where a void exists because of the absence of legislative mandates. This has already been happening for some time. However, two precedent-setting decisions have recently attracted great attention because of their departure from previous court opinions and because they essentially establish new guidelines. By far the most important of these is the Conroy Decision of the New Jersey State Supreme Court in 1985. It is beyond the scope of this article to delve into all of the ramifications of this decision. Essentially, it eliminated the distinction between ventilators and artificial feedings in the case of an 84-year-old patient in a nursing home. Previously in New Jersey, ending such care was permitted only for patients who were brain dead, that is, individuals who had no brain-stem function or who were comatose and in a persistent vegetative state. The court also said that there is no distinction between a doctor agreeing not to start a particular treatment and agreeing to end treatment once it was started.[7] Many hospital ethics committees have made such a distinction and have viewed fluid and nutrient administration to a patient as different than extraordinary means of life support, such as ventilators.

The Conroy Decision is carefully worded so that it applies only to certain types of patients: the elderly with no hope of survival beyond a year, who, though now incompetent but not brain dead, made clear while competent that they did not favor the use of life-prolonging procedures. Still, this decision will have a major impact on the death and dying issue throughout the country. It also has intensified the debate among the right-to-die and right-to-live groups within the country. It is worth noting that in 1983 the Appellate Division of the NJ State Superior Court ruled that the removal of a feeding tube in the patient would have constituted homicide and would have violated the fundamental principle to do no harm. The state's Supreme Court had overturned that decision.

The second court decision that has set a precedent is that of the New York State Court of Appeals, which in 1984, handed down a decision that recognizes the use of brain-based criteria in determining death. Although brain-death criteria were in use in New York State before this decision, there were many uncertainties because New York has no statutory definition of death, and the conventional concept of cessation of cardiopulmonary functions was frequently the criterion. This did not take into account contemporary means for artificially maintaining respiration and cardiac function.

The court's decision concerning brain death was in response to two separate appeals from defendants convicted of homicide. Both victims died of bullet wounds to the head inflicted by the defendants. They were declared brain dead and their organs removed for transplantation, and they were then disconnected from ventilators. The defendants' argument on appeal was that the respective juries had not been adequately instructed as to what constitutes a person's death. In New York State up until this time, death traditionally meant the cessation of cardiopulmonary function. The defendants argued that the juries may have erroneously concluded that they were guilty of homicide if they caused the victim's brain death rather than their ultimate cardiorespiratory failure. In other words, the defendants claimed that because they did not cause their victims' cardiorespiratory failure, they were not guilty.[8,9]

There will no doubt be a prolonged debate in the years ahead concerning these complex issues raised by modern medical technology use. The development of a societal consensus is a slow process in this area. Already, there are signs that a beginning is being made. The President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research issued 11 separate reports covering numerous issues and recommending guidelines. Pertinent to this discussion are its definition of death and the proposal for a Uniform Determination of Death Act. These have been strongly endorsed by the American Medical Association, the American Bar Association, and many other groups.

In New York State, Gov. Mario Cuomo appointed a 23-member Commission on Life and the Law in December 1984 to review and make recommendations by May 1, 1985, on a wide range of medical technology issues.

The ethical issues raised by current medical technology pale in the view of some against what lies ahead in the next century. One has only to consider genetic engineering to realize the wisdom of this point of view.

FUTURE HEALTH-CARE TRENDS

As if all of these issues related to technology are not enough, changes in the structure of our health-care system are also occurring. Essentially, two parallel processes are taking place, the emergence of new types of organizations (eg, preferred provider organizations, surgicenters, and emergicenters) and the evolution of new roles for established organizations. There are a number of forces that are bringing about the development of these new modes of medical care. However, in my view the resource crucial to their rapid growth is the ever-increasing supply of physicians and other providers of health care.

In 1980 there were 435,545 physicians in the United States. By 1990 there will be 563,276 and by 2000, 665,730. There will be a 54% increase in absolute numbers between 1980 and 2000, making for 248 active physicians per 100,000 persons in the United States in 2000.[10]

Market forces under our economic system have already responded to these increasing numbers in a variety of ways. Geographic diffusion into small towns and rural areas is taking place at a rapid rate. Shifts away from overcrowded specialties to less-crowded ones also are evident. Medical staffs of hospitals, once receptive to new, younger physicians, are increasingly protectionist, either closing the door completely for staff privileges or erecting various barriers that force young physicians to go elsewhere. These strategies buy economic protection in the short run for hospital medical staffs, but they also divert the manpower necessary to staff alternate models of medical care that are and will be their arch competitors.

Compared with older physicians, younger ones tend to hold more positive attitudes toward nontraditional practice arrangements. Besides the barriers to hospital medical staff privileges, these young and often highly indebted (average, $40,000) physicians face high costs in establishing practices. The expansion of nontraditional practice arrangements provides opportunities denied them in the traditional models, the solo and group medical practices. It also enables them to begin practice without incurring further debt.

The hospital sector is likewise going to change dramatically. The number of community hospitals is declining slightly (5,746 in 1972, compared with 5,813 in 1981). However, average size increased by 13% during the same time from 153 to 173 beds. While the number of investor-owned (for-profit) community hospitals decreased from 738 to 729 during this period, the number of beds in these hospitals increased by 54%. Another development in the hospital sector is the management of not-for-profit beds under contract by for-profit hospital chains. The number of such beds increased by 105%, from 16,089 in 1976 to 33,058 in 1981. The growth of multihospital systems also will continue.[10]

Within hospitals it is reasonable to predict a steady erosion of the influence of the medical staff on hospital policy. Hospitals will have to become more competitive in outlook and their management more businesslike. These factors, coupled with hospitals joining multihospital systems, will erode medical staff influence. Most physicians, by reason of their training, experience, and outlook, possess skills that are ill-matched to the complex management and fiscal issues that hospitals currently and will continue to face in the future. The level of polemic will rise in the physician sector, and organized medicine, because it represents physician interests, will have to assume a major role. The surplus of physicians will give managers and administrators enormous leverage with uncooperative physicians. Increasingly larger numbers of physicians will be employed by hospitals under a variety of arrangements, further eroding the traditional historical relationship between physician and hospital.

Overall we will see a progressive integration of medical practice with various types of health-care institutions, including hospitals. Private practice physicians will be in competition with hospitals and other models of care for patients. More and more physicians will come to work as staff members of health maintenance organizations, surgicenters, hospitals, and others, avoiding the competition at least for themselves. The driving engines of all these structural changes are obviously price and cost. However, without a physician surplus, it is doubtful that many of them could take place.

There are many other trends that will develop as we approach the millenium. For many in the health care field, these trends will prove unsettling to their long-established and comfortable way of doing things. For others, however, they will usher in new changes that will prove to be a rewarding challenge.

REFERENCES

1. Perry S: Special report: The brief life of the National Center for Health Care Technology. *N Engl J Med* 1982; 307:1095-1100.
2. Legislation expands federal role in medical technology assessment. *JAMA* 1984; 252:3294.
3. Cahill N, Beljan J: Technology assessment: Differing perspectives. *JAMA* 1984; 252:3294-3295.
4. Novak N: 'National death acts' let patients refuse treatment. *Hospitals* 1984; 58:71-73.
5. Arguing the right to die. *Newsweek*, Jan 7, 1985, p 18.
6. Kleiman D: Prolonging life: The new dilemma. Part 3: Uncertainty clouds care of the dying. *The New York Times*, Jan 18, 1985, pp B1-B2.
7. Sullivan R: "Right to die" rule in terminal cases widened in New Jersey. *The New York Times*, Jan 18, 1985, pp A1, B2.
8. *The People v John W. Eulo*, State of New York Court of Appeals, no. 402, October 30, 1984.
9. *The People v Robert Bonilla*, State of New York Court of Appeals, no. 403, October 30, 1984.
10. *The Environment of Medicine: Report of the Council On Long Range Planning and Development*. Chicago, American Medical Association, 1984, pp 39-45.

FROM THE LIBRARY

ANAESTHESIA

Among the discoveries of the present age, wonderful as they are in variety and extent, and beneficient in their results, nothing, in its power to relieve human suffering, equals in importance that of chloroform, and its kindred compounds. If it be not the "elixir of life," so eagerly sought for by the ancient visionary, it is at least the prompt assuager of pain, the mitigator of suffering, to an extent unequaled by any other known agency. Almost do we behold, converted into realities, those phenomena which heretofore have been regarded as only amongst the pleasing illusions of mythological fiction. The Lethean stream is no longer a fable, for we have only to breathe the liquid atmosphere of this new creation than the same marvellous effects are wrought as are claimed to have resulted from bathing in its limpid waters. The pains and sorrows of life are not only buried in temporary oblivion, but give place to sensations of pleasure and joy.

Much as we are indebted to the results of chemical research for the discovery and appliance of those principles of nature science, by which commerce is revolutionized, civilization advanced, and the means of human enjoyment multiplied and exalted, yet when we consider how large a proportion of our race are unfitted, by reason of pain and sickness, from participating in these advantages, we feel as though, to the discovery of a therapeutic substance which effectually abolishes pain, and thus takes from disease its keenest sting, we, as members of the "healing art," are more indebted than to any other which chemical science has developed during the present century.

For after all, of what values are discoveries in science to the sick man, absorbed with the desire of relief from pain, if they bring no balm to assuage his sufferings? Man is, to a large extent, the creature of circumstances; he is influenced by the condition of things within and around him; he is interested with, and values most, that which contributes to his present need and enjoyment. Thus, when wasted by disease, or racked with pain, of what interest to him is the discovery of any new principle in science, if it has no power to ameliorate his condition?

It has been said that he who can make one additional blade of grass to grow is worthy to be named a public benefactor,—how much more is he who has added a new article to the list of life-preserving health-restoring agencies entitled to this distinction.

The discovery and introduction of anaesthetic remedies in the treatment of disease, and especially chloroform, marks a new epoch in the history of medicine. It places in our hands a new power, not doubtful in its effects, but sure and reliable, and almost instantaneous, enabling us, in many instances, to fix the precise limitation of suffering, and say of pain, so long may it continue, but no longer.

PETER VAN BUREN, MD
(*Trans Med Soc State NY* 1858; 85:49-50)

Implications of changes in payment practices for health care

The insurer's perspective

Michael R. McGarvey, MD, Sharon B. King, JD, MPA

National health expenditures (public and personal spending), which amounted to $27 billion in 1960, increased from $133 billion in 1975 to $249 billion in 1980, and reached $355 billion in 1983.[1] Thus, while in earlier years it took 15 years (1960 to 1975) for health care expenditures to show a $100 billion increase, such expenditures increased by more than $100 billion in the last three years alone. It is apparent that such progressive increases cannot long be sustained without severe dislocations in the economy.

Where does the health care dollar go? Forty-two cents of every dollar is spent on hospital care; 19 cents goes to physician services; nine cents is spent on nursing-home care; 19 cents goes to other personal health care including such services as dentistry, podiatry, and drugs; and 11 cents goes to other health spending, including basic biomedical research and the costs of medical education.[2]

National health expenditures as a percentage of the gross national product have steadily increased, from 4.4% in 1955 to 10.8% in 1983.[3]

The annual percentage change in national health expenditures has consistently exceeded the percentage change in the consumer price index. Though the gap began to narrow in the late 1970s and early 1980s, it is beginning to widen again.

Notwithstanding these worrisome national figures, Blue Cross and Blue Shield of Greater New York can take pride in the impact of its continuing cost-containment efforts. For example, Blue Cross and Blue Shield of Greater New York hospital admissions per 1,000 people from 1975 to 1983 were significantly below the Blue Cross national average and the admissions per 1,000 of the US general population under age 65. In 1983, for example, Blue Cross and Blue Shield of Greater New York had 76 hospital admissions per 1,000 population, compared with 108 for all other Blue Cross plans and 134 for the US general population under age 65.[4-6]

The Blue Cross and Blue Shield of Greater New York average length of stay from 1975 to 1983 for all admissions declined steadily from 7.52 days in 1975 to 6.34 in 1982, with a slight increase in 1983 to 6.39.[4-6]

Hospital days per 1,000 population during this period also showed a steady decline, from 650 in 1975 to 487 in 1983. This compares with the 1983 figure for all other Blue Cross plans of 671 inpatient days per 1,000 population and 775 for the US general population under 65.[4-6] One might well ask, "What accounts for these dramatic differences? Why is Blue Cross and Blue Shield of Greater New York so significantly below the US general population and the national Blue Cross experience in its utilization of hospital services?"

Part of the answer undoubtedly relates to the fact that our corporation is in a state that has led the nation in the area of regulatory activities in health care. New York had the first Certificate of Need mechanism in the nation and continues to have one of the most stringent. As a result of the fiscal crisis of the city and state of New York in the mid-1970s, some 50 marginal hospitals closed, and literally thousands of unnecessary hospital beds were removed from the system. The state's Public Health Law requires each hospital to have a utilization review mechanism that applies to all patients, regardless of payment source. Finally, New York was home to several of the nation's most aggressive and effective Professional Standards Review Organizations.

These facts do not, however, explain the entire difference. Other Blue Cross and Blue Shield Plans in the state report utilization experiences closer to the national Blue Cross figures. We can only conclude that the many activities in which Blue Cross and Blue Shield of Greater New York has been involved during many years, work done in connection with our member hospitals, and participating providers aimed at assuring the responsible use of health resources, have been effective.

From 1971 to 1983, average hospital expenditures per inpatient day in New York City increased at a slower rate than in other major metropolitan areas across the country.

From the early 1970s, when New York was unquestionably the most expensive city in America for a day of hospital care, our effective efforts to control costs brought us to the rank of 47th among cities in the nation. It must be emphasized that this cost containment has been accomplished without sacrificing quality of medical care, which

---

From Health Services Management, Empire Blue Cross and Blue Shield, New York. In July 1984, Blue Cross and Blue Shield of Greater New York merged with Blue Cross of Northeastern New York to form Empire Blue Cross and Blue Shield. The speech upon which this article is based was given prior to the merger.

Address correspondence to Dr McGarvey, Vice-President, Health Services Management, Empire Blue Cross and Blue Shield, 3 Park Ave, New York, NY 10016.

Based on a presentation at the symposium entitled, *The U.S. Health Care System into the 21st Century,* January 21, 1985, New York University, New York City.

continues to rank among the best in the world. The Blue Cross and Blue Shield of Greater New York basic philosophy holds that unnecessary services are not only financially wasteful but also pose the risk of damage to the patient. By limiting unnecessary utilization, patients, employers, and payors all benefit.

COST CONTROL

Blue Cross and Blue Shield of Greater New York's methods for cost control have been many and varied during the years, beginning in 1935. These include careful scrutiny of claims in the benefits administration process, cost-based hospital reimbursement on a per-diem basis, the introduction of a pioneering home health care benefit as early as 1960, institutional and professional services utilization review, the introduction of an ambulatory surgery incentive in 1970, implementation of a strong coordination of benefits program, the initiation of numerous demonstration projects designed to test the quality and cost effectiveness of alternate delivery systems, and the introduction of the health maintenance organization (HMO) in 1973.

More recently, these initiatives have included the Program for Elective Surgical Second Opinion, which began in 1976; an expansion and strengthening of program security operations to detect and prevent fraud and abuse; significant expansion of voluntary health education programs; the encouragement and support of hospital energy management programs; and, in 1980, the implementation of hospital discharge planning projects, health services utilization reports, computerized hospital management and information systems, and a hospital laboratory effectiveness program.

Since 1980, and in response to increasing market demands for cost sharing, cost shifting, and cost containment, Blue Cross and Blue Shield of Greater New York has developed with participating hospitals its Due Care/Hold Harmless Program, designed to strengthen hospital utilization review programs while protecting subscribers from financial risk due to medically unnecessary and inappropriate utilization. The new TEAM Care Program was begun in cooperation with those accounts that desire to introduce preadmission and concurrent utilization review programs for their member hospitals. Also, the new account-specific data analysis and utilization analysis are aimed at providing accounts with the basis for appropriate action plans. The new Ancillary Services Review Program, in cooperation with member hospitals, highlights and corrects costly overutilization of selected ancillary services in the hospital setting.

The new cost-sensitive environment requires that Blue Cross and Blue Shield of Greater New York be more responsive and efficient than ever before in its contract administration and ever more creative in developing cost-containment programs.

ADEQUACY OF ECONOMIC MODEL

Payment practices and how they are administered have a major impact on the demand for or the utilization of health care services. A threshold question in trying to understand or predict that impact is the adequacy of the economic model. Unlike most areas commonly viewed through the economist's lens of supply and demand in health care, adding doctors to a given area has not driven prices or costs down. Instead, it has reduced individual productivity and added several hundred thousand dollars to the cost of health care for every additional practitioner.

A major problem in applying principles of economics to the health care field until very recently has been a failure to identify the actors correctly. The most common example of this is failure to determine who the consumer really is. Because of rapidly enriched health-care insurance benefits during the last four decades, the individual patient is almost never the type of consumer envisioned in economics. The patient may be the beneficiary or victim, but rarely someone who makes his decisions in a way influenced by the price of services. Insurance companies have been intermediaries, conduits through which vast sums have been pumped. The real consumer has been the agent who pays the health-care insurance premiums: employers, union welfare funds, the government, and the direct-pay insured. These real consumers are finally waking up, with interesting consequences.

First, a variety of barriers are being erected against continued expansion and inflation. These include prospective payment systems using diagnosis-related groupings; cost sharing and cost shifting; benefits management including interventional techniques such as second surgical opinions, mandatory ambulatory surgery and preadmission and continued-stay reviews; and preferred provider arrangements including preferred provider organizations and HMOs.

Second, competition is finally occurring among providers and among insurers.

Against the background of what has been accomplished to date, any future efforts to reduce the rate of increase in health-care costs must have two basic goals: (1) to reduce average length of stay and (2) to encourage more appropriate utilization of resources.

INTERVENTIONAL APPROACH

An increasing number of accounts are looking to their health-insurance carriers or consultants to recommend specific cost-containment activities that take an aggressive approach to cost management; we describe such activities as "interventional." Blue Cross and Blue Shield of Greater New York has a growing number of such activities available to our customers.

The TEAM Care unit derives its name from the phrase To Encourage Appropriate Medical Care. This program incorporates cost-management methods designed to minimize unnecessary health services and to channel individuals who do need care toward appropriate high-quality, cost-effective services.

The TEAM Care Program, at the option of the group, can include preadmission certification of benefits for planned hospital admissions, incentives for second surgical opinions and ambulatory surgery, concurrent utilization review, discharge planning, catastrophic or individual case management, and subscriber health education programs. Indirect results of the program are more informed consumer use of health services; better quality of care; morale, productivity, and corporate image enhancement; disease prevention; and reduced absenteeism. Direct results include less unneeded care and dollar savings.

The TEAM Care staff includes a physician director, a nurse as manager, nurses and care review analysts, physician consultants from many specialties, a health resource specialist, and a research associate.

Preadmission review entails review of detailed information on scheduled hospitalizations and/or procedures. This review provides not only an opportunity to assess the appropriateness of the proposed level of care, but also an opportunity to advocate cost-effective alternatives.

Second surgical opinions can be promoted where there is concern that a proposed procedure may not be necessary. Ambulatory surgery can be promoted when it offers potential savings without compromising quality of care.

Account-specific reporting aims at helping our customers understand more fully where their health-benefit dollars are going. It may include analysis by diagnosis in terms of frequency, dollars, length of stay, and the relationship of the patient to the policy holder.

It may also include analysis of hospital services in terms of dollars, length of stay, day of admission, short stays, and comparison with other hospitals.

It can also include analysis of outpatient services in terms of dollars, frequency, and rates.

On the basis of such analyses, Blue Cross and Blue Shield of Greater New York will be able to offer major accounts an interpretation of utilization results, recommendations for specific cost-management activities, and personalized consultation with account staff.

Account-specific consulting provides a tailored report and action plan. The report is designed and produced to address the specific management needs of an account. Each report includes a thorough analysis and interpretation of the data. Based on that information, a program that is responsive to the identified needs of a specific account is formulated, implemented, evaluated, and modified as necessary.

Looking further into the future, one can predict that cost-containment activities will continue to proliferate and begin to focus on such new areas as the expansion of physician independent practice associations, preferred-provider arrangements with both physicians and hospitals, imposing restrictions on one-day hospital stays, extending outpatient benefits to include such items as outpatient diagnostic services and outpatient chemotherapy and emergency care, and developing new and less-expensive long-term care modalities. Many of these changes will require a redesign of contract benefits and the premium structure, and must be accompanied by the creation of extensive new advanced computerized cost and data management systems.

It has been suggested that by the year 2000, essentially all health services in the nation will be organized under 30 or 40 huge, multifaceted organizations that will provide traditional insurance, direct health services in varied forms, and a wide variety of related cost-management services.[8] We are inclined to agree. We are all in for interesting and challenging times.

REFERENCES

1. Office of Financial and Actuarial Analysis, Bureau of Data Management and Strategy, US Health Care Financing Administration.
2. *Health Care Financing Review*, fall 1983, vol 5, no. 1, p 5.
3. Office of Financial and Actuarial Analysis, Bureau of Data Management and Strategy, US Health Care Financing Administration.
4. *Hospital Discharge Survey*. Bureau of Health Statistics, US Dept of Health and Human Services.
5. Information Services, Communications Division, Blue Cross and Blue Shield Association.
6. Actuarial Department, Blue Cross and Blue Shield of Greater New York.
7. *Hospital Statistics*. Chicago, American Hospital Association, 1972 and 1984.
8. Starr P: *The Social Transformation of American Medicine*. New York, Basic Books, 1982.

MARY AND DAVID HOAR FELLOWSHIP
Musculo-Skeletal Trauma Research July 1, 1987

- In 1975 the Mary and David Hoar Fund was established at the New York Community Trust to promote research to improve treatment of fractured hips.
- The fellowship shall be applied to research in musculo-skeletal trauma under the aegis of an orthopedic surgery department, preferably at an institution in New York City.
- Fellowships are available to physicians and basic science investigators.
- Every applicant must be recommended by a letter from the Orthopedic Surgery Department Chairman of his/her institution, and a letter from the individual who will supervise the fellowship program. These letters should specify plans for support and identify what resources the institution will make available to the Fellow.
- Application forms, which must be submitted by March 31, 1986, may be obtained from—

  Office of Medical Education
  The New York Academy of Medicine
  2 East 103rd Street
  New York, New York 10029
- Interviews may be required and selection of Fellows will be made by the Mary and David Hoar Fellowship Committee. The award will be announced by May 15, 1986.
- Reports mid-year and upon completion of the fellowship will be required. In addition, a paper on hip fracture prepared in conjunction with the research in musculo-skeletal trauma and suitable for publication in a peer review journal must be submitted.

Health care payment reform

Projections for the next 15 years

GERARD F. ANDERSON, PHD, CATHERINE R. RAPOZA

The **health-care** system is undergoing dramatic changes. As the percentage of the gross national product devoted to health care has grown, a cost-containment movement has developed that has affected both the delivery and method of payment for medical care. In this article, the forces contributing to the changes in the method of financing health care are reviewed and payment reforms that are likely to occur during the next 15 years are suggested.

Our initial hypothesis is that payment reform can be viewed in three overlapping periods. The first period, which is slowly ending, is one in which providers set prices. In this period hospitals, physicians, and other providers of health-care services are able to set their own prices without significant constraints imposed by the purchasers of health-care services. In the next period, which has already begun, the purchasers of health care services determine the price they are willing to pay. This has been done using a variety of methods, including the Medicare Prospective Payment System and preferred provider organizations. In the third period, which has been initiated in only a few isolated places, providers set prices and consumers choose among competing providers based on price.

Obviously this classification system is oversimplified. Neither the passage from one period to the next nor the characteristics described are as clear in reality as will be portrayed. We have classified the various systems in this manner to highlight the essential differences in the three systems.

PROVIDERS SET PRICES

Historically, hospitals have had considerable latitude in setting both charges and costs. Cost-based payors, which include Medicare, Medicaid, and some Blue Cross plans, have paid hospitals the amount the providers report it costs to provide care. These payors' participation in determining the level of payment has been limited to establishing categories of allowable costs and disallowing certain categories of expenditures. Charge-based payors have had even less input in establishing the level of payment; they pay the provider the amount they are charged. Many hospitals have based their charges not on the actual cost of providing patient care but on their revenue needs. Simply, they have set charges to cover their costs and to allow for the cross-subsidization of money-losing services (eg, outpatient clinics), services that are not explicity funded (eg, graduate medical education), and uncompensated care. In this same period, physicians have also been free to determine their own fees.

Providers have had the power to set their own prices with little interference from payors or consumers for a number of reasons. First, widespread medical coverage by third-party payors virtually eliminated consumer concern about price. As medical care became more complex and expensive during the last four decades, private insurance coverage became both more common and comprehensive. Consumers were rarely affected by the provider-set prices and thus there was little, if any, incentive for the consumer to question the fees charged. In addition, when Medicare and Medicaid were established in 1965, there was a widespread concern that providers would refuse to treat patients covered under the public insurance programs. The fear of nonparticipation was a principal factor in the federal government's willingness to accept the hospitals' own costs and physicians' own fees. Finally, providers were able to set their prices because the methodology for determining the cost of producing efficient medical care had not been developed. Thus, payors accepted provider-set prices because there was no methodology for establishing a fair price.

The most notable outcome of this period is that health-care costs have escalated. In 1965, total expenditures for health care were 6.1% of the gross national product, and by 1984 they had increased to 10.8%. Along with these higher costs came substantial improvements in medical care such as dramatic increases in new life-saving medical technology, improved access to care and health status, increased quality of care, and decreased morbidity and mortality.

PAYORS SET PRICES

As the concern about the increasing cost of health care has spread from the government to insurers to the business community and finally to consumers, numerous cost-containment proposals have emerged. We are now moving into an era where payors set prices for health-care ser-

---

From the Center for Hospital Finance and Management, the Johns Hopkins Medical Institutions, Baltimore.

Address correspondence to Dr Anderson, Center for Hospital Finance and Management, the Johns Hopkins Medical Institutions, Baltimore, MD 21205.

This work was supported by a grant from the John A. Hartford Foundation, New York, NY.

Based on a presentation at the symposium entitled, *The U.S. Health Care System into the 21st Century*, January 21, 1985, New York University, New York City.

vices. Most commonly, prices are set by payors on a per-unit basis (eg, per day, per procedure, per admission), in an effort to regulate the amount paid. Medicare, for example, now reimburses hospitals for inpatient care based on prospectively set rates for each of 467 diagnosis-related groups (DRGs). Under the prospective payment legislation, Congress also required the US Department of Health and Human Services to propose new methods for prospectively paying for physician services. It is reasonable to assume that in the near future Medicare will establish prospectively set prices for nursing home and home health care.

Prospective payment is being explored by other payors as well. Some Medicaid programs and Blue Cross plans have already adopted versions of Medicare's payment methodology. Other payors have developed alternative methods of cost containment. Some insurance companies have established preferred provider organizations (PPOs) to steer their enrollees to low-cost providers. In many cases, the insurance company sets the price it is willing to pay the hospital to be a preferred provider.

These changes have been precipitated by a number of interrelated events. Providers have accepted payor-set prices because their financial stability is in question and thus their bargaining power has eroded. For example, the spread of competitively priced alternative delivery systems (eg, HMOs and PPOs) has created heavy competition for insured patients. Concurrently, hospital occupancy rates are at a historically low rate: an average of almost 70 percent across the nation. Consequently, many hospitals cannot afford to lose Medicare and Medicaid patients, which represent almost half of their patient population. Even individual commercial insurance companies (Aetna and Travelers, for example), which may cover only 1% or 2% of the patient population in any hospital, are increasingly able to negotiate prices with hospitals.

Physicians have a similar problem stemming from an excess supply. Various studies on physician supply indicate that by 1990 there will be a significant surplus of physicians.[1,2] The pressures of oversupply are already evident. Young physicians entering the work force are no longer guaranteed an opportunity to practice in the community of their choice. One barometer of the employment environment is that HMOs are able to recruit staff physicians with ease, and these physicians are accepting lower starting salaries than those entering the job market even five years ago. Thus, the economic environment is pressuring providers, both hospitals and physicians, to participate in programs where others set prices.

A second factor contributing to the change in payment patterns is that methodologies for measuring case-mix and setting prices have improved. DRGs measure the case-mix of the hospital, while adjustments for regional cost differences, indexes that measure inflation in specific health sectors, and other factors have permitted payors to set prices. Research is continuing on improved case-mix determination, better measures of input costs, separating the cost of education and research from that of patient care, and in other areas that will continue to improve the price-setting methodology. All of these improvements will increase payors' confidence that they have identified the low-cost and most efficient provider.

Finally, there is a belief that as a nation we are spending too much on health care. Between 1977 and 1982, Congress repeatedly debated the need to control health-care spending through some type of payment reform. In 1977, President Carter introduced hospital cost-containment legislation explicity designed to control hospital costs. Finally, in 1982, Congress passed the Tax Equity and Fiscal Responsibility Act, which set limits on how fast expenditures per admission could increase in the Medicare program. This was a prelude to the system of administered prices that is the basis of the Medicare Prospective Payment System.

The long-run impact of this payment reform depends substantially on how prices are set. This is an area where we have very little guidance from economics because we have few models of how prices will be set by the government when the government is the major purchaser. However, regardless of how prices are set, we know that providers will respond to the new environment. Generally, providers can be expected to (1) reduce the amount of cross-subsidization, (2) slow the growth in technology acquisition, and (3) shift the site where care is delivered.

As externally established prices become the rule rather than the exception, providers' flexibility in cross-subsidizing various products will be reduced. Hospitals, for example, will no longer be able to use patient care revenues to subsidize other products such as graduate medical education, research, and uncompensated care because prices will be set to cover only the cost of providing patient care. Once the implicit funding for these additional products is removed, hospitals will need to identify explicit funding sources for these other products. Without this funding, these activities wil be reduced.

The trend toward payment reform will also affect the development of new technology. The method for establishing prices for patient care can have a major impact on the development and diffusion of technology. This is perhaps best illustrated through an example of the effect of DRG prices on technology.[3] A major corporation has recently developed a new prosthetic device to be used in hip replacement surgery. The DRG pricing formula provides one price for hip replacement based on the cost of the old prosthesis. The new technology is a permanent implant, whereas the old technology had to be replaced every five years. Consequently, the new technology is better for the health of the patient and allows for long-run cost savings. The major drawback, however, is that it is considerably more expensive than the old technology. Without an increase in the DRG price for hip replacement, a number of questions arise. Will the hospital be willing to purchase the product if it incurs a financial loss on each implantation? Will the corporation developing the technology be willing to incur the marketing costs to offer the prosthesis if hospitals have an economic disincentive to use the product? Will consumers ever have access to this new technology? The answer to these questions depends largely on how the DRG prices are adjusted to accommodate technologic advances, and this could have a major impact on the diffusion of new technology.

Payment reform would also affect where and when care is provided. The Medicare Prospective Payment System creates an incentive for providers to increase admissions and may encourage readmissions. Because the DRG payment is a fixed rate per inpatient stay, there is an incentive to discharge patients as soon as possible. This may lead to the discharge of some patients earlier than is medically advisable.[4]

Access to care may also be reduced. As payors steer consumers to low-cost delivery alternatives through the development of preferred provider arrangements, consumers' freedom of choice may be curtailed. In addition, prospective reimbursement may encourage hospitals to "dump" severely ill patients, who are more expensive to treat, on area teaching and public hospitals.

PROVIDERS SET PRICES AND CONSUMERS CHOOSE PLANS

Future payment reform will represent a merger of provider and payor interests in which providers set prices and consumers have the option of choosing among competing plans. In this competitive model, providers have greater control over their own prices but have to compete on the basis of price for patients.

Before moving into this third period of payment reform, many problems must be solved. One of the most important is that consumers will need information upon which to base their choices of a health plan. In the past, health insurance information has been so complex that consumers were overwhelmed by detail. An example is the Federal Employee Health Benefit Plan, which offers government employees 20 different health plans. Few federal employees are motivated to invest the time necessary to understand the nuances of each plan, and therefore many are not making truly informed decisions.

The second major policy question is how prices can be set to eliminate favorable and adverse selection. On Jan. 10, 1985, Medicare issued regulations allowing HMOs to enroll Medicare beneficiaries and receive 95% of the average annual per capita cost (AAPCC) of treating this group. Payment is made regardless of whether the beneficiary receives medical care. The expectation is that the Medicare program will save 5% for each HMO enrollee because it pays only 95% of the AAPCC. However, nearly half of the Medicare-eligible population use no Medicare-covered services in a given year. If HMOs attract a higher percentage of the good risks, and the high utilizers continue to choose the Medicare program as their source of coverage, then this new system will increase Medicare costs. The methodology for determining price is critical to the development of a payment that is equitable to both payors and providers.

IMPLICATIONS OF PAYMENT REFORM

Payment reform, as it is occurring today and as it is envisioned in the future, has a variety of implications for the health-care industry in the years ahead. The pressures of these reforms will result in decreased funding for products other than patient care such as new technology, graduate medical education, research, and uncompensated care. Consequently, there will be a decrease in the development and marketing of new technology, the size of graduate medical education programs, the amount of hospital based research, and the number of hospitals financially able and willing to care for the indigent population. At the same time there will be more nonhospital services as hospitals unbundle their services to maximize reimbursement. Competition will grow as providers lower their charges, pricing services with an eye to the competing market. Finally, there will be significant redistribution of wealth within the health-care industry. The redistribution will occur among hospitals, across regions of the country, between urban and rural settings, and among physician specialties.

REFERENCES

1. Graduate Medical Education National Advisory Committee (GMENAC): *Report of Graduate Medical Educational Advisory Committee to the Secretary*, US Dept of Health and Human Services, September 1980, pp 1-41.
2. *Medicare Benefits and Financing: Report of the 1982 Advisory Council on Social Security to the Secretary*, US Dept of Health and Human Services, February 1984, pp 69-70.
3. Anderson G, Steinberg E: To buy or not to buy: Technology acquisition under prospective payment. *N Engl J Med* 1984; 311:182-185.
4. Anderson GF, Steinberg EP: Hospital readmissions in the Medicare population. *N Engl J Med* 1984; 311:1349-1353.

A 74-year-old man with a lung mass, arthritis, and difficulty walking

CASE PRESENTATION

Mark Darrow, MD (Medical Resident): A 74-year-old retired farmer first came to the hospital complaining of difficulty breathing, a 10 lb weight loss, upper abdominal discomfort, a decrease in appetite, and generalized weakness for three weeks. He also had had a nonproductive cough for several months associated with a sharp interscapular pain. He denied any change in bowel habits, jaundice, nausea, vomiting, fever, or chills.

The patient had a history of rheumatoid arthritis, although the possibility of gout had not been excluded according to limited information obtained from another institution. The arthritis manifested as joint stiffness, most often in the morning, and difficulty walking due to discomfort in both knees that had been present for several months. This was successfully treated prior to admission with ibuprofen and indomethacin.

Past medical history also included a 100 pack-year history of cigarette smoking, which he had discontinued 25 years earlier. He had consumed two six-packs of beer per day for "many years," up to the present. The patient said he had a peptic ulcer, tarry stools, and had vomited blood in the past.

Physical examination at that time revealed a blood pressure of 158/78 mm Hg; pulse rate, 72/min; respiratory rate, 22/min; and temperature, 37.6°C. Mild-to-moderate ulnar deviation of the hands, without gross deformities, was noted. The wrists showed thickened synoviae and the hips, knees, and ankles were mildly stiff on examination, with a normal range of motion but with some degree of discomfort. The back was tender to deep pressure over the spinous processes of the seventh to tenth thoracic vertebrae. Chest examination demonstrated expiratory wheezing over the left upper lobe. On abdominal examination, the area of liver dullness extended for 15 cm in the midclavicular line; the liver edge was nodular and palpable 6 cm below the right costal margin. An ulcer, 2 cm in diameter, was noted over the lateral malleolus of the left ankle.

Laboratory examination showed moderate abnormalities of liver function tests: serum glutamic oxalic transaminase (SGOT), 205 units/L (normal range, 0–40 units/ L); serum alkaline phosphatase level, 761 units/L (normal range, 50–136 units/L); and serum gamma glutamyl transpeptidase (GGPT), 387 units/L (normal range, 15–85 units/L). Serum creatinine was 2.0 mg/dL; blood urea nitrogen (BUN), 43 mg/dL; and serum uric acid, 14 mg/dL. Complete blood count was normal except for mild hypochromic microcytic anemia (hematocrit, 40%). The urine analysis was normal.

A radiograph of the chest revealed a mass 5 cm in diameter in the left upper lobe, as well as a small density in the left lower lung field and a density near the anterior portion of the first right rib (Fig 1). Widening of the mediastinum was compatible with lymphadenopathy, and there was a compression fracture of the eighth thoracic vertebra.

A bronchoscopy was performed, directed to the left upper lobe lesion; biopsy revealed a poorly differentiated adenocarcinoma of the lung. Computed tomography (CT) of the abdomen, liver/spleen scan, bone scan, and films of the ribs and spine indicated metastases in the liver and in the 8th, 11th, and 12th thoracic vertebrae.

Radiotherapy and oncology consultants agreed that chemotherapy should be withheld; radiation therapy was recommended but the patient refused. He had been under the impression that the primary purpose of radiotherapy was to relieve pain; by this time his pain had subsided spontaneously. Throughout this entire admission the

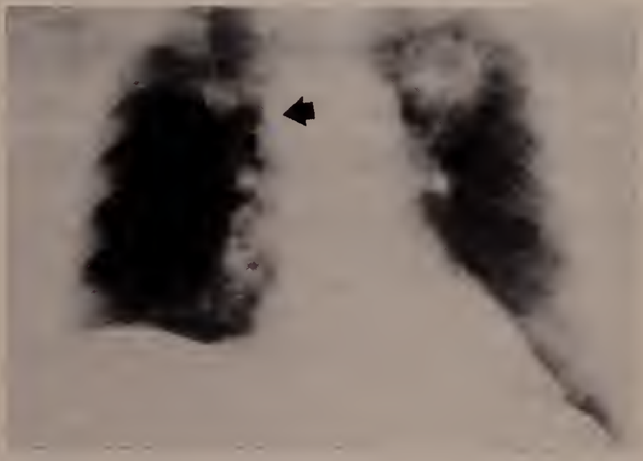


FIGURE 1. Chest film on admission to hospital showing large left upper lobe mass; azygos lymphadenopathy at arrow.

Based on the clinicopathologic conference (CPC) held at the Mary Imogene Bassett Hospital, Cooperstown, NY, on December 18, 1984.

Case Presentation: Mark Darrow, MD, Medical Resident; *Radiology Presentation*: Peter T. Wright, MD, Attending Radiologist; *Clinical Discussions:* Patrick N. Dwyer, MD, Attending Physician in Medicine—Hematology and Oncology; Bruce Harris, MD, Attending Neurosurgeon; *Pathology Presentation*: Harlan D. Alpern, MD, Pathologist-in-Chief.

Editor: JOHN T. FLYNN, MD

Address correspondence to Dr Flynn, Consulting Editor, *New York State Journal of Medicine,* PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

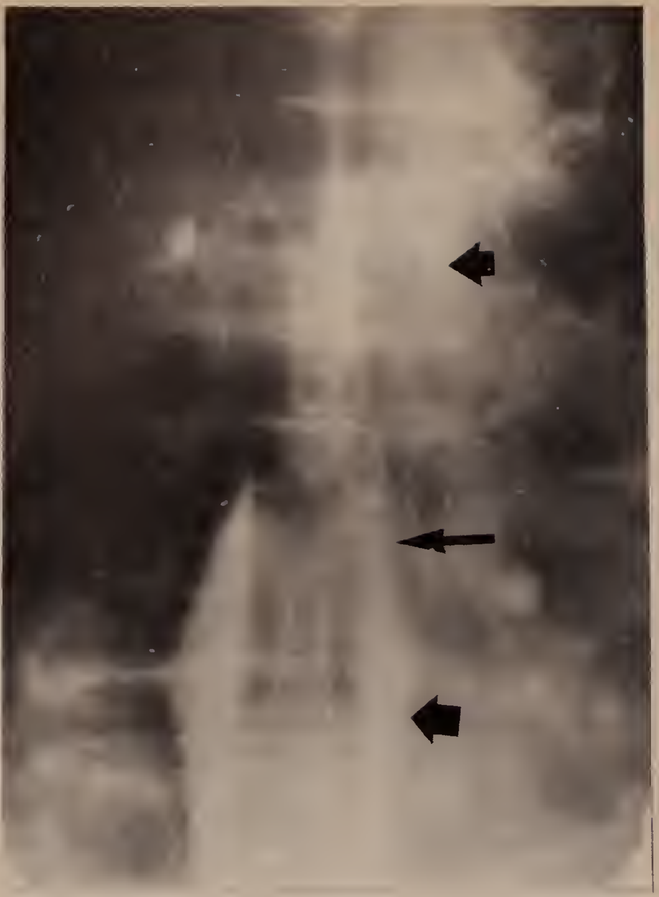


FIGURE 2. Emergency myelogram showing radiocontrast material **(lower arrow)** with cut-off due to extradural block **(middle arrow)** at level of the ninth thoracic vertebra; collapse of the eighth thoracic vertebra **(upper arrow)**.

nurses' notes reflected his inability to walk, and his need for a walker. There was no description of a specific weakness, but the entries indicated that some type of walking difficulty was present, probably caused by his chronic joint disability. The patient was discharged on the 13th hospital day. He was to be followed by the oncology division, with the understanding that radiation therapy might be initiated if back pain recurred.

One week after discharge the patient awakened with urinary incontinence and leg weakness that progressed over several hours. On readmission to the hospital the physical examination indicated paresis of the legs, except for internal rotation of the thighs at the hips. There was no sensory response to sharp or light touch below the level of the tenth thoracic dermatome. The deep tendon reflexes in the lower extremities were absent. The cerebrospinal fluid showed an elevation of protein to 590 mg/dL (normal range, 15–45 mg/dL), a glucose level of 69 mg/dL (normal range, 40–80 mg/dL), and 2 white blood cells and 25 red blood cells per ml^3. Spinal fluid cultures and cytologic studies were negative. An emergency myelogram demonstrated a complete block at the level of the eighth thoracic vertebra (Fig 2).

An emergency decompression laminectomy was performed on the evening of admission. Tumor tissue was identified grossly and later by histopathologic study in the epidural space, with cord compression at the level of the eighth thoracic vertebra. Recovery was slow, but by the second postoperative day there was improvement in the motion of toes and ankles. Later that evening the patient developed respiratory distress. He had an arterial blood Po_2 of 57 mm Hg, and thereafter suffered a pulmonary arrest. He was successfully resuscitated, and was transferred to the Intensive Care Unit. The patient was noted to be in atrial fibrillation that was reverted to normal sinus rhythm by electrical cardioversion. He failed to regain consciousness, however, and died later that same evening.

Dr Wright will review the initial radiographs as well as the films related to the laminectomy. I would like to know why the abdominal CT scan and the liver/spleen scan were both done as part of the search for metastases, and whether one of these tests has a particular advantage over the other in this regard.

RADIOLOGY PRESENTATION

Peter T. Wright, MD (Attending Radiologist): The posteroanterior film taken during the patient's initial admission shows a normal-appearing cardiac silhouette, and a large left upper lobe mass with irregular margins (Fig 1). The other nodules mentioned earlier include a small density that may represent a nipple shadow, and another density that may represent a pulmonary nodule or possibly a costochondral junction. We have no follow-up studies of these reported densities. There is widening of the mediastinum, especially in the right paratracheal region in the region of the azygos node. The lateral chest roentgenogram shows a compression fracture of the eighth thoracic vertebra. The radiologic findings thus suggest a primary pulmonary neoplasm with mediastinal and bony metastases.

The abdominal CT scan obtained shortly after the first chest radiographs showed focal areas of decreased density within the liver, and a normal-appearing pancreas. The left adrenal gland was abnormally rounded, suggesting the possibility of metastasis of the pulmonary lesion. A radionuclide liver/spleen scan done on the same day confirmed the presence of multiple focal hepatic lesions.

It is not clear from the requisition forms why both types of examinations were requested. Both of these scans would not be necessary in this circumstance. A patient with a known primary lesion and liver enlargement usually requires only a radionuclide liver/spleen scan to diagnose hepatic metastatic disease. If a patient has an enlarged liver and an unknown primary neoplasm, a CT scan would be better because of its capacity to delineate the pancreas, kidneys, and other abdominal organs. In this patient, since I assume the primary lesion was in the lung, a radionuclide liver/spleen scan would have been sufficient.

The bone scan performed several weeks later showed an area of increased activity on the posterior view of the spine—at approximately the 11th thoracic vertebra—and not in the area of the compression fracture seen on the lateral chest film. In addition, there are subtle diffuse abnormalities that involve many of the dorsal vertebrae and ribs. The rib lesions could not be documented on the plain radiographs; they are elongated and not the focal type of lesions which are usually seen as a consequence of trauma. It would appear then that the patient had diffuse metastases in the dorsal spine and ribs.

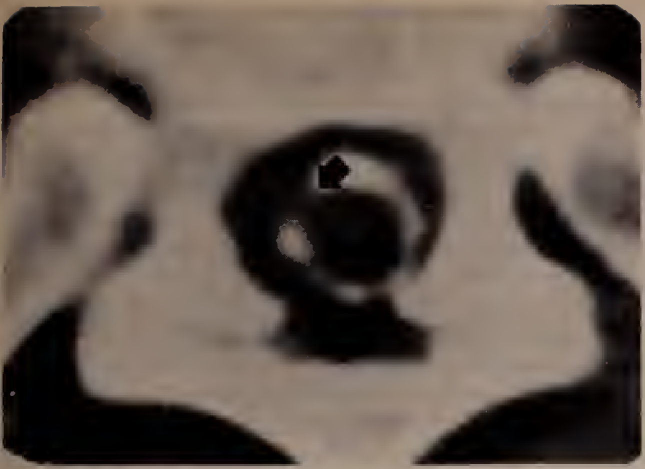


FIGURE 3. CT scan of the ninth thoracic vertebra with thinning of thecal sac (arrow) due to pressure of the extradural mass.

At the second admission, an emergency myelogram was done by instilling water-soluble contrast material into the lumbar region. A spot-film centered roughly at the thoracolumbar level showed both the compressed eighth thoracic vertebra, and an obstruction to the flow of the contrast material approximately at the level of the ninth thoracic vertebra (Fig 2). There was also thinning of the thecal sac in an extradural manner, suggesting extrinsic compression.

A CT scan through the region of abnormality was obtained immediately following the myelogram (Fig 3). Below this level, the spinal cord appeared normal, as outlined by metrizamide. At the level of the abnormality, the thecal sac was significantly thinned, and there was an abnormally increased density in the epidural region. Normally we would note the presence of fat in the epidural space, but in this case the epidural density is almost that of the cord itself. There is no contrast material evident at all further cephalad, and in the paravertebral regions there is abnormal soft-tissue density surrounding these dorsal vertebrae. These abnormalities are compatible with bony metastases with extension into the paravertebral region and into the epidural space, causing spinal cord and nerve root compression.

The final chest radiograph did not contribute further significant information; it confirmed the continued presence of the left upper lobe mass. The pulmonary vessels appeared prominent; there was no evidence of pulmonary infiltration.

In summary, the patient appeared to have a primary pulmonary neoplasm with mediastinal, liver, and bone metastases extending into the epidural space.

Dr Darrow: Dr Dwyer saw this patient in consultation on the first hospital admission. I would like him to discuss the following: what treatment can be offered to a patient with poorly differentiated adenocarcinoma of the lung; the therapeutic options that are appropriate for this patient; and the reasoning behind the eventual treatment decisions in this case.

Patrick N. Dwyer, MD (Attending Physician in Medicine—Hematology and Oncology): This patient illustrates a typical clinical course for a nonresectable adenocarcinoma of the lung. Most patients die, as this patient did, from advanced disseminated metastases; the prognosis in cases where the initial clinical presentation includes evidence of metastatic disease is limited. The survival rate is usually only several months.[1]

The methods of treatment for metastatic adenocarcinoma fall into two groups: combination chemotherapy and palliative radiation therapy. Since combination chemotherapy is a systemic form of treatment, it can reach all metastases, and potentially can treat multiple sites of neoplastic growth. The current regimens for non-small cell carcinoma of the lung are successful in approximately 30 to 40% of patients, and the response lasts for a median of six months.

This particular patient, however, presented many of the symptoms that indicate the likelihood of chemotherapy failure. We know that patients with poor performance status—who are not ambulatory and who cannot take care of themselves—respond poorly to chemotherapy.[2] Patients with weight loss, those who are elderly, and patients with multiple sites of disease also appear to respond less often. This particular patient also presented evidence of impaired renal function, which prevented us from using cisplatin. All of these considerations contraindicated the use of chemotherapy.

In such circumstances, radiation therapy is useful for such patients, primarily for palliation. Radiation therapy is used to treat a local site of disease which is either causing symptoms or threatening to cause difficulty. This patient's back pain, rib pain, or pain due to neoplastic involvement of the liver all could be construed as indications for radiation therapy. Hemoptysis or obstructive pneumonia related to the lung mass would also have been indications for palliative radiation therapy. Since hypoadrenalism is not commonly seen in carcinoma of the lung metastatic to the adrenal gland, such gland involvement would not in itself be a reason for radiotherapy to that area.

The prevention of serious symptoms is extremely important in the case of epidural cord compression. Had it been known that this patient's epidural tumor was on the verge of producing catastrophic symptoms, he would have benefitted from radiation therapy toward the end of his first hospital admission. At that time, however, his neurologic compromise was minimal.

Patients with cancer who have involvement of the spine—unless they have other disease sites that may shorten their lives—will ultimately develop epidural cord involvement, neurologic compromise, and paraplegia with high frequency.[3] This patient had advanced disease in other organs, especially in the liver. He was initially reluctant to undergo radiation therapy that was offered because of his complaint of pain in the spinal area; this he later denied. Additionally he was free of neurologic signs at that time. In many patients the physician must rely on their statements that they are developing leg weakness, sphincter disturbances, or changes in superficial sensation. Myelography should be recommended in these patients. If it had been performed on this patient during his first hospitalization it may have at least avoided this terminal clinical course which developed so soon after the laminectomy. I am sure that an earlier myelogram would have demonstrated a high degree of vertebral canal obstruction two or three weeks prior to the onset of his paraplegia; he could have been given radiation therapy at that time.

The alternative course for this patient with his poor physical performance status and advanced metastases was first to suggest radiation therapy, and secondly to reassess his condition periodically, and to offer palliative radiotherapy to the regions of local symptoms.

David Svahn, MD (Attending Physician): Did the patient have any symptoms at the end of the first hospital stay which would suggest that a myelogram should have been done?

Dr Dwyer: If this patient had been a young man with no liver involvement, who was complaining of back pain, and whose radiographs were positive for spinal metastases, one would most certainly recommend a myelogram. Pain is the first symptom of which a patient complains when epidural cord compression is developing, because the metastatic tumor usually localizes initially within the vertebrae. It is best to treat epidural compression before the onset of neurologic signs and symptoms. Once paraplegia develops or sensory changes are detected on physical examination, it is harder to reverse the process by surgical intervention. We need to be more aggressive in using myelography as a diagnostic tool, especially in those patients who have new onset of back pain due to spinal lesions.

Dr Darrow: This patient regained some degree of motor function one to two days after surgery. However, Dr Harris, how common is it to regain function after this kind of insult to the spinal cord? Also, what overall functional improvement might be expected after this type of surgical intervention?

Bruce Harris, MD (Attending Neurosurgeon): The neurologic outcome in patients with epidural metastases depends on the neurologic status at the time of surgery. Once there is complete cessation of cord function, with loss of both sensory and motor transmissions in the cord, recovery of any function is almost impossible. One can expect that the patient will probably not have any significant recovery of neurologic function. I do not know if this is due to vascular compromise, or simply to compression of the spinal cord.

This patient had residual adductor function prior to surgery, with some preserved posterior column sensory function. It was necessary to try to retain at least control of sphincter function, in order to avoid the nursing problems related to incontinence. We know that in this type of patient we will not cure the disease, and we will probably not even be able to restore ambulation with a walker.

If neurologic function at the time of the decompression is preserved, the patient can be assured of a better prognosis. One can frequently hope to restore the patient to an ambulatory status, if in fact there are only some vague sensory losses or minimal weakness in the lower extremities before surgery. One patient who was bedridden due to epidural prostatic carcinoma metastases not only was totally continent, but was able to walk back into the clinic one month after cord decompression.

I agree with Dr Dwyer that at the very earliest hint of cord dysfunction, the patient's condition must be investigated. Many patients with malignant disease will complain of back pain with or without metastases, but the next most important symptom which can alert the physician to the possibility of early cord compression is the complaint of vague sensory disturbances in the lower extremities and around the perineum. It may be difficult to document neurologic changes on physical examination with pinprick, touch, and position sensations intact. However, the patient is aware that something is wrong, and that is the signal to investigate the possibility of cord compression.

Dr Darrow: What information is the CT scan going to provide, beyond what the myelogram indicates?

Dr Harris: A myelogram is sufficient if there is a blockage or a large defect in the vertebral canal. If the neurologic examination is normal, it is unlikely that a complete block is present. However, a tumor in the vertebra, the paraspinal area, and the epidural space may be detected by a combination of CT and myelography; either test alone would not do so. CT may also help to define the extent of the lesion; otherwise myelographic contrast would have to be injected into a more cephalad location via cisternal puncture or a lateral C1-C2 route.

When treating with surgery, the intent is to decompress beyond the proximal and distal extent of the tumor. One vertebral space above and one space below the involvement is considered the minimal decompression.

Richard J. Horner, MD (Attending Physician in Medicine—Hematology and Oncology): I would like to add an optimistic note on this type of case. We followed two patients with multiple myeloma, one of whom was paraplegic on admission four and one half years ago, and the other patient was quadraplegic on admission one and one half years ago. Today both of these patients are walking and generally doing well. Even with significant neurologic deficit at diagnosis, the outlook is not hopeless. Successful treatment of cord compression can be followed by prolonged survival in patients with treatable malignancies, such as breast cancer or myeloma.

Dr Darrow: Is there a role for steroid treatment in this setting?

Dr Harris: Yes. In the early phase of radiotherapy for epidural compression by neoplasm, we frequently treat with steroids in order to reduce neural edema. Within 48 hours the patient is usually making tremendous gains. I do not know which treatment should get the credit for the improvement, but I suspect that early improvement is due to steroid therapy. In fact, there is a role for the use of steroids in all types of spinal cord problems, including trauma.

Dr Darrow: In reviewing this patient's chart, especially the clinical events during his last few hours, two possible causes of death were suggested. Pulmonary embolism seems most likely, followed by pneumonia as a second possible cause, although radiographs taken when the patient was transferred back to the Intensive Care Unit did not show any evidence of pneumonia.

CLINICAL DIAGNOSES

Adenocarcinoma of the lung with metastases to bone, epidural space (with spinal cord compression at T-8), liver, and mediastinal lymph nodes.

Rheumatoid arthritis (from history).

Pulmonary embolism.

Pathology Presentation

Harlan D. Alpern, MD (Pathologist-in-Chief): The postmortem examination provides an explanation for this patient's death, and I believe that surgical intervention played an indirect role in his demise.

Bronchial biopsy established the diagnosis of poorly-differentiated adenocarcinoma involving the lung. Nests of large polygonal malignant tumor cells with high nuclear-to-cytoplasmic ratios were noted within dense fibrous connective tissue in the bronchial mucosa. The nuclei were pleomorphic and contained coarse chromatin; the cytoplasm was amphophilic and microvacuolated. The periodic acid-Schiff with diastase stain showed some neutral mucin within the cells and along the luminal surfaces.

The incidence of adenocarcinoma has increased and it is now probably the most common primary tumor occuring in the lung, supplanting squamous carcinoma.[4] This reflects an increase in adenocarcinoma among both men and women with lung cancer; the high rate of adenocarcinoma among women has persisted.[4] The correlation between smoking and adenocarcinoma is weaker than for squamous or small-cell (oat-cell) carcinoma. In cases of adenocarcinoma in the lung, clinical and radiologic correlation is necessary to exclude the possibility of metastatic rather than a primary tumor. In this particular case, the autopsy confirmed that this adenocarcinoma was a primary lesion in the lung.

The biopsy also demonstrated endogenous lipoid pneumonia secondary to bronchiolar obstruction, with lipid-laden macrophages in the alveolar air spaces. This is a common finding in bronchogenic carcinoma. The bronchial-brush cytologic specimen indicated malignant tumor cells. Examination of paraspinal soft tissue removed at the time of the laminectomy also demonstrated poorly-differentiated metastatic adenocarcinoma.

At the time of autopsy, in the left upper lobe—corresponding to the radiologic lesion that Dr Wright demonstrated—there was a mass measuring 5 cm in maximum dimension with central scarring and necrosis (Fig 4). Low-power photomicrograph of the Gomori trichrome-stained section showed the blue-staining fibrous scar; it has been traditionally, though questionably, believed that tumors arise in such scars. Approximately 80% of scar cancers are adenocarcinomas. Higher power view demonstrates the extreme degree of pleomorphism of the tumor cells, with many mitotic figures and a striking degree of variation in nuclear size and shape. An Alcian blue stain demonstrated acid mucin within these tumor cells. There was extensive pulmonary endolymphatic and blood-vessel invasion (Fig 5). One of the nodules noted by Dr Wright in the lungs was a 1.5 cm lesion attached to the parietal pleura. On gross examination many mediastinal and pulmonary hilar nodes contained tumor and anthracotic pigment. Microscopic sections indicated tumor, some residual lymph node tissue, and anthracotic pigment.

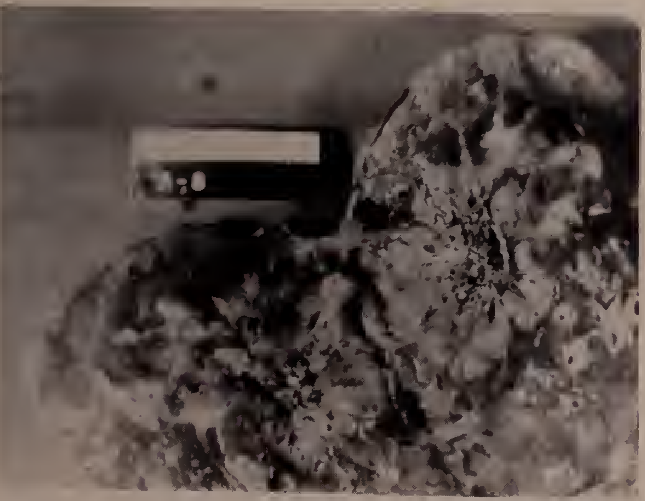


FIGURE 4. Cut surface of the upper lobe of the left lung showing a 5 cm adenocarcinoma arising from scar tissue.

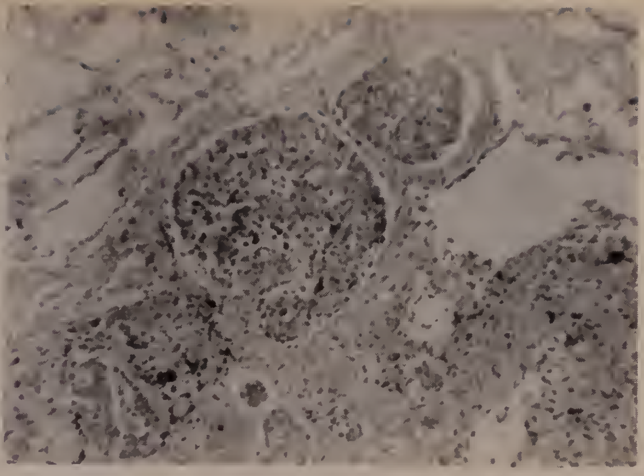


FIGURE 5. Extensive pulmonary endolymphatic metastases of the adenocarcinoma. (hematoxylin and eosin stain; original magnification × 40)

There was no gross tumor in the right lung, but its lymphatics were the site of metastatic spread. There were no tumor nodules found within the right lung, so that the radiologic interpretation of the density as perhaps nipple or rib shadow was correct.

The cause of death was extensive bronchopneumonia in the right lung, most likely a postoperative complication. Sheets of polymorphonuclear leukocytes were present in the alveoli. Bacterial cultures grew *Hemophilus influenzae* and *Escherichia coli.*

The liver had massive tumor involvement, with little residual normal hepatic tissue. After regional nodes, the liver is the most common site of metastasis of lung cancer, followed closely by adrenal involvement. Both adrenal glands in this patient contained metastatic tumor; one adrenal vein contained a large tumor embolus. However, as previously mentioned, extensive adrenal metastases rarely produce adrenal insufficiency.

Extensive tumor was noted on the spine on gross examination, and replaced both marrow and bone. The spinal canal was narrowed at the level of the eighth thoracic vertebra with compression of the spinal cord (Fig 6).

There were multiple subcutaneous nodules on the hands, fingers, and toes that were thought to be secondary to rheumatoid arthritis. The patient's serum uric acid was

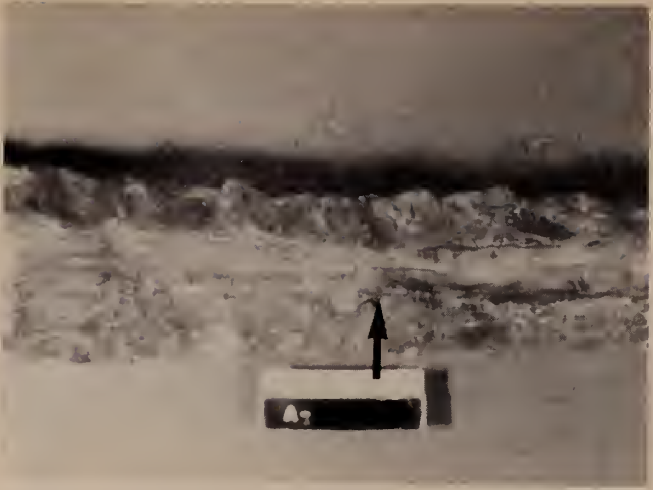


FIGURE 6. Constriction of the spinal canal **(arrow)** by metastatic adenocarcinoma in vertebrae.

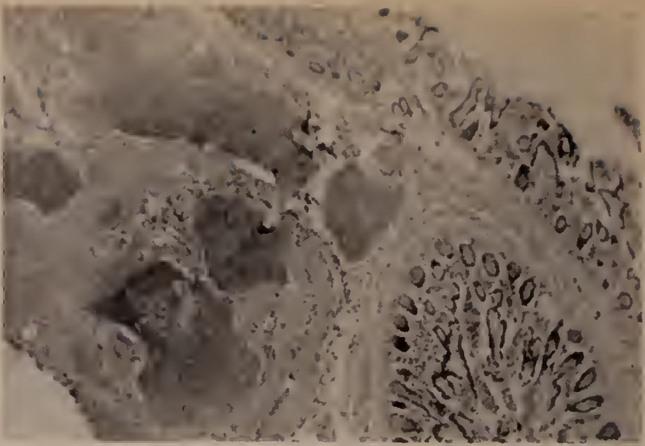


FIGURE 9. Dilated thin-walled vessels representing blue-rubber-bleb nevus in the submucosa of the small intestine. (hematoxylin and eosin stain; original magnification X 25)

elevated. Gross cross-section of these nodules showed a white chalky material characteristic of gouty tophi (Fig 7). There was no evidence of rheumatoid arthritis. The synovia demonstrated masses of spindle-shaped monosodium urate crystals with an intense and characteristic giant-cell reaction (Fig 8). In the early course of gout, polymorphonuclear leukocytes may infiltrate an area, but this is followed by a typical giant-cell reaction with granuloma formation. Urate crystals processed in alcohol retain the typical spindle-shaped fibrils and are birefringent when examined under polarized light.

The kidneys demonstrated moderately severe nephrosclerosis, accounting for the elevation of BUN and serum creatinine. Numerous sclerotic glomeruli were present, along with a mild chronic interstitial inflammatory infiltrate secondary to vascular disease. There was no evidence of uric acid in the kidneys nor in any of the other parenchymal organs.

This patient was thought to have had peptic ulcer disease, with a history of tarry stools and vomiting of blood; however, there was no evidence of either healed or active peptic ulcer. There were numerous hemangiomas in the small bowel, indicative of the blue-rubber-bleb nevus syndrome. This is a rare autosomal dominant disorder that involves hemangiomata in the gastrointestinal tract, skin, lips, and oral mucosa, though rarely in other organs.[5] This is the second case we have seen in the past eight years. These hemangiomas were most likely the source of the gastrointestinal bleeding rather than peptic ulcer disease, since there was no evidence of the latter at autopsy. It should be noted that the patient had a mild hypochromic microcytic anemia, which is common with chronic blood loss. Microscopic examination of these intestinal lesions showed numerous dilated thin-walled blood vessels (Fig 9). Careful examination revealed that the patient had vascular lesions on the lip and on the oral mucosa.

In summary, this patient had gout, rather than rheumatoid arthritis; and the blue-rubber-bleb nevus syndrome—most likely the source of bleeding rather than peptic ulcer disease. The adenocarcinoma of the left upper lobe had

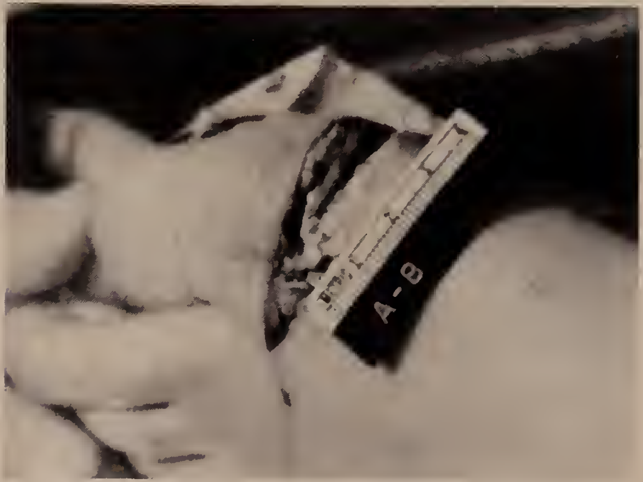


FIGURE 7. Chalky white gouty tophus in the metatarsal joint of the left great toe.

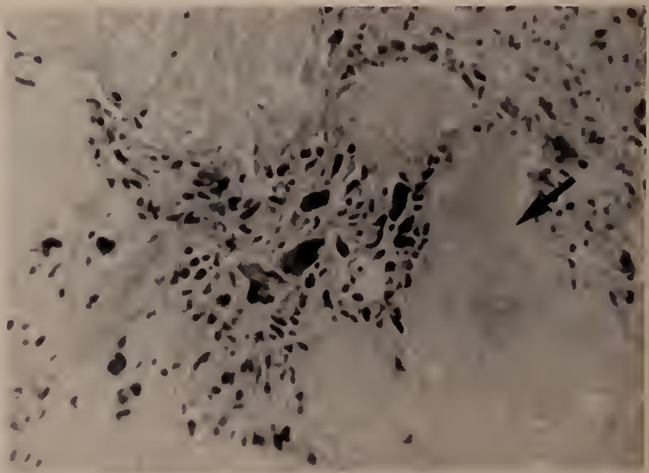


FIGURE 8. Masses of monosodium urate crystals of gout **(arrow)** with peripheral giant-cell reaction (hematoxylin and eosin stain; original magnification X 40)

metastasized widely, including to the spine with spinal cord compression. Death was attributed to *E. coli* and *Hemophilus influenzae* bronchopneumonia involving the right lung.

FINAL DIAGNOSES

Poorly-differentiated adenocarcinoma of the upper lobe of the left lung, with metastases to the vertebral column (with cord compression), liver, adrenals, right lung (intravascular), and multiple lymph node groups.

Acute bronchopneumonia *(Hemophilus influenzae* and *E. coli)* of the right lung.

Gouty synovitis with subcutaneous tophi.

Blue-rubber-bleb nevus syndrome involving the small intestine, lip, and oral mucosa.

Arterionephrosclerosis (kidneys).

REFERENCES

1. Lanzotti VJ, Thomas DR, Boyle LE, et al: Survival with inoperable lung cancer. *Cancer* 1977; 39:303-313.
2. Minna JD, Higgins GA, Glatstein EJ: Cancer of the lung, in DeVita VT Jr, et al (eds): Cancer—Principles and Practice of Oncology, ed 2. Philadelphia, Lippincott, 1985, pp 507-597.
3. Rodichok LD, Harper GR, Ruckdeschel JC, et al: Early diagnosis of spinal epidural metastases. *Am J Med* 1981; 70:1181-1188.
4. Valaitis J, Warren S, Gamble D: Increasing incidence of adenocarcinoma of the lung. *Cancer* 1981; 47:1042-1046.
5. Belsheim MR, Sullivan SN: Blue-rubber-bleb nevus syndrome. *Can J Surg* 1980; 23:274-275.

Pregnancy obtained by in vitro fertilization in an ambulatory-care surgical facility

ANDREW Y. SILVERMAN, MD, PhD, STEPHEN RENZIN, MD, OTTO RATZ, MD, FLORENCE BACK, STEVEN G. KAALI, MD, ROBERT LANDESMAN, MD

In vitro fertilization (IVF) and embryo transfer (ET) have become well established forms of treatment for human infertility over the last two years. The first successful pregnancy by this method was reported by Steptoe and Edwards in 1978.[1] With rare exception all of these programs are confined to tertiary care medical centers throughout the world.[2]

The Women's Medical Pavilion in Dobbs Ferry, NY is a free-standing surgical facility offering IVF and ET since January 1, 1985, consisting of a gynecologic endocrinologist, embryologist, laparoscopists, counselor, and a program coordinator. The facility is located one block away from a general hospital. The following is a case report of the first embryo transfer attempted here that resulted in an ongoing, viable intrauterine pregnancy.

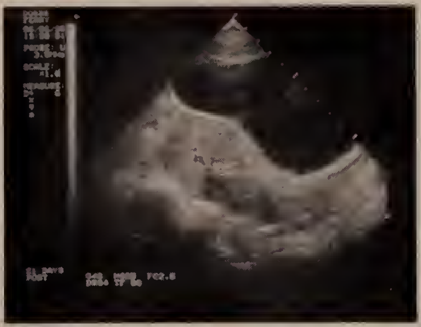


FIGURE 1. Pelvic sonographic examination 21 days after embryo transfer showing a 6-mm early gestational sac.

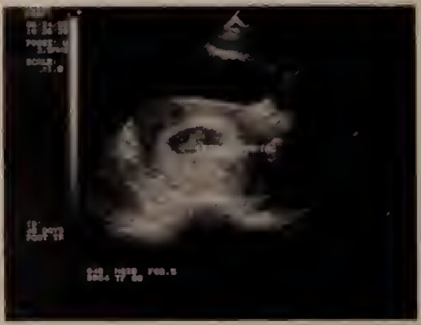


FIGURE 2. Pelvic sonographic examination 40 days after embryo transfer showing an intrauterine gestational sac measuring 35 × 21 × 18 mm.

CASE REPORT

A 33-year-old white woman had been married for 13 years with nine years of involuntary infertility; her husband was also 33 years old with unproven fertility. Her relevant past medical history includes a diagnostic laparoscopy in 1981 that demonstrated pelvic endometriosis. One year later she underwent a laparotomy; the endometriotic nodules were coagulated and a cystadenoma of the right ovary was resected. Repeat laparoscopy in 1984 revealed a right hydrosalpinx and left tubal adhesions without endometriosis. These findings correspond with a previous hysterosalpingogram. The rest of the couple's infertility studies were normal.

Psychologic assessment studies revealed that they had well-adapted attitudes towards their infertility, and that they possessed a realistic attitude towards the success of IVF-ET. Her last menstrual period was on April 29, 1985, and ovarian hyperstimulation was started with human menopausal gonadotropin (HMG) on day 3 of her cycle. She received a total of 21 ampules of menotropins and was monitored by daily serum estradiol, sonography, and cervical score.[3] On day 10 when the dominant follicle reached 20 mm and the estradiol level was greater than 600 pg/mL, 5,000 IU of human chorionic gonadotropin (HCG) was administered intramuscularly. Laparoscopy was carried out under general anesthesia in the out-patient operating room and two oocytes were removed by direct aspiration approximately 36 hours after HCG administration. A semen sample obtained prior to laparoscopy revealed that 76.9% of the sperm swelled in the hypoosmotic swelling test.[4] Six hours after incubation 50×10^3 washed sperm were applied to each mature egg. Fourteen hours after the sperm application only one of the oocytes showed signs of early fertilization (two pronuclei). After 26 hours of additional incubation, a two-cell embryo was observed while the other oocyte started to disintegrate. Forty-five hours after sperm application, a four-cell embryo was transferred under sonographic guidance[5] in an examining room adjacent to the tissue culture laboratory. One hour prior to embryo transfer, the patient had received 50 mg progesterone in oil intramuscularly, and 550 mg naproxen sodium per os. Both the patient's husband and a counselor were present at the time of the embryo transfer, making the experience a very positive one. The patient rested for one hour and left the office with 25 mg progesterone vaginal suppositories to be administered twice a day for ten weeks.

Since the embryo transfer, a counselor called and supported the patient daily. Ten days post-ET the serum HCG beta-subunit was less than 2 mIU/mL; however, the estradiol level was 91 pg/mL. Repeat serum tests four days later revealed an HCG beta-subunit of 61 mIU/mL and an estradiol level of 266 pg/mL, confirming an early gestation. The patient experienced several days of dark-colored spotting at the time of her expected menses. Her first post-ET pelvic ultrasound 21 days later showed a 6-mm early gestational sac (Fig 1). Her laboratory data including HCG, estradiol level, progesterone, 17-hydroxyprogesterone, and prolactin are presented in the Table. A repeat pelvic sonogram 40 days post-ET revealed an intrauterine gestational sac measuring 35 × 21 × 18 mm, with a clearly identifiable fetal heart beat in the developing fetus (Fig 2).

An obstetrical ultrasound obtained on September 9, 1985 (20 weeks' gestation) showed a cephalic presentation with a bipar-

---

From the Westchester Fertility Group, Dobbs Ferry, NY.

Address correspondence to Dr Silverman, Westchester Fertility Group, 88 Ashford Avenue, Dobbs Ferry, NY 10522.

TABLE. Laboratory Data for Month Following In Vitro Fertilization and Embryo Transfer

	HCG beta-subunit (mIU/mL)	HCG (mIU/mL)	Estradiol (pg/mL)	Progesterone (ng/mL)	17-OH Progesterone (ng/dL)	Prolactin (ng/mL)
5/24/85	2	negative	91.0	11.0	0.8	9.6
5/29/85	61	85	266.2	20.0	4.5	11.1
5/31/85	160	160	498.4	24.8	6.2	8.6
6/05/85	1,200	2,000	—	38.0	10.3	11.7
6/12/85	8,000	15,300	789.3	28.3	9.0	20.2
6/19/85	24,000	59,000	1,070.1	28.3	5.3	20.0
6/28/85	74,000	126,000	1,205.0	33.6	5.0	32.5

fetal diameter of 44 mm that corresponded to the patient's expected date of confinement. Parameters of fetal well-being were normal with normal anterior placenta formation.

DISCUSSION

All data confirm that this patient has a normally-developing, single fetus in utero. Her expected date of confinement is February 3, 1986. The patient has a 98% chance to complete her pregnancy with a 3% possibility of birth defects—the same as in the general population.[6] The current success rate of a live birth for IVF-ET is 13%,[7] compared to the natural conception rate of 31%.[8] Couples with infertility problems are already under stress and IVF-ET is a further demand on their time as well as their emotional, physical, and financial reserves. Considering the complexity of human reproduction, if the more stressful factors are eliminated, the outlook for success is brighter. An ambulatory approach provides equal quality and safety not only in laparoscopic[9] ovum retrieval but in other aspects of IVF-ET as well. If the overwhelming logistical burden can be eased it might result in higher pregnancy rates than medical center-based programs. Reduction in the cost by 50% will bring in vitro fertilization and embryo transfer within reach of the less affluent in the community.

Acknowledgment. The authors wish to thank I.J. Drekter of Drekter Medical Laboratories for performing the hormonal steroid analyses, and B. Saxena of New York Hospital-Cornell Medical Center for analyzing the data.

REFERENCES

1. Steptoe PC, Edwards RG, Purdy JM: Clinical aspects of pregnancies established with cleaving embryos grown in vitro. *Br J Obstet Gynecol* 1980; 87:757-768.
2. Feichtinger W, Kemeter P: In vitro fertilization as an outpatient/office procedure. *J In Vitro Fertil and Embryo Trans* 1984; 1:108.
3. Insler V, Melmed H, Eichenbrenner I, Serr DM, Lunenfeld B: The cervical score. *Int J Gynaecol Obstet* 1972; 10:223-228.
4. Jeyendran RS, Van der Ven HH, Perez-Pelaez M, Crabo BG, Zanenveld LJ: Development of an assay to assess the functional integrity of the human sperm membrane and its relationship to other semen characteristics. *J Reprod Fertil* 1984; 70:219-228.
5. Strickler RC, Christianson C, Crane JP, Curato A, Knight AB, Yang V: Ultrasound guidance for human embryo transfer. *Fertil Steril* 1985; 43:54-61.
6. Schlesselman JJ: How does one assess the risk of abnormalities from human in vitro fertilization? *Am J Obstet Gynecol* 1979; 135:135-148.
7. *Proceedings of the World Congress of In Vitro Fertilization and Embryo Transfer,* Helsinki, Finland, May 1984.
8. Silverman AY: The success rate of in vitro fertilization: what can the patient expect? *Am J Obstet Gynecol* 1982; 144:360-361.
9. Kaali SG, Landesman R: Tubal sterilization with the Falope ring in an ambulatory care facility. *NY State J Med* 1985 85:98-100.

Ultrasound diagnosis of interstitial pregnancy

DEBORAH J. COADY, MD, JON R. SNYDER, MD, STEVEN R. GOLDSTEIN, MD,
BALA R. SUBRAMANYAN, MD

Ectopic pregnancy accounts for a substantial number of maternal deaths each year. In New York City, 5% to 10% of maternal mortality is related to ectopic pregnancy.[1] Interstitial ectopic pregnancy, although relatively rare and comprising approximately 2.5% of all tubal gestations, causes more than its share of maternal mortality and morbidity.[2] In this form, implantation occurs in the intrauterine portion of the Fallopian tube where it passes through the myometrium. The greater distensibility and vascularity of this site lead to rupture at a later gestational age and rapid intraperitoneal hemorrhage. In the following case, an interstitial pregnancy in an asymptomatic patient is discussed.

CASE REPORT

A 40-year-old gravida 7, para 3 obese woman came to the gynecology clinic one week prior to admission for confirmation of pregnancy. Her last menstrual period was 13 weeks before, and a urine pregnancy test was positive. The patient had had a salpingectomy for a ruptured tubal pregnancy, and denied recent abdominal pain or vaginal bleeding. Because of her marked obesity, advanced maternal age, and history of ectopic pregnancy, an outpatient ultrasound examination was scheduled.

A transverse scan of the pelvis (Fig 1) revealed a slightly enlarged uterus with a central uterine cavity echo. An intact gestational sac attached to the uterus but eccentrically placed toward the left was seen. The sac was partially lined with placenta, and the myometrial mantle was incomplete at the sac's lateral edge. The fetal crown-rump length was consistent with 12 to 13 gestational weeks. Real-time ultrasound demonstrated fetal movement and heart activity. Longitudinal ultrasound scanning showed clearly the central uterine cavity echo. A diagnosis of probable left interstitial pregnancy was made, and the patient was admitted to the hospital for treatment.

Physical examination showed a markedly obese female in no distress, with a normal pulse and blood pressure. Her abdomen was not tender; her cervix was not tender to motion. The corpus and adnexa were not palpable due to the obesity, but no tenderness was elicited. Laboratory results were within normal limits, with a hematocrit of 41%.

From the Departments of Obstetrics and Gynecology (Drs Coady, Snyder, and Goldstein) and Radiology (Dr Subramanyan), New York University-Bellevue Medical Center, New York, NY.

Address correspondence to Dr Snyder, New York University Medical Center, 530 First Ave, Suite 5G, New York, NY 10016.

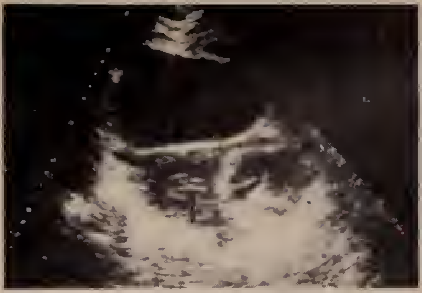


FIGURE 1. Transverse ultrasound scan showing the uterus with central uterine cavity echo (arrow), and eccentrically placed, intact gestational sac. Myometrial mantle is incomplete laterally (arrow head).

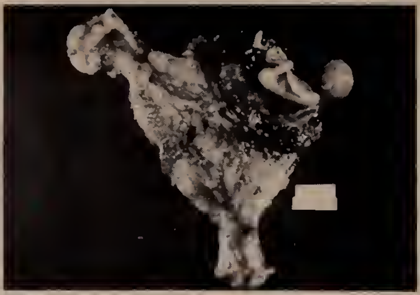


FIGURE 2. Pathology specimen showing opened uterus with gestational sac implanted within interstitial portion of the left tube. The endometrial cavity is separate and lined by thick decidua. Placenta is visible medially.

At exploratory laparotomy, a 14-week sized, highly vascularized uterine corpus was distorted by a tense, very thin-walled swelling of the left cornual area into which the left tube inserted. The right tube was absent, and there was no blood in the peritoneal cavity. A total abdominal hysterectomy was performed. The pathology specimen is shown in Figure 2. The gestational sac within the interstitial portion of the left tube was separate from the endometrial cavity. The fetal crown-rump length was 7.5 cm, consistent with a 13 to 14 week gestation. The patient's postoperative course was uncomplicated.

DISCUSSION

At the present time, the routine use of diagnostic ultrasound is not recommended for all pregnant patients.[3] However, in view of the 23% risk of a recurrent ectopic pregnancy after previous salpingectomy,[4] early ultrasound examination in this group of high-risk asymptomatic patients should be employed.

The diagnosis of interstitial pregnancy prior to rupture may be difficult. As seen in this case, the patient may be asymptomatic and the pelvic examination unremarkable. The usual finding of a tender, asymmetrically enlarged uterus without an adnexal mass is nonspecific. Sonographic signs of interstitial pregnancy are critical to the diagnosis. The differential diagnosis by ultrasound should include an angular pregnancy (an intrauterine pregnancy in which implantation has occurred at the extreme lateral angle of this uterine cavity),[5] a leiomyomatous uterus with distortion of the anatomy, and a more distal tubal pregnancy. The first two of these conditions represent intrauterine pregnancies in which the outcome is usually favorable.

It is important in all ectopic gestations to demonstrate an empty endometrial cavity by depicting the central linear uterine cavity echo on sonographic examination.[6,8] This echo is thought to result from the interface between the two layers of endometrium. An intrauterine gestation will obliterate this echo as early as three fetal weeks.[7]

The finding of an eccentrically placed gestational sac is important although nondiagnostic alone since it can also be seen in angular intrauterine pregnancy or leiomyomatous uteri. The significance of the presence or absence of a myometrial mantle around the gestational sac is demonstrated by Graham and Cooperberg.[9] In an intrauterine pregnancy, even if angular or distorted by myomata, the myometrium should completely encircle the gestation. In an interstitial pregnancy, however, the myometrial mantle is incomplete, with a thin rim visible laterally at the cornual end of the Fallopian tube. The presence of fluid in the cul-de-sac is a helpful finding in cases of rupture. This finding was, however, absent in the case reported here.

In summary, sonographic examination improved the outcome in this case by distinguishing an interstitial pregnancy prior to its inevitable rupture in an asymptomatic patient.

REFERENCES

1. *Summary of vital statistics.* Department of Health, The City of New York, 1980.
2. Pritchard JA, MacDonald PC, Gant NF (eds): *Williams Obstetrics,* ed 17. New York, Appleton-Century-Crofts, p 423-441.
3. *The use of diagnostic ultrasound imaging in pregnancy.* NIH Consensus Development Conference, 1984.
4. Kitchin JD, Wein RM, Nunley WC Jr, Thiagarajah S, Thornton WN Jr: Ectopic pregnancy: current clinical trends. *Am J Obstet Gynecol* 1979; 134:870-876.
5. Jansen RP, Elliott PM: Angular intrauterine pregnancy. *Obstet Gynecol* 1981; 58:167-175.
6. Weiner CP: The pseudogestational sac in ectopic pregnancy. *Am J Obstet Gynecol* 1981; 139:959-961.
7. Callen PW, De Martini WJ, Filly R: The central uterine cavity echo: a useful anatomic sign in the ultrasonographic evaluation of the female pelvis. *Radiology* 1979; 131:187-190.
8. Smith HJ, Hanken H, Brudelet PJ: Ultrasound diagnosis of interstitial pregnancy. *Acta Obstet Gynecol Scand* 1981; 60:413-416.
9. Graham M, Cooperberg PL: Ultrasound diagnosis of interstitial pregnancy: findings and pitfalls. *JCU* 1979; 7:433-437.

Type I herpes simplex esophagitis with candidal esophagitis in an immunocompetent host

JOHN V. YACONO, MD

Herpetic esophagitis in the immunocompetent host has been recently recognized[1-9] and is believed to occur more frequently than reported. Candidal esophagitis has been noted in both debilitated and healthy persons.[9] Esophagitis caused by herpes simplex virus type I (HSV-I) with concomitant esophageal moniliasis in the immunocompetent host is rare.[6] The following is a report of combined herpes simplex and candidal esophagitis in a healthy female confirmed by biopsy, culture, and serologic studies.

From the Department of Medicine, United Health Services, Wilson Hospital, Johnson City, NY.

Address correspondence to Dr Yacono, 913 River Oaks Dr, Pittsburgh, PA 15215.

CASE REPORT

A 67-year-old woman hospitalized with syncope and hypotension was found to have had a 16 lb weight loss in the three weeks since her husband's death. During that period she noted anorexia, heartburn, dysphagia, and fatigue.

Her heartburn was described as an intermittent retrosternal burning sensation associated with meals and temporarily relieved by antacid. The dysphagia had been present as intermittent difficulty in swallowing solids since upper esophageal webs were noted three years previously. The patient denied odynophagia, ingestion of corrosive substances, nausea, vomiting, hematochezia, or melena.

Past medical history included psoriasis, hiatal hernia, oral candidiasis, hypertension, chronic mild anemia, and depression since her husband's death. At that time she was taking hydrochlorothiazide, methyldopa, sulindac, and diazepam.

Physical examination revealed a pale, dehydrated woman in moderate distress. Her supine blood pressure was 100/60 mm Hg and decreased to 75/40 mm Hg when upright. There were concomitant orthostatic changes in her pulse. Her temperature was 39.4° C orally. Skin turgor was poor and no cutaneous lesions were present. Her tongue was dry with longitudinal furrows; a whitish exudate was evident in the posterior oropharynx. There was no lymphadenopathy. She had a grade 2/6 systolic ejection murmur at the lower left sternal border without radiation. Abdominal and rectal examinations were unremarkable. Her stool was negative on testing for occult blood.

Laboratory data on admission showed the following: hemoglobin, 10.9 g/dL (normal, 12–14 g/dL); hematocrit, 32.5% with normal red blood cell indices; white blood cell count, 19,800/μL, with 80% polymorphonuclear lymphocytes and 10% band forms; corrected reticulocyte count, 0.8%; and direct Coombs test was negative. Serum iron was 23 μg/dL (normal, 60–135 μg/dL); total iron-binding capacity, 144 μg/dL (normal, 250–350 μg/dL); and haptoglobin, 408 mg/dL (normal, 40–180 mg/dL). Serum electrolytes were as follows: sodium, 134 mEq/L; potassium, 2.4 mEq/L; chloride, 89 mEq/L, bicarbonate, 23 mEq/L; serum creatinine, 1.6 mg/dL; and nonfasting blood glucose, 147 mg/dL. Calcium was 7.4 mg/dL; cholesterol, 98 mg/dL, total protein, 5.0 g/dL; and albumin, 2.8 g/dL. Urine analysis showed greater than 100 white blood cells per high power field, 2 to 3 red blood cells per high power field, and numerous bacteria. Urine culture showed *Escherichia coli* sensitive to ampicillin. Electrocardiograph showed nonspecific changes and the chest radiograph was negative.

Intravenous hydration and supplemental potassium corrected the patient's fluid and electrolyte imbalance, and her urinary tract infection was treated with intravenous ampicillin. Her blood pressure returned to normal.

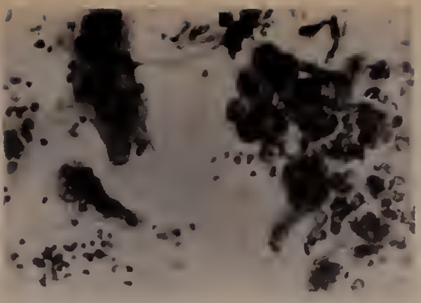


FIGURE. Photomicrograph of esophageal endoscopic biopsy demonstrating the characteristic multinucleated giant cells with ground glass appearance of the nuclei. (Hematoxylin and eosin stain; original magnification × 1,000)

An esophagram to evaluate her intermittent dysphagia showed linear filiform defects throughout the esophagus, suggestive of either small varices or diffuse ulcerations with raised normal mucosa. A hiatal hernia was seen, but no esophageal web was noted.

Upper gastrointestinal endoscopy was performed on the fifth day of hospitalization and a hiatal hernia was found. The entire distal third of the esophagus was coated with a thick whitish, though easily removable exudate. The underlying mucosa was found to be easily friable. Brushings and biopsies were taken for fungal, bacterial, and viral cultures and stains. Neither vesicles nor esophageal webs or rings were observed. The stomach and duodenum were normal.

A presumptive diagnosis of candidal esophagitis was made, and treatment with viscous xylocaine and nystatin lozenges was initiated.

The esophageal biopsy showed suppurative inflammation and granulation tissue. Epithelial cells showed prominent intranuclear eosinophilic inclusion bodies (Cowdry type A). There were numerous multinucleated giant cells present containing "ground glass" nuclei (Fig). HSV-1 grew abundantly in a culture using MRC-5 and MK african green media. Complement fixation titers for HSV-1 were 1:8 initially and rose to 1:128 after eleven days. Fungal culture grew *Candida albicans*.

The patient developed odynophagia for 48 hours after endoscopy. After that her condition improved. Her hemoglobin and hematocrit rose to 12.3 g/dL and 37.4%, respectively. Antinuclear antibody, rheumatoid factor, thyroxine, and thyroid stimulating hormone were normal. Serum protein electrophoresis at that time revealed the following: albumin, 2.85 g/dL; alpha$_1$ globulin, 0.37 g/dL; alpha$_2$ globulin, 0.89 g/dL; beta globulin, 0.75 g/dL; and total protein, 5.5 g/dL. The patient refused further test ing and repeat endoscopy. She was discharged with resolution of all symptoms after ten days and was in good health one year and one half later, with no recurrence of esophagitis.

DISCUSSION

Herpes simplex as a cause of esophagitis is well described in the immunocompromised host.[10-14] There have been reports of twelve cases of herpetic esophagitis where cancer or immunosuppression did not play a role.[1-8] This patient differs from previously reported cases in that she appears to have had combined herpetic and candidal esophagitis without immunologic dysfunction.

Accepted histologic criteria for herpetic infection[14] were fulfilled in this case with the identification of acute inflammatory mucosal changes, eosinophilic intranuclear inclusion bodies of Cowdry type A, and multinucleated giant cells with typical "ground glass" nuclei in the biopsy specimen. Herpes simplex lesions may be overlooked if the epithelium bordering the ulcer is not included in the biopsy microsections. This is the site where histologic signs of active herpes are most obvious.[14] The positive viral culture and the greater than four-fold rise in complement fixation titers for HSV-I further supported this diagnosis.

It has been documented that herpetic ulcers may become overgrown with *Candida*.[2,14] It is likely that injury to esophageal epithelium by HSV-I provides an environment in which candidiasis is easily established.[13] This patient's prior oral candidiasis may have provided the setting for a secondary infection. Alternatively, the growth of *Candida* in a culture may have represented oral contamination of the biopsy specimen, and this patient may have had pure herpetic esophagitis.

Radiographic characteristics of herpetic and candidal esophagitis while generally variable have been claimed to be distinct. The herpetic lesion shows discrete ulcers while the candidal lesion shows multiple ulcers and plaques.[15] The endoscopic appearance of herpes esophagitis has been described as discrete punched-out ulcerations of the mucosa. The visualization of vesicles is dependant on the stage of the lesion.[2] Candidal esophagitis appears as a whitish membrane overlying a friable and edematous mucosa.[9] These descriptions are consistent with the findings in the present case.

Stress is a recognized cause of recurrent herpes infections.[16] This patient's recent psychologic trauma may have played a role in her illness. Although thorough immunologic testing was not completed, her complete recovery and good health over the following year and one half suggests immunocompetence.[5,17]

The treatment of herpetic esophagitis in the immunocompetent host has been directed toward symptomatic relief. The recent appearance of relatively nontoxic antiviral agents[18] raises the question of their use in the immunocompetent host with herpetic esophagitis. Vidarabine has been shown to be effective in immunocompromised patients with localized varicella zoster virus infection.[19] Similarly, acyclovir has been shown to be effective in the prevention of herpes simplex virus infections in recipients of bone marrow transplants.[20] It would seem that vidarabine and acyclovir should be evaluated in the therapy of the severely incapacitated immunocompetent patient with herpetic esophagitis.

Early recognition and treatment of herpetic esophagitis in the immunocompetent host requires clinical awareness of its existence, manifestations, and means to diagnostic confirmation. Current information suggests that this entity may occur more frequently than reported. The symptoms of retrosternal chest pain and dysphagia/odynophagia may be seen in both herpetic and candidal esophagitis; and as demonstrated here both infections may occur simultaneously, even in the immunocompetent host. Proper diagnosis requires appropriate biopsy specimens and microsection preparation, as well as cultures. Therapy directed toward symptomatic relief may be augmented with antiviral agents. Until further evaluation is completed, their use should be reserved for patients either unresponsive to conservative medical management, or with significant hemorrhage.

REFERENCES

1. Depew WT, Prentice RS, Beck IT, Blakeman JM, DaCosta LR: Herpes simplex ulcerative esophagitis in a healthy subject. *Am J Gastroenterol* 1977; 68:381-385.
2. Owensby LC, Stammer JL: Esophagitis associated with herpes simplex infection in an immunocompetent host. *Gastroenterology* 1978; 74:1305-1306.
3. Springer DJ, DaCosta LR, Beck IT: A syndrome of acute self-limiting ulcerative esophagitis in young adults probably due to herpes simplex virus. *Dig Dis Sci* 1979; 24:535-539.
4. Solammadevi SV, Patwardhan R: Herpes esophagitis. *Am J Gastroenterol* 1982; 77:48-50.
5. Bastian JF, Kaufman IA: Herpes simplex esophagitis in a healthy 10-year-old boy. *J Pediatr* 1982; 100:426-427.
6. Brayko CM, Kozarek RA, Sanowski RA, Lanard BJ: Type I herpes simplex esophagitis with concomitant esophageal moniliasis. *J Clin Gastroenterol* 1982; 4:351-355.
7. DiPalma JA, Brady CE: Herpes simplex esophagitis in a nonimmunosuppressed host with gastroesophageal reflux. *Gastrointest Endosc* 1984; 30:24-25.
8. Deshmukh M, Shah R, McCallum RW: Experience with herpes esophagitis in otherwise healthy patients. *Am J Gastroenterol* 1984; 79:173-176.
9. Mathieson R, Dutta SK: Candida esophagitis. *Dig Dis Sci* 1983; 28:365-370.
10. Pearce JM, Dagradi A: Acute ulceration of esophagus with associated intranuclear inclusion bodies. *Arch Pathol* 1943; 35:889-897.
11. Berg JW: Esophageal herpes: complication of cancer therapy. *Cancer* 1955; 8:731-740.
12. Buss DH, Scharyj M: Herpesvirus infection of the esophagus and other visceral organs in adults. *Am J Med* 1979; 66:457-462.
13. Rosen P, Hajdu SI: Visceral herpesvirus infections in patients with cancer. *Am J Clin Pathol* 1971; 56:459-465.
14. Nash G, Ross JS: Herpetic esophagitis. A common cause of esophageal ulceration. *Hum Pathol* 1974; 5:339-345.
15. Levine MS, Laufer I, Kressel HY, Friedman HM: Herpes esophagitis. *AJR* 1981; 136:863-866.
16. Oleske JM, Minnetar AB: Viral and chlamydial infections, in Grieco MH (ed): *Infections in the Abnormal Host.* New York, Yorke Medical Books, 1980, p 401.
17. Fulginiti VS, Sieber OF: Immune mechanisms in infectious diseases, in Stiehm ER, Fulginiti VA (eds): *Immunologic Disorders in Infants and Children.* ed 2. Philadelphia, WB Saunders Co, 1980, pp 687-693.
18. Wong KK, Hirsch MS: Herpes virus infections in patients with neoplastic disease. Diagnosis and therapy. *Am J Med* 1984; 76:464-478.
19. Whitley RJ, Soong SJ, Dolin R, Betts R, Linnemann C Jr, Alford CA Jr: Early vidarabine therapy to control the complications of herpes zoster in immunosuppressed patients. *N Engl J Med* 1982; 307:971-975.
20. Saral R, Burns WH, Laskin OL, Santos GW, Lietman PS: Acyclovir prophylaxis of herpes-simplex-virus infections. *N Engl J Med* 1981; 305:63-67.

Farmer's lung in a 10-year-old boy

ALLAN S. CUNNINGHAM, MD, JOHN J. MAY, MD, JORDAN N. FINK, MD

Farmer's lung, caused by the exposure to organisms in moldy hay, is a serious and sometimes fatal occupational hazard of agricultural workers. The division of labor in the families of dairy farmers dictates that children participate in the chores, and it is customary for them to provide for the bedding in the stalls of dairy cows. Spoiled (moldy) hay is generally reserved for this purpose. It is most likely that other farmers also follow this custom that results in the exposure of children to moldy feed material. It is somewhat surprising, therefore, that Farmer's lung has rarely been reported in children.[1,2] One major pediatric textbook does not even index the disease[3]; this most likely reflects underdiagnosis as much as rarity of the condition.

A young boy was recently treated for Farmer's lung. This case calls attention to this life-threatening though treatable condition.

CASE REPORT

A 10-year-old boy came to the Mary Imogene Bassett Hospital pediatric clinic because of breathlessness that had gradually begun two weeks before. He had also lost approximately eight pounds. The patient had been in good health until one month previously when he was given the first of three antibiotics for bronchitis. Initially there was scanty yellow sputum production; fever, chills, aches, and acute attacks of breathlessness or wheezing during this period were all denied. A dry, hacking cough persisted, apparently unaffected by treatment, and breathlessness supervened.

The patient was afebrile but appeared pale and anxious; his breathing was shallow.

From the Departments of Pediatrics and Medicine, the Mary Imogene Bassett Hospital, Cooperstown, NY and the Department of Medicine, Medical College of Wisconsin, Milwaukee, WI.

This research was supported in part by grants from the US Public Health Service (AI 19104, HL 29319).

Address correspondence to Dr Cunningham, Attending Pediatrician, Department of Pediatrics, Bassett Hospital, Cooperstown, NY 13326.

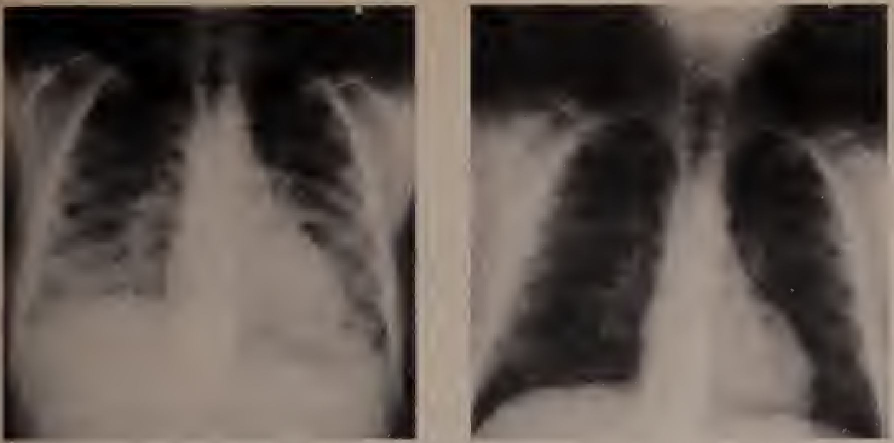


FIGURE. **Left.** Diffuse, reticulonodular opacities at the lung bases in the first set of films taken. **Right.** Normal films taken 14 weeks after exposure to hay and birds was terminated, and seven weeks after treatment with prednisone.

Despite encouragement he could not breathe deeply. Breath sounds were inaudible at the lung bases, but sparse crackles could be heard halfway up the chest posteriorly. A radiograph revealed diffuse, reticulonodular opacities at the lung bases (Fig).

Laboratory examination revealed a hematocrit of 50%, and the white blood cell count was 16,000/μL with 64% neutrophils in an otherwise normal differential count. Forced vital capacity was .32 L, and the technician commented that "he was unable to cooperate for testing." Arterial blood gas studies were not performed. The results of sequential pulmonary function testing are summarized in the Table.

At this time the patient's mother stated that they lived on a dairy farm. There was a chicken coop on the property and a parakeet in the living room, acquired four months previously. The patient regularly replaced the hay used for bedding in the barn stalls and occasionally worked in the chicken coop. It was then thought that he may have hypersensitivity pneumonitis due to the hay, parakeet, or chickens.

The patient was permitted to return home with the requests that close contact with the physicians be maintained, and that he stay out of the house until it was carefully cleaned and the parakeet removed. The family had a vacant trailer where he was housed temporarily. He was not allowed to visit the barn or chicken coop, and the family was instructed to wear separate coveralls in those buildings and remove them before returning to the house.

Within two days improvement was noted. By one week his forced vital capacity was 0.78 L and he was eating and sleeping better. After one month the patient had gained seven pounds and was active athletically. At that time his forced vital capacity was 1.07 L and there was marked clearing of the infiltrate in the chest radiograph, though his breathing was still visibly and audibly restricted. A tuberculin test was nonreactive.

The result of gel diffusion tests for precipitating antibody was reactive to *Saccaromonospora viridis, Thermoactinomyces vulgaris, Micropolyspora faeni,* and *Aspergillus fumigatus.* There was a weak reaction, judging by the intensity of the precipitin arc, to pigeon droppings. No reactions could be detected to *Thermoactinomyces candidus,* pigeon serum, parakeet droppings, chicken droppings, and chicken serum.

Based on the clinical and laboratory data, the patient was assumed to have Farmer's lung. To enhance clinical improvement, treatment with prednisone 40 mg daily in divided doses was initiated. After two weeks his breathing was unrestricted, he had gained an additional four pounds, and his forced vital capacity was 2.47 L. Prednisone was then given in single, tapering doses every other morning and gradually discontinued. He has remained well without a decline in pulmonary function. A chest radiograph performed three months later was normal (Fig). He returned to the farmhouse but did not visit the barn or the chicken coop. Work clothes and birds were not permitted in the house.

Table. Pulmonary Function Testing[*]

	Initial	After one week	After three weeks	After five weeks	After seven weeks	After nine months
Forced vital capacity (FVC)	.32 (11)[†]	.78 (26)	.90 (31)	1.07 (36)	2.47 (83)	2.59 (88)
Forced expiratory volume 1 sec (FEV_1)	.32 (12)	.69 (27)	.72 (28)	.82 (32)	1.94 (75)	2.02 (78)
FEV_1/FVC	1.00	.89	.80	.77	.79	.78
Total lung capacity[‡]	—	1.67 (44)	—	—	—	—
Residual volume	—	.77 (91)	—	—	—	—
Single breath diffusing capacity	—	1.2 (6)	—	—	—	—

All volumes are recorded in liters.

[*] Studies performed with a Collins Maxi-System, W. E. Collins, Braintree, MA.

[†] Percent of predicted normal value.

[‡] Determined by helium dilution.

DISCUSSION

Farmer's lung is a hypersensitivity pneumonitis caused by the inhalation of organic dust particles less than 5 μm in diameter. It is a nonasthmatic allergic reaction resulting from both immune complexes and cell-mediated immunity[4] against the organisms contained in moldy hay: *Saccaromonospora viridis, Thermoactinomyces vulgaris, Thermoactinomyces candidus,* and *Micropolyspora faeni.* The presence of serum precipitins against these antigens in patients with a history of breathlessness and exposure to old hay strongly suggests the diagnosis. The chest radiograph may show fine nodular shadowing. There may be a restrictive defect in pulmonary function with a reduction in vital capacity and impaired diffusion. Farmer's lung has rarely been reported in children. Bird-fancier's lung (pigeon-breeder's disease) is an identical hypersensitivity pneumonitis that is more common in the young.[5] These diseases in children are summarized in Phelan's textbook,[6] and in a more exhaustive manner in the textbook edited by Kendig and Chernick.[7]

Several important features of Farmer's lung and other types of hypersensitivity pneumonitis are exemplified by the case discussed. The relationship between symptoms and exposure to the pathogen is frequently obscure. The illness often develops without acute attacks of respiratory distress or fever, chills, and myalgias. Even when acute attacks occur there are several hours between antigen exposure and onset. The relationship is often recognized only after long delays in diagnosis. The chest radiograph may be normal in the presence of profound tissue disease and pulmonary dysfunction,[8] a fact that may contribute to errors and perhaps delay the diagnosis. Although Farmer's lung is a rural disease, it should be noted that individuals living in urban areas are not immune to similar disorders.[9,10]

Severe disease may occur with subtle

antigen exposure. No attempt was made to quantitate this boy's inhalation of antigen. His exposure to hay in the barn was regular though generally brief, and the environment was not unusually dusty. The family wore the same clothes in the house and barn, a practice that contaminated the home environment and added to his exposure. This factor alone is sometimes a sufficient cause of serious lung disease.[11] It is not known why other members of the family had not been ill, though their antigen exposure had been far more intense and prolonged. The boy's father could not be persuaded to visit the clinic or to be tested.

Treatment of Farmer's lung is simple and effective. By merely interrupting antigen exposure significant subjective and objective improvement was seen in this patient, and a short course of steroid therapy produced rapid objective benefit. Moreover, treatment can be instituted even when the causal antigen is not known with certainty, as seen in this case. When the clinical presentation is less clear, other diseases must be considered and a more exhaustive evaluation pursued.

It should be remembered, however, that fatalities from hypersensitivity pneumonitis do occur in children[12] as well as in adults. Although this patient did not require hospitalization, he appeared ill and had an alarming reduction in vital capacity to 11% of normal. Some cases of idiopathic pulmonary fibrosis begin with the inhalation of avoidable antigens[13] such as those found in moldy hay and animals. This patient and his family were advised that he should avoid future exposure to both hay and domestic birds.

Acknowledgement. The authors would like to thank Thelma Cortright, F.E. Hargreave, MD, and J. Dolovich, MD, for their assistance in the preparation of this manuscript.

REFERENCES

1. Heersma JR, Emanuel DA, Wenzel FJ, Gray RL: Farmer's lung in a 10-year-old girl. *J Pediatr* 1969; 75:704-706.
2. Hughes WF, Mattimore JM, Arbesman CE: Farmer's lung in an adolescent boy. *Am J Dis Child* 1969; 118:777-780.
3. Rudolph AM (ed): *Pediatrics*, ed 17. Norwalk, Conn, Appleton-Century-Crofts, 1982.
4. Schatz M, Patterson R, Fink J: Immunologic lung disease. *N Engl J Med* 1979; 300:1310-1320.
5. Cunningham AS, Fink JN, Schlueter DP: Childhood hypersensitivity pneumonitis due to dove antigens. *Pediatrics* 1976; 58:436-442.
6. Phelan PD, Landau LE, Olinsky A: *Respiratory Illness in Children*, ed 2. Oxford, Blackwell Scientific Publications, 1982, pp 379-380.
7. Kendig EL Jr, Chernick V: *Disorders of the Respiratory Tract in Children.* ed 3. Philadelphia, W.B. Saunders, 1977.
8. Epler GR, McLoud TC, Gaensler EA, Mikus JP, Carrington CB: Normal chest roentgenograms in chronic diffuse infiltrative lung disease. *N Engl J Med* 1978; 298:934-939.
9. Korn DS, Florman AL, Gribetz I: Recurrent pneumonitis with hypersensitivity to hen litter. *JAMA* 1968; 205:44-45.
10. Fink JN, Banaszak EF, Thiede WH, et al: Interstitial pneumonitis due to hypersensitivity to an organism contaminating a heating system. *Ann Intern Med* 1971; 74:80-83.
11. Riley DJ, Saldana M: Pigeon breeder's lung. Subacute course and the importance of indirect exposure. *Am Rev Respir Dis* 1973; 107:456-460.
12. Vergesslich KA, Götz M, Kraft D: Vogelzüchterlunge mit Ubergang in letale fibrosierende Alveolitis. Dtsch Med Wochenschr 1983; 108:1238-1242.
13. Turner-Warwick M: A perspective view on widespread pulmonary fibrosis. *Br Med J* 1974; 2:371-376.

Cocaine-induced myocardial infarction

LAWRENCE GOULD, MD, CHITRA GOPALASWAMY, MD, CHANDRAKANT PATEL, MD,
ROBERT BETZU, MD

Cocaine, extracted from the leaves of *Erythroxylon coca*, is a potent local anesthetic and sympathomimetic agent with central nervous system stimulant effects. In small doses cocaine may slow the heart rate due to central vagal stimulation, but larger doses increase cardiac rate thereby elevating the blood pressure. The latter effects are caused by stimulation of the central and peripheral sympathetic nervous system.[1]

Cocaine acts indirectly as a vasoconstrictor.[1] This action is most likely due to its interference with norepinephrine uptake by the adrenergic nerve terminals, with resultant sensitization to catecholamines. Cocaine-induced coronary artery vasoconstriction, with resultant myocardial infarction (MI), has been reported.[2-6]

CASE REPORT

A 32-year-old man was admitted to the hospital because of chest pain of three hours duration. The patient stated that he had been in excellent health. He denied any history of hypertension, diabetes, lipid abnormalities, or family history of coronary artery disease. He had smoked one half of a pack of cigarettes daily for approximately five years. Four months prior to admission he had smoked a cocaine-tobacco cigarette for the first time. One hour later the patient developed chest pains that lasted for 15 minutes. He again smoked a cocaine-tobacco cigarette on the day of admission. Severe chest pains developed one hour later that persisted until the hospital admission. The patient discarded the cigarettes; therefore an analysis of their content could not be accomplished.

Physical examination revealed a blood pressure of 170/90 mm Hg, with a cardiac rate of 68/minute. The neck veins were flat. The lungs were clear to percussion and auscultation. Cardiac examination revealed an S_4 gallop. The abdomen was normal, and there was no peripheral edema. Several electrocardiograms showed changes consistent with an anterior-lateral wall MI (Fig). The diagnosis was supported by an increase in the creatine phosphokinase (CPK) concentration to 680 units/ml (normal range, 5-50 units/ml); MB isoenzyme of the CPK was also positive. The SGOT level was increased to 178 units/ml (normal range, 5-40 units/ml); the LDH rose to 522 units/ml (normal range, 200-680 units/ml).

The hospital course was uneventful. The patient's blood pressure returned to normal one day after admission. A gated-pool scan revealed akinesia of the apico-inferior region and hypokinesia of the antero-septal region, with extension into the inferior wall. Left ventricular ejection fraction was 69%, and right ventricular ejection fraction was 61%.

From the Department of Medicine, The Methodist Hospital, Brooklyn, NY.

Address correspondence to Dr Gould, Chief, Division of Cardiology, The Methodist Hospital, 506 Sixth St, Brooklyn, NY 11215.

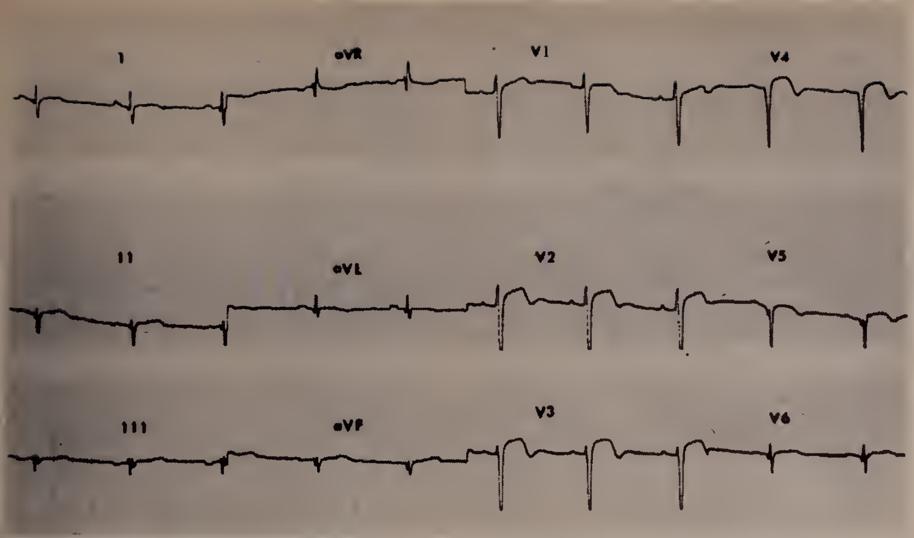


FIGURE. Electrocardiogram showing a myocardial infarction in a 32-year-old cocaine user.

The patient was scheduled for a coronary angiogram, but he left the hospital before the test could be performed. He was advised to avoid the use of cocaine, and was placed on nifedipine 10 mg three times a day.

DISCUSSION

Cocaine has become a popular drug in all socio-economic groups. In the past it was believed that casual cocaine use was not harmful. However, Wetli and Wright[7] reported 24 deaths directly caused by the toxic effects of cocaine. They observed that respiratory collapse and death occurred rapidly after intravenous injection of cocaine. Oral and nasal ingestion resulted in a symptom-free interval lasting as long as one hour, and followed suddenly by generalized seizures and eventually death. The data suggest that the rate of absorption, the peak blood concentration, and the prior use of cocaine all contribute to the possibility of a fatal reaction.

Cocaine abuse may produce cardiac arrhythmias. Benchimol and associates[8] reported that intravenous injection of cocaine produces an accelerated ventricular rhythm. Three modes of action may account for the enhanced automaticity: direct central sympathetic stimulation; alterations at the peripheral sympathetic nerve endings; and direct action on the impulse-forming and myocardial tissues.

Coleman and coworkers[2] first reported MI associated with cocaine use in a man with presumed coronary artery disease. However a coronary angiogram was not performed to verify this diagnosis. Schachne and associates[3] later reported on a 21-year-old man who had a subendocardial MI after inhaling 0.25 g of cocaine. He then had coronary angiograms that revealed normal coronary arteries. Pasternack and coworkers[4] described three patients who had MIs coincident with increased frequency of cocaine use. The presence of coronary artery disease and left ventricular dysfunction was documented by left ventriculography and coronary angiography in all three patients. The authors postulated that acute MI may have been induced by cocaine, with resultant sympathetic overstimulation, systemic hypertension, and tachycardia. These hemodynamic changes may increase myocardial ischemia and necrosis. The authors also discovered that cocaine could have exerted a direct vasoconstrictor effect that may have resulted in coronary artery spasm in a tightly stenotic artery.

Kossowsky and Lyon[5] reported on six young cocaine users who experienced MI. There was a close correlation between cocaine use and MI in two cases. In the other cases, such a correlation was not available. However, the degree of atherosclerotic blockage of their coronary arteries as demonstrated by either autopsy or angiography was minimal. Recently Howard and associates[6] reported on a 28-year-old woman who had an MI after sniffing cocaine. A serum toxicology screen by thin-layer chromatography with confirmation by gas chromatography revealed only cocaine. A urine screen by thin-layer chromatography with confirmation by immunoassay was positive only for the cocaine metabolite benzoylecgonine. A subsequent coronary angiogram revealed normal coronary arteries, and an ergonovine challenge test did not induce coronary spasm.

The etiologic mechanisms that produced the MI after cocaine use have not been firmly established. Coronary artery spasm has not been documented in any of the case reports available and there has been no clinical evidence of variant angina. It is thought that agents commonly used as adulterants in illegally-obtained cocaine may be responsible for the pathogenesis of this drug.

In the case reported here, cocaine-tobacco cigarettes caused chest pain on two separate occasions. Thus it appears reasonable to distinguish cocaine as the provocative agent that led to the MI. Unfortunately, coronary angiograms were not obtained in this patient. However, in view of the experience in the literature, it appears that cocaine may cause MI in patients with or without coronary artery disease. The clinician should be alerted to this potential complication. Further studies are necessary, however, to determine the prevalence of myocardial ischemia or MI associated with cocaine use.

REFERENCES

1. Ritchie JM, Cohen J: Local anesthetics, in Goodman LS, Gilman A: *The Pharmacologic Basis of Therapeutics.* ed 5. New York, Macmillan, 1975, pp 379-401.

2. Coleman DL, Ross TF, Naughton JL: Myocardial ischemia and infarction related to recreational cocaine use. *West J Med* 1982; 136:444-446.

3. Schachne JS, Roberts BH, Thompson PD: Coronary-artery spasm and myocardial infarction associated with cocaine use. *New Engl J Med* 1984; 310:1665-1666.

4. Pasternack PF, Colvin SB, Baumann FG: Cocaine-induced angina pectoris and acute myocardial infarction in patients younger than 40 years. *Am J Cardiol* 1985; 55:847.

5. Kossowsky WA, Lyon AF: Cocaine and acute myocardial infarction. A probable connection. *Chest* 1984; 86:729-731.

6. Howard RE, Hueter DC, Davis GD: Acute myocardial infarction following cocaine abuse in a young woman with normal coronary arteries. *JAMA* 1985; 254:95-96.

7. Wetli CV, Wright RK: Death caused by recreational cocaine use. *JAMA* 1979; 241:2519-2522.

8. Benchimol A, Bartall H, Desser KB: Accelerated ventricular rhythm and cocaine abuse. *Ann Intern Med* 1978; 88:519-520.

This section is designed to offer rapid publication (usually within 1-3 months) of new clinical, epidemiologic, and laboratory observations, as well as of comments on articles published in the Journal *or on any topic related to the role of the physician. Letters should be typed double-spaced and should include the professional affiliation, highest academic degree, address, and signature of each author. Preference is given to letters not exceeding 450 words. All letters are personally reviewed and acknowledged by the Editor. Address correspondence to Editor,* New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042.*

Spontaneous complete remission in acute promyelocytic leukemia

TO THE EDITOR: The recent article by Raza et al[1] on the occurrence of a spontaneous partial remission in a patient with acute promyelocytic leukemia (APL) prompts us to recall a similar case of APL with a more favorable outcome. This particular case was discovered during a retrospective review of adult acute leukemia in Rochester, NY[2]; however, the case was not included in the analysis and therefore has not been published previously.

Case Report. A 28-year-old man was admitted to the hospital in January 1967 with a several-week history of frequent episodes of epistaxis, a pale complexion, and easy bruising. Pertinent physical findings included petechiae on his right forearm, bilateral ecchymoses on his lower extremeties, bilateral conjunctival hemorrhages, and scattered cervical and supraclavicular lymphadenopathy.

Laboratory examination revealed a white blood count of 1.2 × 10^9/L with 23% blast cells; hemoglobin, 7.0 g/dL; and platelet count, 48 × 10^3/L. The Lee-White whole blood clotting time and fibrinogen levels were normal, but the prothrombin time was increased to 15.5 seconds (control, 13.0 seconds). Bone marrow aspirate showed the marrow to be hypercellular with 49% heavily granulated leukemic promyelocytes containing numerous Auer rods (Fig 1).

The patient was started on prednisone 30 mg and 6-mercaptopurine (6-MP) 200 mg daily, and was given a four-unit whole blood transfusion. After two weeks of treatment with prednisone and 6-MP, a repeat bone marrow examination was unchanged, and the patient was discharged. During the next month the patient remained granulocytopenic, thrombocytopenic, and anemic with no bone marrow change despite continued treatment with prednisone and 6-MP, transfusion with 11 units of whole blood, and two hospitalizations.

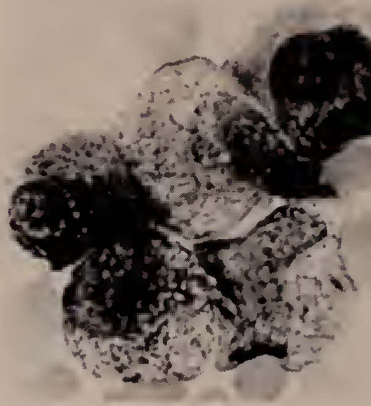


FIGURE 1. Bone marrow aspirate demonstrating abnormal heavily granulated malignant promyelocytes with Auer rods.

On the patient's last hospitalization, he was admitted in what was described as a "preterminal condition" with lethargy, a temperature of 104.6° F, nausea, vomiting, diffuse petechiae, and induration in the right axilla. White blood cell count at that time was 0.250 × 10^9/L with a platelet count of 16 × 10^3/L. Treatment with 6-MP was discontinued and broad spectrum antibiotics were administered. Three days after admission, two blood cultures were positive for Klebsiella species with evidence of a localizing infection in the right axilla. Antibiotics were continued and two units of whole blood were transfused.

Seven days after admission dramatic improvement occurred as well as an increased white cell count to 1.050 × 10^9/L and platelet count to 60 × 10^3/L. On the ninth hospital day, Klebsiella was cultured from a purulent drainage from the right axilla. During the remainder of the 20-day hospitalization the right axilla abscess cleared with complete normalization of the blood counts, including absence of any blast cells in the peripheral blood. Prior to discharge the patient refused a follow up bone marrow examination. The patient was lost to follow-up until this author came across his file and contacted him in May 1975. Physical examination and blood counts were normal, and the patient refused additional care.

Discussion. Spontaneous remission, whether partial or complete, of acute leukemia is rare. Prolonged survival, as in this case of more than eight years, is even more unusual. As noted by Raza et al,[1] these spontaneous remissions are usually preceded by severe life-threatening infections and, as recently suggested by Ifrah et al,[3] by blood transfusions. In the case described here, dramatic clinical and laboratory improvement occurred four days after Klebsiella bacteremia was documented. In addition, the patient was transfused with a total 17 units of whole blood over the two month history of his disease. It seems unlikely that the treatment with prednisone and 6-MP had any impact on the patient's disease since serial bone marrow examinations showed no change with more than one month of therapy.

Although the present technology for detecting cytogenetic abnormalities in leukemic cells was not available in 1967, the morphology of the bone marrow and peripheral blood was clearly that of APL. Despite the fact that there has been no follow-up, the clinical course of his disease remains fascinating. Indeed, cases such as this serve as a reminder that the unpredictable may occur and that not every case fits neatly into survival curves.

ROBERT E. ENCK, MD
Director of Oncology
Regional Cancer Center at
Lourdes Hospital
169 Riverside Drive
Binghamton, NY 13905

1. Raza A, Gill LM, Rakowski I, Preisler HD: Spontaneous remission in acute nonlymphocytic leukemia. *NY State J Med* 1985; 85:269-270.
2. Enck RE, Bauman AW, Bennett JM: Adult acute leukemia. The Rochester (NY) Experience. *Arch Intern Med* 1976; 136:1256-1261.
3. Ifrah N, James JM, Viguie F, Marie JP, Zittoun R: Spontaneous remission in adult acute leukemia. *Cancer* 1985; 56:1187-1190.

The physician/participant in war

TO THE EDITOR: I have read the correspondence in the Letters to the Editor column of the *New York State Journal of Medicine*, dealing with the role of the physician when called into military service (1985; 85:484). I agree completely with the sentiments of Dr Robert Rothenberg. I also was a physician who served in the military in an "unpopular" war—spending a year in the Republic of Vietnam as an orthopedic surgeon. My recollections, sentiments, and views are identical to those of Dr Rothenberg. I believe that I speak for the majority of my generation who served in Vietnam as physicians that this was a job that had to be done, and that we did, indeed, derive some satisfaction from rendering treatment to American boys who were wounded. I never had to shoot at anyone; neither did most of my fellow physicians in Vietnam. The satisfaction that we felt was not necessarily caused by performing brilliant or heroic surgery, but by treating American casualties. I further agree with Dr Rothenberg that satisfaction has absolutely nothing to do with being male or female. I believe that the majority of nurses in military hospitals in Vietnam felt the same way, whether they were men or women.

Unfortunately, war has become an established part of human history and as long as there will be wars, there will also be a need for physicians and surgeons to treat the casualties. The military physician's concern for the care of the injured soldier or non-combatant should be the same regardless of what his personal feelings are about the righteousness, or unrighteousness, of war.

RONALD E. ROSENTHAL, MD
Chief, Division of
Trauma Surgery
Department of Orthopaedic Surgery
Long Island Jewish-
Hillside Medical Center
1554 Northern Boulevard
Manhasset, NY 11030

Placental pseudotumor of the uterus: an iatrogenic disease?

TO THE EDITOR: I read with great interest the case report by Dr Badawy and colleagues on uterine tumor due to placenta increta (1985; 85:512-513). In my opinion neither the clinical picture nor the pathologic findings of this case indicate placenta increta.[1,2] I had a case similar to the one reported and I would like to offer an alternate explanation for the formation of the uterine "tumor."

Our patient was a 34-year-old woman with persistent vaginal bleeding and an enlarged, nodular uterus six weeks after endometrial curretage for an incomplete abortion.[3] The clinical impression was also that of uterine myomas, and a hysterectomy was performed. Within the right cornual myometrium there were two hemorrhagic "tumors" that revealed fibrinohemorrhagic meshwork on histologic examination that contained scattered necrotic chorionic villi. We postulated that these masses had resulted from iatrogenic instrumental implantation of the placental villi at the time of endometrial scraping by an ill-directed curette that had penetrated deeply into the myometrium.

It seems plausible that a similar event may have occurred in this patient during the piecemeal removal of her placenta by curettage when she was 21 weeks pregnant. The diagnosis of placenta accreta, although suggested clinically, was apparently never proven on microscopic examination by demonstrating myometrial fragments attached to the placental villi. The histopathologic findings of a necrotic area with ghost-like villi, hemosiderin-laden macrophages, foreign body reaction, fibrosis, and calcifications are compatible with the healing of an old traumatic injury, such as the iatrogenic implantation of the villi by a curette. Furthermore, what the hysterosalpingogram might have actually demonstrated is the tract leading from the endometrial cavity to the fundal mass made by the curette. Parenthetically, the monitoring of this patient with human chorionic gonadotropin levels would not be effective because the villi in the myometrium were necrotic.

Implantation of fetal tissue in the uterus secondary to curetting has been reported in the literature.[4,5] We believe that similar iatrogenic implantation of chorionic villi has occurred in the present case, as in ours, resulting in the formation of this intramyometrial placental pseudotumor.

MARIAN WAXMAN, MD
Department of Pathology
St. Vincent's Hospital and
Medical Center
153 West 11th St
New York, NY 10011

1. Luke RK, Sharpe JW, Greene RR: Placenta accreta: the adherent or invasive placenta. *Am J Obstet Gynecol* 1966; 95:660-668.
2. Breen JL, Neubecker R, Gregori CA, Franklin JE Jr: Placenta accreta, increta, and percreta. A survey of 40 cases. *Obstet Gynecol* 1977; 49:43-47.
3. Waxman M, Pardanani R: Implanted placental tissue simulating uterine fibroids ('inverted placental polyp'). *Diagn Gynecol Obstet* 1980; 2:287-289.
4. Newton CW III, Abell MR: Iatrogenic fetal implants. *Obstet Gynecol* 1972; 40:686-691.
5. Grönroos M, Meurman L, Kahra K: Proliferating glia and other heterotopic tissues in the uterus: fetal homografts? *Obstet Gynecol* 1983; 61:261-266.

In reply: Dr Waxman's letter raises several points with regard to the diagnosis and cause of a uterine tumor due to placenta increta. The diagnosis of placenta increta in the reported case was based on three facts:

- The history of an adherent placenta in the last pregnancy that required uterine curettage.
- During surgery a circumscribed mass measuring 4.5 × 3.0 × 2.5 cm was removed from the right corner of the uterus. This mass was intramural, surrounded with uterine musculature, and protruded into the endometrial cavity.
- On microscopic examination both smooth muscle fibers and chorionic villi were noted. This demonstrated the presence of chorionic villi in direct contact with myometrial muscle fibers and also penetration inside the wall of the uterus.

In discussing the cause of placenta increta, Dr Waxman and colleagues in their publication suggested a traumatic implantation of chorionic tissue inside the uterine wall by a curette. This is difficult to prove since clinical perforation of the uterus was not noted in this case. Also perforation of the uterus usually heals with no problems. Extensive curettage with myometrial injury results in uterine synechiae with intrauterine fiberotic lesions. In the present case, it is much easier to suggest either a poor endometrium or the presence of intrauterine synechiae as a cause of placenta increta since this patient had previous uterine curettage on two occasions following her miscarriages. It is known that the post partum and post abortal endometrium is vulnerable to trauma; placenta accreta is known to occur in patients with history of Asherman's syndrome.

The study of the beta subunit of human chorionic gonadotropin levels is found to be valuable in the follow-up of patients with ectopic pregnancy, both before and after conservative surgery. Also it is a valuable method in studying the course of patients with molar pregnancies. Follow-up studies of the beta subunit of human chorionic gonadotropin levels are suggested in patients with placenta accreta who have had uterine curettage. The failure of declining titers or the persistence of these titers may suggest active chorionic tissue that may require treatment with chemotherapy.

SHAWKY BADAWY, MD
Reproductive Endocrinology Unit
Department of Obstetrics and Gynecology
Upstate Medical Center
Syracuse, NY 13210

Breast self-examination

TO THE EDITOR: Having recently attended an Update-Your-Medicine lecture at New York Hospital-Cornell Medical Center that reminded me of the complexity of discovering and treating breast cancer, I am emboldened to challenge the author of "Breast self-examination—an idea whose time has come" (1985; 85:482-483).

I have long suspected that self-examination may give a momentary sense of false security; false because few women are familiar with the inner structure of the breast, and are unlikely to do an adequate examination. But my reservations are on other grounds; as a psychiatrist. The breast is the focus of many emotional currents and anxieties: developmental problems of adolescence; fantasies; and mother-daughter, father-daughter, and other interrelationships often make the breast an emotional battleground, precipitating ongoing anxiety and disease. Add to this the preoccupation with appearance in our society, and the dissatisfaction with all but a 'perfect 10' appearance, and it becomes apparent that to ask already anxious women to be consciously preoccupied with this body part—and to regularly search for signs of pathology—is to seriously reduce their quality of life. The concern about appearance leading to minimal surgery in spite of risk confirms this view.

I am inclined to believe that a semiannual visit to the family practitioner or gynecologist (unless some chance discovery precipitates an earlier visit) places the burden of discovery where it belongs—on the doctor—and relieves to some degree the almost ever-present cloak of anxiety for the woman.

In this "year of breast self-examination" sponsored by Betty Ford, this view may be unpopular, but it cries for consideration.

NATALIE SHAINESS, MD
140 East 83rd St
New York, NY 10028

TO THE EDITOR: Despite much educational effort regarding breast self-examination, the compliance rate is exceedingly low.

Since 1980 I have instructed patients to wash their breasts daily with their bare hands (no wash cloth). The idea is to make it a routine procedure, such as brushing one's teeth or combing one's hair. Scalp and mouth lesions are often discovered by these daily exercises.

Due to greater compliance I believe that early breast lesions will be discovered far more quickly this way.

KEVIN M. O'GORMAN, MD
229 Main St
Hamburg, NY 14075

AIDS and the alleged Haitian connection

TO THE EDITOR: Since 1982 much has changed about the acquired immune deficiency syndrome (AIDS). The factors that cause the syndrome have been discovered and the case definition for national reporting has been revised. Epidemiologic studies have also shed light on the subject, and have caused the alleged Haitian link to be discarded. Because of the hysteria caused by AIDS and in the interest of the public, it is appropriate to hereby review the facts.

In April 1982 during the 18th Congress of the "French-speaking Physician of the American Hemisphere" held in Port-au-Prince, Liautaud and associates from the dermatology and pathology services of the State University Hospital of Haiti reported for the first time on 12 cases of Kaposi's sarcoma. Meanwhile, in the United States cases of AIDS among Haitians had been collected by the Center for Disease Control, and were later reported in two major medical journals.[1,2] The findings were vulgarized by the lay press, mistranslated, and misinterpreted.

For a while it had been popular belief that AIDS had originated in Haiti, and that being Haitian was a risk factor. In the words of a non-Haitian physician, "the American media, with the help of some of our colleagues, seems intent on establishing a connection between Haitians and AIDS, even though none may exist."[3] This stigma not only caused psycho-social damage to a particular ethnic group, but also worsened the AIDS hysteria. Much emotional reaction emerged from Haitian circles. However, as epidemiologic studies are presently being looked at, it has been found that Haitians are no more at risk than any other ethnic group.

The rate of HTLV-III seropositivity among Haitian immigrants was found to be less than 5% in New York City.[4] "From these data, it does not appear that being of Haitian extraction by itself, in isolation of other risk factors, increases the relative risk of being exposed to HTLV-III."[5] Corroborating this new finding, another major study had been conducted in Montreal where approximately 35,000 Haitians reside; 189 Canadians were compared to 189 Haitians matched for age, sex, neighborhood of residence, and with comparable demographic data including rate of employment and income. The Haitians, in comparison to non-Haitians, had no demonstrable abnormalities in cellular immune function.[6]

In view of the hysteria and fear caused by the AIDS epidemic, it is of great importance to publicize these reassuring data. As was sensed before,[7] some Haitians may be victims of AIDS. However, being Haitian is not a risk factor for AIDS, and Haiti is not the source of the disease. It was appropriate indeed that the Center for Disease Control had finally discontinued the previous separate listing for Haitian-born patients.[8]

JEAN-ROBERT LEONIDAS, MD
Clinical Assistant Professor of
Medicine
SUNY Downstate
967 Ocean Avenue
Brooklyn, NY 11226

1. Vieira J, Frank E, Spira TJ, Landesman SH: Acquired immune deficiency in Haitians: opportunistic infections in previously healthy Haitian immigrants. *N Engl J Med* 1983; 308:125-129.
2. Pitchenik AE, Fischl MA, Dickinson GH, et al: Opportunistic infections and Kaposi's sarcoma among Haitians: evidence of a new acquired immunodeficiency state. *Ann Intern Med* 1983; 98:277-284.
3. Greco RS: Haiti and the stigma of AIDS, letter. *Lancet* 1983; 2:515-516.
4. Frank E, et al: *Cancer Res*, in press.
5. Landesman SH, Ginzburg HM, Weiss SH: The AIDS epidemic. *N Engl J Med* 1985; 312:521-525.
6. Adrien A, Desrosiers M, Frappier-Davignon L, Spitzer WO, Dupuy JM: Immunologic findings in healthy Haitians in Montreal. *Can Med Assoc J* 1985; 133:401-406.
7. Leonidas JR, Hyppolite N: Haiti and the acquired immunodeficiency syndrome. *Ann Intern Med* 1983; 98:1020-1021.
8. Centers for Disease Control. Update: Acquired immunodeficiency syndrome—United States. *MMWR* 1985; 34:245-248.

Disseminated histoplasmosis in a man with AIDS

TO THE EDITOR: Disseminated histoplasmosis has been encountered only rarely in patients with the acquired immunodeficiency syndrome (AIDS) and the detection of histoplasma in the peripheral blood smear is unusual.[1,2] The following is a report of a case of AIDS initially noted to have disseminated histoplasmosis by routine examination of a Wright-Giemsa-stained peripheral blood smear.

Case Report. A 47-year-old homosexual man was found to have AIDS when first admitted to Cabrini Medical Center in April 1984. The patient had a fever, weight loss, skin rashes, leukopenia, a T4/T8 lymphocyte ratio of 0.69, *Pneumocystis carinii* pneumonia, oral thrush, and thoracic herpes zoster. He had a past history of gonococcal urethritis, hepatitis type B, intestinal amebiasis, and perianal herpes simplex. His subsequent clinical course included hepatomegaly, diarrhea caused by *Giardia lamblia*, and persistent fever due to *Mycobacterium avium-intracellulare* that was isolated from bone marrow and for which the patient was readmitted to the hospital in April 1985. He was transferred to Cabrini Hospice in June 1985 because he was considered moribund.

In July 1985 a peripheral blood smear obtained from this patient while in Cabrini Hospice showed intraleukocytic (Fig) and extracellular budding yeasts characteristic of *Histoplasma capsulatum*. Wright-Giemsa-stained smears of buffy-coat preparations of peripheral blood confirmed the diagnosis of disseminated histoplasmosis and special stains were considered unnecessary. Serum from this patient tested by the New York State Department of Health was negative for histoplasma serology. *Histoplasma capsulatum* was cultured first in the mycelial phase and then converted to its yeast phase from an antemortem blood specimen; the final report was available only postmortem. Since the patient was considered moribund, treatment for *Histoplasma capsulatum* was not administered. The patient died on July 30, 1985, and an autopsy could not be performed.

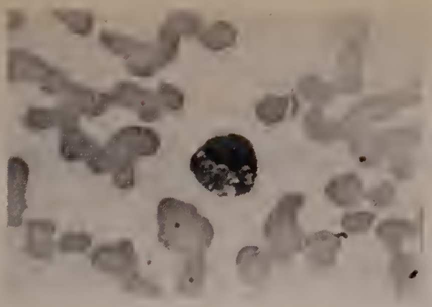


FIGURE. Peripheral blood smear showing intraleukocytic budding yeasts characteristic of *Histoplasma capsulatum.*

Discussion. Six patients in high-risk groups for AIDS who developed disseminated histoplasmosis while long-term residents of New York City were reported by Small and associates.[3] All of the patients were born in Puerto Rico, all were intravenous drug abusers, and one was homosexual. Our patient, born in Oregon, was a long-term resident of New York City and was not an intravenous drug abuser. Neither Oregon nor New York City are endemic for *Histoplasma capsulatum*. It appears that patients with AIDS may acquire histoplasmosis even if they reside in nonendemic areas.

In a patient with a positive serologic or virologic test for HTLV-III/LAV, disseminated histoplasmosis—not confined to the lungs or lymph nodes—diagnosis by culture, history, or antigen detection has now been included in the revised definition of AIDS by the Center for Disease Control.[4] However, the identification of *Histoplasma capsulatum* in a stained peripheral blood smear may make the initial diagnosis of disseminated histoplasmosis in patients with AIDS. Therefore, *Histoplasma capsulatum* should be considered when peripheral blood smears from these patients are routinely examined.

SELWYN J. BAPTIST, MD
JOHN B. MONTANA, MD
SHELDON B. ARDEN, PhD
LUCINA LEON
MARIE DUTEL
Cabrini Medical Center
227 East 19th St
New York, NY 10003

1. Bonner JR, Alexander WJ, Dismukes WE, et al: Disseminated histoplasmosis in patients with the acquired immune deficiency syndrome. *Arch Intern Med* 1984; 144:2178-2181.

2. Henochowicz S, Sahovic E, Pistole M, Rodrigues M, Macher A: Histoplasmosis diagnosed on peripheral blood smear from a patient with AIDS. *JAMA* 1985; 254:3148.

3. Small CB, Hewlett D, Duncanson FP, et al: The acquired immunodeficiency syndrome and disseminated histoplasmosis in a nonendemic area (abstr). International Conference on Acquired Immunodeficiency Syndrome (AIDS). Atlanta, Georgia, April 14-17, 1985, p 26.

4. Center for Disease Control. Revision of the case definition of acquired immunodeficiency syndrome for national reporting—United States. *MMWR* 1985; 34:373-375.

THE WORLD CIGARETTE PANDEMIC, PART II

Additional copies of this issue are available for $7.50 postpaid (foreign, add postage) from *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042, Tel: (516) 488-6100. (Reduced prices on bulk orders (foreign, add postage): 2-9 copies—$7.00 each; 10-24 copies—$6.50; 25-49 copies—$6.00; 50-99 copies—$5.50; 100-149 copies—$5.00; 150-500 copies—$4.00.)

A free copy of this issue will be sent with every new one-year subscription ($25 US + Canada, $30 foreign). Copies of *The Cigarette Underworld* (December, 1983 issue) are available for $9.95, plus $1.50 postage, from Lyle Stuart Inc, 120 Enterprise Ave, Secaucus, NJ 07094.

MSSNY Medical Liability Reform Fund Honor Roll

In the September issue the *Journal* began publishing the list of physicians who have generously contributed to the campaign to inform the public about the crisis in medical liability litigation. The names of additional contributors are listed below.

John W Abbuhl, MD
Awny A Abdou, MD
Moise Maurice Abitbol, MD
Albert M Aboody, MD
Arnold Aloysius Abramo, MD
Roy M Abramowitz, MD
Sebastian L Acea Nunez, MD
Albert James Addesa, MD
Milton Harris Adelman, MD
Alexandra Adler, MD
Michael Gregory Adragna, MD
William A Africano, MD
Araceli B Aguila, MD
Benjamin C Agustin, MD
Mir Manzur Ahmed, MD
Gloria Stone Aitken, MD
Judson K Albaugh, MD
Michael J Albom, MD
Eugen Albu, MD
Robert Johnson Alesbury, MD
Benjamin Hall Alexander, MD
Fakhry Younan Alexander, MD
Fred F Allison, MD
Lewis James Alrutz, MD
Reese Fell Alsop, MD
Casco Alston, MD
Michael J Altamura, MD
Aaron David Altman, MD
Flor Villena Alto, MD
Robert Sanford Amelkin, MD
Richard Ament, MD
Anastasios Anastasiades, MD
Charles Leon Anderson, MD
Jack David Anderson, MD
B Wesley Andrew, MD
Joseph Charles Andriola, MD
Carl Huber Andrus, MD
John Angerosa, MD
Bryce Chadwick Anthony, MD
Albert Anthony Apicelli, MD
Jesse Appel, MD
I Joseph Aprile, MD
Lydia S Aquino-Mapua, MD
John Arcidiacono, MD
Thomas George Argyros, MD
Daniel S Arick, MD
Sheppard Arluck, MD
Donald Joseph Armenia, MD
Victor T Arnao, MD
Marco A Arrascue, MD
Herman Robt Ash, MD
Philip Miller Ashman, MD
Charles Nejm Aswad, MD
Gabriela Magda Aszody, MD
Louis Albert Auletta, MD
Arthur Avedian, MD
Anthony M Avellanosa, MD
Howard Seymour Axelrod, MD
Alexander C Badin, MD
Alegria Depusoy Bahia, MD
Floyd Louis Bajjaly, MD
Daniel Clifton Baker, MD
Nicholas A Balaci, MD
Robert Jay Baliff, MD
Marvin Richard Ball, MD
Kye Hyon Bang, MD
Charles Selig Baraf, MD
Kenneth R Barasch, MD
Anthony Joseph Barbaro, MD
Philip A Barenfeld, MD
John Bernard Barnell, MD
Bruce Alan Barniville, MD
Lawrence Barnowsky, MD
Vincent J Barone, MD
Amilcar A Barreto, MD
Robert Edward Barrett, MD
A Sidney Barritt Jr., MD
Robert S Bart, MD
Rela Bartfeld, MD
Sanjit Kumar Basak, MD
Giuseppe Basile, MD
Frank A Bassen, MD
Herbert Bauer, MD
Gilbert Baum, MD
Rosalina L Bautista, MD
Gulam Rasool Bazaz, MD
Harris Robert Bearnot, MD
Joseph Edward Bebry, MD
Mary Alice Becker, MD
E Mark Beckman, MD
Richard Townsend Beebe, MD
Jackson B Beecham, MD
Michael Lee Beehner, MD
Julius Belinkoff, MD
Vincent Sal Beltrani, MD
Marrietta A Benedicto, MD
Thomas Carmine Beneventano, MD
David Monae Benford, MD
Otelia J Bengtsson, MD
Craig Lyon Benjamin, MD
Alexander Berenblit, MD
Herbert V Bergamini, MD
Allan Alexander Berger, MD
Eugene Y Berger, MD
Ruth Pirkle Berkeley, MD
Mordecai A Berkun, MD
Herbert L Berman, MD
Leonard Berman, MD
Harold Bernhard, MD
David Bernstein, MD
James S Bernstein, MD
Richard Mark Bernstein, MD
Edgar Powell Berry, MD
Frederick A Bevelaqua, MD
Purshottam B Bhangdia, MD
Dinesh Bhargava, MD
Anthony Andrew Bianco, MD
Ken Paul Biegeleisen, MD
Roger David Billig, MD
Otto Emil Billo, MD
Ralph Edward Binder, MD
Gerald Alan Black, MD
Jeffrey W Blake, MD
Willard James Blauvelt, MD
Josephus TH Bloem, MD
Robbert H Blom, MD
William H Bloom, MD
Naomi T Bloomfield, MD
Dwight C Blum, MD
Isaac Blum, MD
Albert E Blundell, MD
George Walter Bock, DO
Richard Lewis Bodkin, MD
Virgil Henry F Boeck, MD
Susan L Bogdan, MD
John N Boger, MD
Maria Dziak-Ova Bohm, MD
William James Boland, MD
Angelo Robert Bologna, MD
Kenneth WW Bone, MD
Alfred R Bonsignore, MD
Maccabae Boorstein, MD
A Gale Borden, MD
Seymour Robt Bortner, MD
Reinhard Erich Bothe, MD
Roger Joseph Boulay, MD
Edward Thos Bowe, MD
George Loveridge Bowen, MD
Thomas K Boyce, MD
Arthur Dean Boyd, MD
Roger Boykin, MD
Mary E Bradford, MD
Warren Robert Brady, MD
Bruce Howard Braffman, MD
Daniel Brauman, MD
Arnold Breecker, MD
John Brendan Brennan, MD
Jack Leon Brenner, MD
Schiele Alcuin Brewer, MD
Philip Walter Brickner, MD
Bernard Brill, MD
David N Broadbent, MD
Eric R Brocks, MD
Cecil Balfour Broderick, MD
Adrian Brodey, MD
Samuel Alan Brody, MD
Theodore Tobias Bronk, MD
G Freeman Brooks, MD
Franklin L Brosgol, MD
Charles Bruce Brown, MD
Charles Mc Donnell Brown, MD
Malcolm Mcdougal Brown, MD
Salvatore Joseph Brucato, MD
Stanley Fred Brunn, MD
Morton S Bryer, MD
Richard Joseph Buckley Jr., MD
Robert Leonard Bull Jr., MD
Bruce Lawrence Burgreen, MD
John Heckman Burke, MD
Mark Roger Burns, MD
Miguel Angel Cabral, MD
Alfredo Mendoza Cabusora Jr., MD
John Donald Cahill, MD
Sesinando S Calalang, MD
Angelo Cammarata, MD
Bruce Campbell, MD
Otto E Campos, MD
Elizabeth T Cancroft, MD
Joel Canter, MD
Antonio S Carbonell, MD
Leo Robert Cardillo, MD
Timothy M Cardina, MD
Vincent Olin Carfagna, MD
Aurora P Lao Carino, MD
Joseph Francis Carlucci, MD
Neville Wayne Carmical, MD
Dennis Salvatore Caruana, MD
Robert M Carvalho, MD
Catalino B Casals Jr., MD
Mario Cerabona, MD
Maxwell Mani Chait, MD
John Kenneth Chamberlain, MD
Joseph G Chanatry, MD
Chu Jiang Chang, MD
John Byong-Ho Chang, MD
Patricia Gail L Chapman, MD
Alex Charlton, MD
Patrick N Chauhan, MD
Robert S Chavkin, MD
C James Chen, MD
Robert Chernaik, MD
Herbert Sylvan Cherry, MD
Roland Fan-Hung Cheung, MD
Ruangsak Chewaproug, MD
Jae-Hak Choe, MD
Adel E Chouchani, MD
Gabriel Emile Chouchani, MD
Faizur Rahman Chowdhury, MD
Judith Chown, MD
Foen B Chu, MD
Sterling Chudow, MD
Shockpin C Chungfat, MD
John A Cinelli, MD
Lawrence A Cioffi, MD
William Edward Cioffiro, MD
Howard Citrin, MD
Thomas Allan Clark, MD
William Thomas Clark, MD
Edwin Garfield Claxton, MD
Marshall Clinton, MD
Elbert Lester Close, MD
Roscoe Roy Coats, MD
Barry H Cohen, MD
Harvey Jay Cohen, MD
Richard James Collins, MD
George Philip Congram, MD
C Stephen Connolly, MD
Gino Anthony Consigliere, MD
Barry Constantine, MD
C Perry Cooke III, MD
John Arthur Cooke, MD
Glen Ernest Cooley, MD
Joel Jay Cooper, MD
Kent Pettit Cooper, MD
Robert Alan Cooper, MD
Adrian R Coren, MD
Robert Thomas Corey, MD
Florian M Cortese, MD
Jose A Costo, MD
Russell J Crider, MD
Ruth J Cudmore, MD
Jack Cukierman, MD

Gabriel G Curtis, MD
Rouvoe Cushing, MD
Ignatius Cyriac, MD
Gabriel A P D'Amato, MD
Joseph Francis D'Amore, MD
Joseph D'Aniello, MD
John A D'Anna Jr., MD
Carmelo D'Anna, MD
Armand Joseph D'Errico, MD
Robert F D'Esposito, MD
Boris Dadic, MD
Edwin Thomas Dailey, MD
Rajendrakumar S Dalal, MD
Basil Geo Dalavagas, MD
Robert Ward Daly, MD
Paul Shibli Damus, MD
Sadegh Danesh, MD
Irwin Marvin Dannis, MD
Douglas A Danosos, MD
Fred Danziger, MD
Pranab Kumar Datta, MD
Rajesh J Dave, MD
Robert Carlton Davenport, MD
Gail Davidson, MD
Anthony Jos Davino, MD
Robert Earle Davis, MD
Mary Catherine Daye, MD
Gary De Angelis, MD
Alfredo Pasia De Castro, MD
Jose Hernandez De Castro, MD
Albert M De Fabritus, MD
Charles Thomas De Lise, MD
Ralph De Luca, MD
John James De Marchi, MD
Dwight Carlton De Risi, MD
Vincent J De Risio, MD
Francis Rocco De Rossi, MD
Felice J De Stefano, MD
George Amos Del Monte, MD
Celestino M Del Rosario, MD
Robert W Delorme, MD
Joseph P Demeri, MD
Robert William Dennis, MD
George Rehmi Denton, MD
J Richard H Deschamp, MD
Jane E Devens, MD
Kirit M Dharia, MD
Anthony John Di Fabio, MD
George Di Giacinto, MD
Robert Albert Di Giacinto, MD
Albert B Diamante, MD
James Dick, MD
Augustine Diji, MD
Danilo Aliling Diokno, MD
Arthur Dishman, MD
Ariel Distenfeld, MD
Michael Edmund Dobmeier, MD
Leo Dobrin, MD
Justin E Doheny, MD
Daniel L Dombroski, MD
Augustin Emilio Don, MD
Anthony Donn, MD
Stuart L Dorfman, MD
Gerald Dale Dorman, MD
John M Doyle, MD
Samuel Dominick Drago, MD
Dennis Draizin, MD
Mary Ellen Drislane, MD
Emanuel Dubow, MD
Richard H Dunkelberger, MD
John Knight Dustin, MD
Leo Dvorken, MD
William C Dwyer, MD
Edward Francis Dziob, MD
Nurettin Eber, MD
John A Edwards, MD
George Egri, MD
Henry C Eichelmann Jr., MD
Sidney N Eichenholtz, MD
Jack Eisert, MD
Daniel C S Ellison, MD
Albert Marvin Ellman, MD
Storer William Emmett, MD
Peter Bruce Engber, MD
Gordon L Engler, MD
Benjamin David Erger, MD
Mark Steven Erlebacher, MD
Stanley Leonard Erney, MD
David Ernstoff, MD
Charles Escriva, MD
Manuel R Esguerra, MD
Benjamin Esterman, MD
Napoleon N Estrada, MD
Abdul E Etemadi, MD
Peter D Ewing, MD
Thomas John Fahey, MD
Edward Francis Falsey, MD
Louis A Farchione Jr., MD
Davood Farhangian, MD
William George Farlow, MD
Hal Allan H Farquhar, MD
Walter John Farrell, MD
Peter A Fauci Jr., MD
Raymond D Fear Jr., MD
Harold Federman, MD
Michael S Feinberg, MD
Joseph Feingold, MD
Alfred Feldbau, MD
Herbert Byron Feldman, MD
Ronald Feldman, MD
William C Feldman, MD
Arthur Felix, MD
Irwin Felsen, MD
Armando S Fernandez-Fox, MD
James C Fernbach, MD
Andre D Fethiere, MD
Sidney S Feuerstein, MD
David H Fields, MD
Gencer Filiz, MD
James Joseph Finnerty, MD
Willard George Fischer, MD
Leo Fishel, MD
Benjamin Samuel Fishman, MD
Thomas Michael Fitzpatrick, MD
Fred Norman Flatau, MD
Richard Fleckner, MD
Roberta E Flesh, MD
Robert Anthony Flint, MD
Arthur John Florack, MD
Morris Fogel, MD
William Gilbert Fogland, MD
Morris S Fond, MD
Donald M Ford, MD
Lewis Freeman Foster, MD
Karen S Fountain, MD
Harold Edward Fox, MD
Matthew Freed, MD
Peter Arthur Freedman, MD
Edwin Ned Freeman, MD
Duane T Freier, MD
Eugenie M Fribourg, MD
Emery Imre Fried, MD
Richard Peter Fried, MD
Alvin E Friedman-Kien, MD
Gerald Friedman, MD
Norman Elliot Friedman, MD
Orel Friedman, MD
Robert Charles Friedman, MD
Sanford James Friedman, MD
George E Froehlich, MD
Irving Gustave Frohman, MD
E L Fronczak-Bukowska, MD
Shih-Hao Fu, MD
Joseph J Fusco, MD
Firmin F Gabriels, MD
Jane Olaksen Galasso, MD
Iago Galdston, MD
George Galos, MD
John Jenkins Gamble, MD
Milagros G Gamoso, MD
Segundino Valdez Gamoso, MD
Benjamin M Garfinkle, MD
Mitchel G Garren, MD
Howard S Garson, MD
Kenneth Lee Gayles, MD
Nathan Geffen, MD
Ronald D Gellis, MD
Robert Matthew George, MD
Seymour Gers, MD
Mohsen P Ghavami, MD
Leonard J Giambalvo, MD
Mario Lawrence Giampe, MD
John J Giardino, MD
Daniel S Giddings, MD
Alice L Arnold Gilbert, MD
Bruce S Gingold, MD
Gerald Ginsberg, MD
Stanley Arthur Ginsberg, MD
Martin Alan Ginsburg, MD
Leonard V Gioia, MD
Louis William Giordano, MD
Jose Alberto Giron, MD
Dante Louis Gismondi, MD
Marc C Gittelman, MD
Harry Michael Givelber, MD
William Price Given, MD
Arthur Henry Gladstein, MD
Richard Eugene Glass, MD
Lawrence Eric Glassberg, MD
Thomas B Glendening, MD
Irving V Glick, MD
Bruce Steven Gneshin, MD
Brij Gopal Goel, MD
Veena Goel, MD
Arthur Alexander Gold, MD
Isadore Roy Gold, MD
Scott David Gold, MD
Arthur Frank Goldberg, MD
Gary S Goldberg, MD
Michael Marvin Goldberg, MD
Philip Goldberg, MD
Howard Jay Goldman, MD
Carl Goldmark, MD
Arthur Hugh Goldstein, MD
Michael Edward Goldstein, MD
Moe Goldstein, MD
Rasela Goldstein, MD
Meyer E Golob, MD
Ubaldo Gonzalez, MD
Michael Alan Goodfriend, MD
Louis Goodkin, MD
Samuel Goodloe Jr., MD
Jack Goodman, MD
Mark L Goodman, MD
M S Gopalakrishnan, MD
Rajagopalan S Gopalan, MD
Philip E Gordon, MD
Stanley L Gordon, MD
Burton L Gorson, MD
Ethel Gottfried, MD
Herman Gottlieb, MD
Haider G Goussous, MD
Jacob J Graham, MD
Roberto C Granato, MD
Stephen Poss Grayer, MD
Leo A Green, MD
Ernest Jos Greenberg, MD
Joseph Greenberg, MD
Elliott Charles Greenfield, MD
Saul Philip Greenfield, MD
Charles R Greenhouse, MD
Herbert Greening, MD
Joel Greenspan, MD
Edward S Greenwald, MD
John Roy Gregory, MD
Michael Elliot Gribetz, MD
Howard Eli Grill, MD
Margaret M Grogan, MD
Bernard Francis Groh, MD
Albert W Grokoest, MD
Jay Grossman, MD
C Samuel Grove, DO
Jeanne Eberhardt Grove, DO
Boris Gueft, MD
Lerma Abes Guerrero, MD
Krishan Gopal Gulati, MD
Stephen J Gulotta, MD
Awadhesh Kumar Gupta, MD
Krishna Kumar Gupta, MD
Swarn Kumar Gupta, MD
George Gilbert Gusset, MD
William Herald Gutstein, MD
P C Ward Glenn Gypson Jr., MD
Robert D Haar, MD
Francis B Haber, MD
George A Habib, MD
James Allen Hadley, MD
Byung Du Hahn, MD
Samuel Aaron Hale, MD
Constantine A Haliasos, MD
Warner Cleland Hall, MD
Herbert Louis Halper, MD
Eugene Halpert, MD
Richard H Hamilton, MD
Vincent Grant Hammond, MD
John W Handel, MD
David Gerard Handron, MD
Bernardo Handszer-Guss, MD
Susan Elizabeth Hanson, MD
James William Hanway, MD
A S Mozammel Haque, MD
Ikram Ul Haque, MD
Andrew A Haraway, MD
Alan Marc Harawitz, MD
Hasmukh Harde, MD
Antoine C Harovas, MD
Raymond Harris, MD
Robert J Harris, MD
Stanley Lee Harris, MD
George Gremple Hart, MD
William T Hart, MD
Alfred A Hartmann Sr., MD
Harry Rodney Hartmann, MD
A Daniel Hauser, MD
Louis Arthur Hauser, MD
Patrick J Hayes, MD
Robert Basil Hayes, MD
David Simon Hays, MD
Peter Joseph Heffer, MD
Robert H Heissenbuttel, MD
Eugene Heller, MD
John P Hemmerlein, MD
Jack I Hentel, MD
Hector Raul Herrera, MD
Paul Hershenson, MD
Ralph Earl L H Hertz, MD
Frank Peter Herzberg, MD
Engel Philip Hevenor, MD
A Douglas Heymann, MD
Joseph Thomas Hickey, MD
Matthew Timothy Higgins, MD
Ann Cathleen Hill, MD
Norman A Hill, MD
George L Hines, MD
Ladislav P Hinterbuchner, MD
Irving A Hirsch, MD
Herman Hirschfeld, MD
Stuart E Hisler, MD
John George Fred Hiss, MD
Robert J Hochstim, MD
Frank Beeckman Hoefle, MD
Stanley Hoff, MD
David E Hoffman, MD
Ronald A Hoffman, MD
Sanford R Hoffman, MD
V Michael Hogan, MD
Edward William Hohensee, MD
Leo James Holmsten, MD
B Smith Hopkins, MD
Alfred Emanuel Horowitz, MD
Helen B Horowitz, MD
Irving I Horowitz, MD
Melvin Horwith, MD
Bert S Horwitz, MD
Raymond L Horwitz, MD
Elliott J Howard, MD
Peter J Howard, MD
William James Howard, MD
Suzanne Adele L Howe, MD
Richard Jos Howell, MD
Chen Chou Hu, MD
Chien-Jen Huang, MD
Le Hieng Gay Huang, MD
Robert Harry Huddle, MD
Richard A Hughes Jr., MD
Richard A Hughes, MD
Wilbur Bingham Hurlbut, MD
Marvin Leroy Huyck, MD
Eugene C Hyzy, MD
Claire Marie Iamele, MD
Angelo Illuzzi, DO
Calvin James F Innis, MD
George Ioannou, MD
Christodoulos Iordanou, MD
Erlinda D Israel, MD
Junichi Iwai, MD
Joseph M Jabbour, MD
Lawrence S Jackman, MD
Robert Alan Jacobs, MD
Robert M Jaffee, MD
Sudarshan K Jagga, MD
Alexander Jakubowycz, MD
Richard Ralph Jamison, MD
Ralph H Janicki, MD
James Louis Januzzi, MD
Parviz Javdan, MD
Kurt Jellinek, MD
Raymond James Jensen, DO
Helen Z Jern, MD
Krishna J Jhaveri, MD
Carlos Jorge Jimenez, MD
C Salomon Jollek, MD
Euclid Howard Jones, MD
Ralph Jones, MD

Edward G B Joseph, MD
Jean Delille Joseph, MD
Harry Mills Judge, MD
Robert Seth Jutkowitz, MD
Raj B Kachoria, MD
William Lowell Kahan, MD
Martin Laurence Kahn, MD
Jay Howard Kalinsky, MD
Michael Alan Kalvert, MD
Daniel H Kamen, MD
Chester Edwin Kaminester, MD
Adrian Chas Kanaar, MD
Baik Hyun Kang, MD
Mehmet Hayati Kanmaz, MD
Alfred Kant, MD
Herbert G Kantor, MD
Kanhaiyalal Kantu, MD
Saul Emanuel Kapel, MD
Lawrence I Kaplan, MD
Max Kaplan, MD
Zelek Micah Kaplan, MD
Adib H Karam, MD
Iqbal Karim, MD
Robert David Karlan, MD
Harry E Karounos, MD
Abraham Karver, MD
Aileen S Fennell Kass, MD
Athanassios S Kassapidis, MD
David Katz, MD
Donald I Katz, MD
Jack L Katz, MD
Marcia A Katz, MD
Herbert M Katzin, MD
Edwin H Kaufman, MD
Roger Stuart Kaufman, MD
Herbert Jacob Kaufmann, MD
John Herbert Kavanaugh, MD
Hugo A Keim, MD
Howard Douglas Kelley, MD
Philip J Kelly, MD
William James Kelly, MD
Isadore Maurice Kendal, MD
Reuben K Kenigsberg, MD
John Anthony Kennedy, MD
John D Kennedy, MD
Douglas R Kerr, MD
Kenneth J Kerwin, MD
Necati Keskin, MD
George J Kessler, DO
Nancy Conrad Kester, MD
Akberali G N Khakee, MD
Haider A Khan, MD
Maidul Islam Khan, MD
Mahmood Ahmad Khwaja, MD
Lewis Foster Kibler, MD
Janith S Kice, MD
Kyung In Kim, MD
Oksoon Kim, MD
Taik Hwan Kim, MD
Young Il Kim, MD
Paul A Kirschner, MD
Ira Alexander Kishner, MD
Robert Ward Klein, MD
Suzanne W Klein, MD
Myron I Kleiner, MD
Alexander Kleinman, MD
Lawrence Michael Kleinman, MD
Thomas Klepetar, MD
David Noel Kluge, MD
Stanley Jay Kogan, MD
Peter Louis Kogut Jr., MD
Michael Stephen Kohn, MD
Basil P Koletsis, MD
Edward J Komora, MD
Donald S Koretz, MD
Elise H Korman, MD
Martin William Korn, MD
Abraham Kosseff, MD
John William Kostecki, MD, P.C.
Hadjispyros Kosti, MD
Jehangir Keki Kotwal, MD
Simon Kove, MD
Daniel Charles Kozera, MD, P.C.
Philip J Kozinn, MD
Robert A Krall, MD
Daniel J Krellenstein, MD
Stojanka Kresic, MD
Boris Krikun, MD
Ludmila Krikun, MD
Ronald Martin Krinick, MD
Marilyn M S Kritchman, MD
Paul S Kruger, MD, P.C.
Eugene F Kuchner, MD
Amy Huei-Mei Kuo, MD
Robert J Kurtz, MD
William C Kwock, MD
Charles A La Marca, MD
Nicolo La Rocca, MD
Richard Benjamin Lacki, MD
Nagla M Laf Loofy, MD
Daniel Chi-Tao Lai, MD
Rima Ellen Laibow, MD
Bernard I Laitman, MD
Drake M Lamen, MD
Edward F Lanigan, MD
Louis Sanford Lapid, MD
Arthur J Lapovsky, MD
James Latimer, MD
Anita Navarro Laude, MD
Herbert A Laughlin, MD
Juan K Laurilla, MD
Harold Wyatt Laws, MD
Rumoldo C Layug, MD
Edward B Leahey, MD
Richard J Leberer, MD
Garwood Emerson Leckband, MD
David Sigmond Lederman, MD
Bum Suck Lee, MD
Jae Ho Lee, MD
Keun Yong Lee, MD
Pen-Fa Lee, MD
Henry J Lefkowits, MD
Jacob M Lehman, MD
R Richard Leinhardt, MD
Amol Shashikant Lele, MD
Ann Marie Lenane, MD
Austin R Leve, MD
Louis S Levenstein, MD
Julius W Levi, MD
Warren Mayer Levin, MD
Alan Martin Levine, MD
David Levine, MD
E Mark Levinsohn, MD
Geraldine L Levitt, MD
Marc Steven Levitt, MD
Morton Gerald Levy, MD
Norman Joel Levy, MD
Walter Levy, MD
James E Lewis, MD
Marjorie Greta Lewisohn, MD
Ronald Li, MD
George Liberman, MD
Irwin Eugene Librot, MD
Jacob Lichy, MD
H David Lieberman, MD
Jerrold S Lieberman, MD
Joel Liebermann, MD
I Yang Lin, MD
Pi Tan Lin, MD
Martin Lipkin, MD
William Lipton, MD
Jack Victor Lisman, MD
Joseph Litwins, MD
Mario Benedict Lo Monaco, MD
Robert C Lockwood, MD
Margaret Mary Loder, MD
James G Lodolce, MD
Thomas A Loftus, MD
Joseph Bruce Logue, MD
James T Lohnes, MD
Louis John Lombardi, MD
Marc David London, MD
Norman R Loomis, MD
Gonzalo Lopez, MD
Elger Horst Lorenzsonn, MD
William C Lowery, MD
John Edward Lowry, MD
Irwin I Lubowe, MD
Henry Horst Ludemann, MD
Lee R Lumpkin III, MD
Charles H Luss, MD
Bennett P Lustgarten, MD
Michael John Lynch, MD
James Bruce Mac Whinney, MD
Lilia Remedio Lim Maceda, MD
Renan Macias, MD
Daniel Rob D Mackenzie, MD
Angelo J Madonia, MD
Vincent V Madonia, MD
Lodewijk A Magre, MD
William Charles Maguire, MD
Bertha Spiegel Maier, MD
Vasistha G Majmundar, MD
Iwan Makarewycz, MD
Servando B R Malabanan, MD
Jay V Malde, MD
Milton R Malev, MD
M C Manasan-Greenberg, MD
Daniel Henry Manfredi, MD
Charles C Mangi, MD
Jack Manpel, MD
Anna Z Manska, MD
Arnolfo Sarrosa Marcella, MD
Frank C Marchetta, MD
Peter J Marchisello, MD
Leon Marienberg, MD
Peter John G Maris, MD
Mary R Headlee Markham, MD
Fredric Marks, MD
Ira Marks, MD
David Marshall, MD
Muriel Ruth Marshall, MD
George L Marthy, MD
Herbert John Marx, MD
Dennis Maser, MD
Daniel Jay Mason, MD
Sam Rankin Mason, MD
Joseph Stanley Matala, MD
Timothy D Mattison, MD
Raymond J Maxwell, MD
John Joseph May, MD
Thomas Francis Joseph May, MD
Raymond Joseph Mc Carthy, MD
Joyce M Mc Chesney, MD
Thomas A Mc Cormick, MD
Richard Smith Mc Cray, MD
Daniel J Mc Cue, MD
William F Mc Cully Jr., MD
Joseph V Mc Donald, MD
James H Mc Donough, MD
Annora Harris Mc Garry, MD
Lillian E Mc Gowan, MD
James Mc Groarty, MD
Thomas K Mc Inerny, MD
Apolinario Medalla, MD
Pravin C Mehta, MD
Jerome L Meisel, MD
Daniel J Melia, MD
James Edward Meltzer, MD
Michael J Melzer, MD
Guido John Memoli, MD
Joseph Mendelow, MD
Tullio Raimondo Mereu, MD
Arthur Harold Mernit, MD
A John Merola, MD
William Henry Merrilees, MD
Bertram S Mersereau, MD
John Chase Mesch, MD
Jack John Messina, MD
William Josef Messinger, MD
Seymour William Meyer, MD
Fouad B N Michael, MD
Stanley Robert Michalski, MD
W Jost Michelsen, MD
Robert Richard Michiel, MD
Frank A Migliorelli, MD
Donald E Miller, MD
Howard Moith Miller, MD
Robert Lee Miller, MD
Carl Mills II, MD
Balshik Min, MD
George M Mina, MD
David Theodore Mintz, MD
Michael R Mirwald, MD
Riaz Ahmad Mirza, MD
Anita Gross Moallem, MD
Jeffrey Steven Mogelof, MD
A Hassan Mohaideen, MD
Niaz Ahmed Momin, MD
Liberatore Paul Monaco, MD
Edward Joseph Monteiro, MD
Robert Michael Mooney, MD
Francis X Moore, MD
Marcelle M Morcos, MD
Henry V Morelewicz, MD
Robert F Morello, MD
Joel Jean Morris, MD
Norman Morris, MD
Robert Haskel Morris, MD
William Edward Morse, MD
Campbell Moses, MD
Armand H Moss, MD
Leo Muido, MD
Thomas Owen Muldoon, MD
Philip Leo Mulholland, MD
Edward Eugene Mullen, MD
James Wm Murphy, MD
Tito Musacchio, MD
Frederick Muschenheim, MD
John A Mycek, MD
John P Myers, MD
Uma Vasant Mysorekar, MD
Rebecca Faith Nachamie, MD
Edith Nachmansohn, MD
Alexander H Nacht, MD
Joshua G F Nagin, MD
Vitali Namer, MD
Mary Susan Napoleon, MD
Jeanmarie A Napolitano, MD
Rhoda K Scharf Narins, MD
Richard Jay Nattis, MD
Lawrence Fred Nazarian, MD
Elliot Seth Nelson, MD
William James Nelson, MD
Selig B Neubardt, MD
Richard R Neufeld, MD
Adrian Neumann, MD
Karl Neumann, MD
James C Newton, MD
Jeffrey H Newton, MD
Benny King Chuen Ng, MD
John Michael Nicoletti, MD
Samuel Nisnewitz, MD
Paul Martin Nonkin, MD
Stephen Nordlicht, MD
Harriett E Northrup, MD
Marko Nyitray, MD
John J O'Brien, MD
William O'Dwyer, MD
Colleen E O'Leary, MD
James M O'Malley, MD
Deolindo Ocampos, MD
Hany M Oghia, MD
David D Olds, MD
Augusto D Ongsiako, MD
Juliana Opatich, MD
Morton Orlov, MD
Dean E Orman, MD
Ghassem Z Oskoui, MD
John A Ostuni, MD
Paolo Ottolenghi, MD
Lawrence Jay Pacernick, MD
Jesus B Pacquing, MD
Vitale Henry Paganelli, MD
Philip Pagano, MD
Salvatore Joseph Pagliaro, MD
Sabg Kee Pahk, MD
Murlidhar Pahuja, MD
Neil M Palladino, MD
Donald J Palmadessa, MD
Richard P Palop, MD
Lung Hsiung Pan, MD
David Pankin, MD
Theodore Papademetriou, MD
Jorge M Pardo, MD
Benjamin B Park, MD
Keun Ho Park, MD
Nicholas Parlamis, MD
Steven M Parnes, MD
Gasper Parrino, MD
Thomas Lawrence Parsley, MD
Benu Pascariu, MD
Jonathan Benj Pasternack, MD
Sureshkant P Patel, MD
James Earl Patterson, MD
David Alan Paul, MD
Dusan Pavlica, MD
Christopher Pavlides, MD
David Franklin Pearce, MD
Herbert Pearl, MD
Henry Paul Pechstein, MD
Fremont Carson Peck, MD
John Joseph Pellettieri, MD
Arturo Pena, MD
Kathaleen Claire Perkins, MD
William A Petersen, MD
Jos John Petrone, MD
Eustace G Phillies, MD
Clay Edison Phillips, MD
Gary Anthony Piccione, MD
John Joseph Pileggi, MD
Murray B Pincus, MD
Diana Marie Pintus-Emma, MD
Anna M Piotrowski, MD

Bernard J M Pisani, MD
Richard Walker Pitkin, MD
Gerald Harris Pitman, MD
Marvin J Pleskow, MD
Henry William Pletcher, MD
Joseph Michael Pober, MD
Robert Hilleary Poe, MD
Benjamin Pollet, MD
Paul A Pomerantz, MD
Alcides C Pomina, MD
Arthur R Poole, MD
Stephen R Pope, MD
George Lionel Popkin, MD
Martin Popowitz, MD
Francesco Porcarelli, MD
Robert Truman Porter, MD
Joseph Post, MD
Mohamad Pourfar, MD
Douglass N Powell, MD
G Allen Power Jr., MD
Prayoon Prabharasuth, MD
Suhas Vasant Pradhan, MD
Balasa V Lakshmi Prasad, MD
Baleshwar Prasad, MD
David S Pratt, MD
Theodore C Prentice, MD
Donald W Preston, MD
George John Primavera, MD
Ronald Alan Primis, MD
Gustave G Prinsell, MD
Edner Prophete, MD
Michael Thos Pugliese, MD
James Harry Puleo, MD
James Edgar Purnell, MD
Amarlal Purohit, MD
John Stanley Puzauskas, MD
William Pyka, MD
John Kevin Quinlivan, MD
Coleman Berley Rabin, MD
Franziska W Racker, MD
Milton Joseph Rader, MD
Robert Paul Raggi, MD
Miguel A Rainstein, MD
Frank F Raio, MD
Henry Ramanathan, MD
Patricia Ann Randall, MD
B R Mylar Rao, MD
Daliparthy V Rao, MD
Abraham I Raskob, MD
Rustom A Rastinehad, MD
Calvin Lewis Rasweiler, MD
George Paul Reading, MD
Raphy R Rebanal, MD
Walter Redisch, MD
Lawrence Alexander Reduto, MD
Arlene B Reed-Delaney, MD
George E Reed, MD
Nathan Parker Reed, MD
Thomas Dee Rees, MD
Ronald Craig Reese, MD
Hafiz Ur Rehman, MD
Khalid L Rehman, MD
Cecil A Reid, MD
Robert Siskind Reiffel, MD
Sylvia Jean Reimer, MD
Raphael Reiss, MD
Stephen M Remen, MD
Michael I Resnick, MD
Gregorio V Reyes, MD
Juan D Reyes, MD
Rosalita Geronimo Reyes, MD
Robert Joel Reza, MD
Victor Daniel Ribeiro, MD
Daniel Stephen Rich, MD
Joseph Richard, MD
Charles Hiram Richards, MD
George D Richards, MD
James Oliver Richardson Jr., MD
Earle Griff Ridall, MD
Bela Redey Rieger, MD
Terry P Rifkin, MD
Charles J Riggio, MD
Donald Henry Rizzuto, MD
Judith Pesany Robertson, MD
Philip James Robinson, MD
Philip Ralph Robinson, MD
Stanko Rodic, MD
Charles E Rogers, MD
Orin Louia Rogers, MD
Adelaide Romaine, MD
Paul Peter Romanello, MD
Joseph Mario Romano, MD
Norman Reid Romanoff, MD
Zenaida S Roque, MD
Roger Miles Rose, MD
Carl Sanford Rosen, MD
David Elliot Rosenberg, MD
Richard J Rosenberg, MD
Stuart Alan Rosenberg, MD
Stuart T Rosenberg, MD
Stephen Alan Rosenthal, MD
Henry Rosner, MD
Kalman Donald Rosner, MD
Gilbert Lee Ross, MD
Michael Sander Rost, MD
Desider J Rothe, MD
Albert Rothman, MD
Gary Roxland, MD
Joseph A Rube Jr., MD
Harry Rubenstein, DO
Henry Rubenstein, MD
Ira Saul Rubenstein, MD
Ira Lloyd Rubin, MD
James Milton Rubin, MD
Leonard Morton Rubin, MD
Morris Rubin, MD
Richard Rubin, MD
G Theodore Ruckert, MD
Nathan Rudolph, MD
Paul Ruegsegger, MD
Joseph F Ruh, MD
Joan Marion Rumberg, MD
John Anthony Rurak, MD
Richard A Ruskin, MD
George L Rutherford, MD
Eugene Saberski, MD
David B Sachar, MD
Virginia Sadock, MD
Leroy Scheller Safian, MD
Hidetaka Sagawa, MD
Mohamed Fawsey Saleem, MD
Zarko Antun Salic, MD
Martin Jay Saltzman, MD
Naeem Samad, MD
Oscar Samuely, MD
Myriam Selinger Sanders, MD
Rafael O Santandreu, MD
Mario D Santilli, MD
Edilberto S Santos, MD
Eduardo Saponara-Milligan, MD
Narendra Saraiya, MD
Bijoy Bhushon Sarmaroy, MD
Janina Sass, MD
Abelardo C Saure, MD
William A Sausmer, MD
Suman Kumar Sawhney, MD
Philip N Sawyer, MD
Lewis Schachne, MD
Arthur John Schaefer, MD
Sidney Mansfield Schaer, MD
Edward Schall, MD
Nathan Scharf, MD
Robert V Schatken, MD
David Chas Schechter, MD
Ronald Steven Scheinzeit, MD
Charles R Schen, MD
Trude Joan Schiff, MD
Harvey S Schildkraut, MD
Ralph George Schilling, MD
Antonino Schimmenti, MD
Robert Allen Schissel, DO
Albert Schlisserman, MD
Ralph Edward Schlossman, MD
Joseph Schluger, MD
Howard R Schneider, MD
Philip Schneider, MD
Michael J Schnur, MD
Myron Royal Schoenfeld, MD
Conrad T H Schroeder, MD
Miller Harry Schuck, MD
Joseph Ghelli Schultz, MD
Albert M Schwartz, MD
Jonathan R Schwartz, MD
Morton Schwimmer, MD
Richard Schwimmer, MD
John Richard Scott, DO
Thomas Peter Sculco, MD
Alapat Mathew Sebastian, MD
Jorge Enrique Secchi, MD
Leroy Allen Seftel, MD
Arthur Jay Segal, MD
Albert Segenreich, MD
S P Sehgal, MD
Harvey Seigerman, MD
Surya Sekar, MD
Michael Meir Sela, MD
Balazs Selendy, MD
Charles J. Sellers and Co.
Denzil A L Sequeira, MD
Linda Q Sese, MD
Joseph Sferrazza, MD
Arvind R Shah, MD
Ashok Kumar S Shah, MD
Jitendra Chimanlal Shah, MD
Jyotindra Gopaldas Shah, MD
Niranjan J Shah, MD
Sassan Shahmanesh, MD
Joseph F Shanaphy, MD
Sydney Shapin, MD
Judith R Shapiro, MD
Lee Schulman Shapiro, MD
Paul Shapiro, MD
Stephen Daniel Shappell, MD
Daniel C Shaughnessy, MD
Edward Yeong-Tai Shen, MD
Wallace M Sheridan, MD
Frederic Morris Sherman, MD
Mark F Sherman, MD
Maurice Elish Sherman, MD
Isaac H Shohet, MD
Joel M A Shugar, MD
Thomas Francis Shultz, MD
Charles Leroy Shute Jr., MD
Oscar J Siberio, MD
Edward Siegel, MD
Howard William Siegel, MD
Irving Siegel, MD
Robert John Silich, MD
Murray Y Silver, MD
J William Silverberg, MD
Sidney M Silverstone, MD
Frank Anthony Simone, MD
Domenico Sincero, MD
Irwin Edward Siris, MD
Douglas M Sirkin, MD
Meredith F Sirmans, MD
Jitendra Singh Sisodia, MD
Louis Rodolph Slattery, MD
James A Slavin, MD
Simon Smelensky, MD
Dennis George Smetana, MD
Joseph A Smith, MD
William Henry Smith, MD
Jeffrey S Smoller, MD
Juanito T So, MD
Samuel Hillel Sobel, MD
Sidney Harry Sobel, MD
Robert Lawrence Soley, MD
Renee Solow, MD
Susan K Soni, MD
Lawrence Sidney Sonkin, MD
Stanley Soren, MD
Wheelock A Southgate, MD
Martin Spatz, MD
Ruth G Spear-Levenbook, MD
Will Nelson Spear, MD
Frank Cole Spencer, MD
James Alexander E Spencer, MD
Paul Robt Spierling, MD
John M Spring, MD
Morris Springer, MD
Efthimios C Spyropoulos, MD
Salvatore P Squitieri, MD
Alice Slater Stahl, MD
Albert William Stahman, MD
John Jeffery Stangel, MD
Eugene R Stanton, MD
Milton Honore Stapen, MD
Thomas David Stapleton, MD
Jesse Donald Stark, MD
Orkan G Stasior, MD
Harry Statman, MD
Stanley A Steckler, MD
Constantine Stefanides, MD
Bennett Mueller Stein, MD
David Isaac Stein, MD
Ephraim Stein, MD
Kenneth M Steinglass, MD
James A Steinman, MD
Fred Bernard Stern, MD
Kaethe D Stern, MD
Richard Stern, MD
Roland E Stevens, MD
William A Stevens, MD
Robert Alan Stookey, MD
Fred Charles Storm, MD
William H Strutton, MD
Lincoln Stulik, MD
Richard P Sullivan, MD
Robert Lyons Sullivan, MD
Abraham Sunshine, MD
Edmond Alfred Suss, MD
Albert Philip Sutton, MD
Reginald Mortimer Sutton, MD
David Sohmer Svahn, MD
Philip Arthur Swiantek, MD
Leo Jerome Swirsky, MD
Emery Szanto, MD
Carl Richard Szot, MD
Pinkus H Szuchmacher, MD
Carl Harris Tafeen, MD
Mahmood N Tafreshi, MD
Merle Nito Tandoc, MD
Sheila S Tanenbaum, MD
Robert Tankel, MD
Ronald J Tatelbaum, MD
Sheldon Bailey Taubman, MD
Gary Bruce Tebor, MD
Frederick H Theodore, MD
Francis Thomas Thomas, MD
Lloyd Allen Thomas, MD
Edith Millina Thompson, MD
Mary Ti, MD
Robert Thomas Todaro, MD
Carl Arthur Todoro, MD
Tamer Toklucu, MD
Matthew Armin Tolchin, MD
Thomas A Toosie, MD
Charles M Totero Jr., MD
Sylvia Gould Traube, MD
Ernest Arnold Triback, MD
Frank John Tricarico, MD
A Albert Tripodi, MD
Kenneth W Trout, MD
Mary Yuen-Yin Tsai, MD
Pavritta Man Tuladhar, MD
Martin Abraham Tuller, MD
Thomas Virgil Tupper, MD
Rudolph Turnbull, MD
James William Turner, MD
Leona A Turner, MD
Peter A Tutschka, MD
Lloyd D Tuttle, MD
Dundar Salih Tuzun, MD
Howard Benson Tykot, MD
Kadri Osman Ulman, MD
Suresh Vrajlal Undavia, MD
Robert Morrow Ungerer, MD
Hernan A Urbano, MD
Ronald V Uva, MD
Daniel N Uwah, MD
Krishna Kumar Vadlamudi, MD
Fredrick A Valauri, MD
Courtland Van Deusen III, MD
John J Vasile, MD
Ralph James Veenema, MD
Raphael M Verdile Jr., MD
Norma Perez Veridiano, MD
John J Vetter, MD
Job R Vibal, MD
Carmine T Vicale, MD
Anita Vigorito, MD
Myrtle M Wilcox Vincent, MD
Timothy Joseph Vinciguerra, MD
Palanisamy Viswakumar, MD
George Vogel, MD
Henry E Vogel, MD
Howard H Volan, MD
Jacobus P Vrolijk, MD
Harry Leopold Wachen, MD
Jay E Wagner, MD
Robert Bruce Wallace, MD
Riley Walter Waller, MD
Stanley H Warmund, MD
Donald J Warner, MD
Willard B Warring, MD
Ira A Wasserberg, MD
Lloyd E Watts, MD
Stuart Harold Wax, MD
George T C Way, MD
Gary Alvin Weaver, MD
Cecile R Wechsler, MD
Harold R Weichert, MD
Samuel Weinberg, MD

Edwin C Weinraub, MD
Gerald Weintraub, MD
David Ira Weiss, MD
Jules Weiss, MD
Leonard S Weiss, MD
Richard L Weiss, MD
Robert Rafael Weiss, MD
Ralph N Weller, MD
Aaron O Wells, MD
Reinhardt Wm Wende, MD
Arthur Robert Wertheim, MD
Martin S Weseley, MD
Robert Elmer Westlake Jr., MD
Aaron Saml Weston, MD
M Jay Wexler, DO
John S Whelan, MD
E Douglas Whitehead, MD
John R Whittier, MD
Wanda S Wieckowska, MD
Gwendolyn Williams, MD
Selden T Williams, MD
Wanda Willig, MD
C Philip Wilson, MD
Donald Merritt Wilson, MD
Marvin Irwin Winston, MD
Harry Wogalter, MD
Robert Edmund Wolf, MD
Ezra A Wolff, MD
David G Wolinsky, MD
Fred Theodore Wolkoff, MD
Stuart Barry Wollman, MD
Martin E Wolpin, MD
Lionel B Wong, MD
Harold Everett Wood Jr., MD
Berwyn Howard Woodman, MD
Jack H Woodrow, MD
Hugh Woodworth, MD
William R Woolner, MD
Carl Stape Worboys, MD
Duncan L Wormer, MD
Bernard Wu, MD
Howard David Wulfson, MD
Changi Yang, MD
George Demetrios Yatrakis, MD
Robert Lee Yeager, MD
Chin Bor Yeoh, MD
E Howard M Young, MD
Michael C Young, MD
Shug-Hong Young, MD
Sol Harold Youngwirth, MD
Horace I-Sen Yu, MD
Stuart Leslie Yunis, MD
Stephen Fred Zaffuto, MD
Rodolfo Serraon Zamudio, MD
Daniel M Zeichner, MD
Bertram E Zeitel, MD
Susan Zilkha, MD
Walter T Zimdahl, MD
Alan J Zimmerman, MD
Erwin H Zimmerman, MD
Sidney Paul Zimmerman, MD
Joseph George Zimring, MD
Leon Norman Zoghlin, MD

BAND-AIDS WILL NOT CURE THE MEDICAL LIABILITY CRISIS.

Make no mistake about it – the medical liability crisis is far from over. Some of the laws passed recently to ease the crisis should be viewed as strictly stop gap as they fail to get at the root of the crisis: a tort liability system that does not work.

The governor acknowledges the shortcomings of the recent legislation, having pledged to undertake further reform this fall. We applaud his willingness to include the input of physicians and hospitals.

Total and comprehensive reform of the tort liability system is the only cure for this crisis that threatens to undermine the health-care system in New York.

The recent legislation falls way short. For instance, physicians had asked for a cap on liability in line with the present limit of $1 million of insurance. The legislature responded to this problem by making an additional $1 million available *at the expense of hospitals and taxpayers.* At a cost to the state of $60 million a year, lawyers can now draw from a much bigger pot and physicians are still personally liable for any awards over $2 million. We do not consider this effective reform.

Another gaping hole in the legislation concerns the obscene insurance premiums physicians are forced to pay. The recent legislation did little more than slightly reduce an already whopping increase and delay to the fall what in all likelihood will be another big rate hike.

The medical liability problem in this state is both serious and complex – there is no easy solution. We trust that our governor and legislators will find the solutions so that we can continue to guarantee to all New Yorkers the high quality medical care you've come to expect.

Malpractice... Nobody wins... Everybody pays

Medical Society of the County of Erie

THANK YOU!

The physicians in Western New York extend a big thank you to our fellow citizens for your support, understanding and cooperation throughout the medical liability crisis.

THANKS to all of the people who wrote letters, signed petitions and called their state legislators to urge reform of the inequitable medical liability system.

THANKS to all the patients who supported their physicians during the crisis, and graciously accepted the inconveniences caused by it.

THANKS to all of the hospital and health-care workers who were forced to take unscheduled vacations, were temporarily furloughed or juggled schedules.

THANKS to the staff at the Erie County Medical Center which did yeoman's work in coping with the increased demand for services.

THANKS to the Medical Emergency Radio System (MERS) and all others involved with emergency medical services for filling the gap created by the crisis.

THANKS to the media organizations whose journalists covered the crisis fairly and objectively.

THANKS to Assemblymen John Sheffer and L. William Paxon, and Senators Walter Floss, Dale Volker and John Daly who exhibited the foresight and conviction to support meaningful reform of the system when others would not.

We have achieved an important first step toward total reform of the medical liability system. Unfortunately, this is only one small step. Much more needs to be accomplished in order to ensure the continued delivery of quality medical care for all New York citizens. It's nice to know we can count on your support.

MEDICAL SOCIETY OF THE COUNTY OF ERIE

CONVERSATIONS IN MEDICINE. THE STORY OF TWENTIETH-CENTURY AMERICAN MEDICINE IN THE WORDS OF THOSE WHO CREATED IT.

By Allen B. Weisse, MD. 443 pp. New York, New York University Press, 1984. $25.00 (softcover)

Medical history buffs will enjoy reading this book. Using a question and answer session preceded by pertinent background material on 16 outstanding physicians, the author succeeds in portraying some of the remarkable medical discoveries made in the last half century. The select group includes three Nobel laureates, two black physicians, and two women physicians.

The first chapter, which sets the tone for the rest of the book, deals with William Dock. Dock is an impressive, articulate, and provocative investigator as well as a superb clinician and teacher. The author aptly recounts Dock's theories on heart sounds, the use of inexpensive ballistocardiography, and the apical localization of pulmonary tuberculosis. Omitted are his thoughts on cholesterol, wine, smoking, and notching of the ribs in patients with coarctation of the aorta.

The remaining chapters provide profiles on the following physicians: Bailey, one of the first to split a stenotic mitral valve; Murphy, who helped resolve the mystery of pernicious anemia; Weintrobe, a renowned hematologist; Cournand, who had the courage to pass a catheter into his own heart; Weinstein, who helped conquer scarlet fever; Pickering, a proponent of Britain's National Health Insurance Program, whose words raised as many blood pressures as his treatment lowered; Moore, a renowned surgeon who helped smooth postoperative metabolic turbulence and made significant contributions in gastric surgery, burns, and health manpower; Wagensteen, whose tube overshadowed his surgical and teaching prowess; Johnson, the black physiologist who overcame many obstacles on his way to the deanship of Howard University Medical College; Goodman, a pharmacologist; Ropes, one of the early white female practitioners whose interest in arthritis was ignited at Johns Hopkins Hospital and Massachusetts General Hospital; and Lena Edwards, a black woman obstetrician and gynecologist who was a devout Catholic, mother of six, a social activist, and a fine practitioner, educator, and philanthropist. There are also accounts of Good, whose superb work in immunology led to life-saving bone marrow transplants, and Kolff, whose pioneering work with the artificial kidney and heart saved thousands of lives.

Finally, the author discusses Kornberg, who was born in Brooklyn and who graduated from City College. He received his medical degree from the University of Rochester, pursued a career in biochemical research, and was awarded a Nobel prize for his work with DNA.

This book is pleasant and easy to read. However, it may disappoint those who have read Arturo Castiglioni's classic or the more recent book, *Two Centuries of American Medicine,* written by Bordley and Harvey.

JULIUS E. STOLFI, MD
SUNY—Downstate Medical Center
New York City 11203

CANADIAN MEDICINE: A STUDY IN RESTRICTED ENTRY

By Ronald Hamowy. 417 pp. Vancouver, British Columbia, The Fraser Institute, 1984. $14.95 (softcover)

This book is a publication of a conservative think-tank, the Fraser Institute, located in Vancouver, British Columbia. It is a supply side-book, a companion to an earlier demand side-volume, *The Health Care Business*, from the same Institute, that attempted to show that the present Canadian national-provincial system of publicly financed health insurance would be more efficient if there were greater reliance on market forces. The current volume is by a history professor from the University of Alberta, trained in history and economics, a prolific writer on social and legal theory, and a graduate of the more recent University of Chicago schools of thought. The book focuses on why medical licensing was adopted in Canada and the economic effects of the licensing of physicians.

Mr Hamowy draws on a wide variety of historical sources to present evidence of the effects of an increasingly powerful Canadian medical establishment. They have succeeded in banning price and other advertising by physicians and have set minimum price schedules. They have acted to prevent an oversupply of physicians by encouraging and having adopted, in various parts of Canada, through time, irrelevant criteria for licensing (examples include a knowledge of grammar, mathematics, Latin, philosophy, language requirements, etc.). They have acted to prevent the study of medicine by persons not controlled by the medical profession. They have prevented foreign doctors from practicing in Canada, where they could compete with domestic Canadian doctors.

The medical establishment has imposed entry examinations, but not examinations to re-certify doctors in practice on an ongoing basis over the years, that would be the case if the profession was truly interested in quality of care. It fought against prepaid contract practice, against doctors testifying for plaintiffs in malpractice cases, and has discouraged charity work because such work undermines minimum fee schedules and professional prestige. It has raised the cost of medical education to increase the cost of entry into the medical profession. It has succeeded in raising physicians' income levels beyond that of other equally skilled professions in Canada.

This book reviewer, a Canadian of left of center persuasion, agrees with this litany of evidence. But is the fact that Canadian medical school tuition is one sixth or one tenth that of the United States good or bad? We really don't know where the author himself stands on all of the issues that he raises. We really don't even learn from him the implications of the evidence he had presented. Instead, the implications are drawn before the book starts, by the Fraser Institute director of the Centre for the Study of Economics and Religion, Mr Walter Block. Why in the world this is a preface rather than a postscript is a mystery to me. Block draws implications from the work of Hamowy.

The present monopoly system of state and provincial licensing of physicians would be replaced by many "certifiers of medical practitioners," each competing with the other for prestige, status, and, of course, money. Competition would be unleashed. Competitive certification would take over, with perhaps a decade of co-existance with the present system of licensing.

On the one hand, supplier-induced demand would increase medical billings. On the other hand, price elasticity with many more providers would lead to lower medical costs. In order to make the new system work properly, the Canadian system of national health insurance would have to be dismantled to remove the public sector middleman, so that market forces could go to work in the newly unleashed system.

To this reviewer, reading the so-called preface before I read the book reduced its appeal immeasurably. If the author drew the implications that Block etched out in the preface, why didn't he say so?

I concluded that the book was a theoretical effort by a committed health care supply sider, who drew on relevant and interesting historical models along the way.

SAMUEL WOLFE, MD
Columbia School of Public Health
New York City 10032

PRACTICAL OPHTHALMIC PROBLEMS

By Wayne F. March, MD. 91 pp, illustrated. St. Louis, Warren H. Green, Inc, 1984. $12.50 (softcover)

The author states in the introduction to this book that its purpose is to provide the reader with a general knowledge of the eye so that important ocular diseases can be diagnosed and understood. A list of common abbreviations ("Short forms in clinical use") follows the introduction.

The body of the monograph is composed of nine chapters that address topics such as background, eye injuries, glaucoma, disorders of the retina, visual fields, cataracts and intraocular lenses, visual handicaps, glasses and contact lenses, and pediatric ophthalmology. The chapter on basic background briefly reviews the anatomy and physiology of the eye, without significant detail. In addition, the various components of an eye examination are described including picutres of a Snellen eye chart, tonometers, biomicroscope (slit-lamp) and direct ophthalmoscope. Chapter 2, on eye injuries, contains useful information regarding the recording of visual acuity as the first procedure in any eye examination. The diagnosis and management of corneal abrasions, ultraviolet burns, acid and alkali burns, corneal and eyelid lacerations, foreign bodies of the conjunctiva and cornea, and hyphema are presented in outline form with corresponding photographs. The ophthalmoscopic findings in trauma to the retina and choroid are recorded as are the stigmata of "blow-out" fractures.

In Chapter 3, the author discusses both open angle and acute closed angle glaucoma, as well as secondary glaucoma. Pilocarpine is termed "a muserinic agent"; this should read muscarinic. In the chapter on disorders of the retina, arterial and venous occlusions, both central and branch, are discussed, with pictures of their characteristic ophthalmoscopic appearances. The author uses the term "amaurosis fugax" to describe the "sudden loss of vision" in one eye in a patient with a central retinal artery occlusion. Amaurosis fugax means "fleeting blindness" and should not be used to describe the "sudden loss of vision" in one eye in a patient with a central retinal artery occlusion. Patients with amaurosis fugax regain vision which has become temporarily impaired. The remainder of this chapter is comprised of short discussions of macular degeneration, ocular histoplasmosis, and diabetic retinopathy.

Several techniques for obtaining visual fields are illustrated in Chapter 5, and cataracts and intra-ocular lenses are discussed in a very short chapter. The chapter on visual handicaps provides examples of low vision aids. In the chapter on glasses and contact lenses, the author states that "a patient who has not needed glasses until age 42 can simply purchase a pair of glasses used only for reading at the local dime store." An ophthalmologic examination should be performed before obtaining glasses at any age. The eye is a mirror of systemic and neurologic dysfunction, and a complete ocular examination is indicated for any visual or ocular motor complaints. The final chapter on pediatric ophthalmology mentions amblyopia and its management, recording of vision in childhood, defects in muscle balance, retinoblastoma, and gonorrhea.

This book presents a bird's-eye view of some common ocular entities. It is not designed for the reader familiar with ophthalmology, and it cannot be recommended for an in-depth discussion of these problems. One practical problem which has not been included is diplopia. In sum, more complete, though not encyclopedic, books are available.

ARTHUR H. WOLINTZ, MD
SUNY-Downstate Medical Center
New York City 11203

COLOR ATLAS OF THE NEWBORN

By R.D.G. Milner, MD and S.M. Herber, MB. 136 pp, illustrated. Oradell, NJ, Medical Economics Co, 1984. $44.95 (hardcover)

The authors of this atlas set out to compile a series of illustrations of both common and unusual clinical manifestations of neonatal disorders, and acknowledge utilizing material from slide collections of 26 of their colleagues. The result is, as they had hoped, a "relatively inexpensive atlas" and a very "useful learning aid." The senior author is well known for his contributions to the neonatal literature. He and his collaborator have succeeded in selecting and organizing the 369 illustrations in the atlas in a sensible manner, grouping major and minor variations from the norm according to the predominant anatomic site that is involved. Several additional chapters review genetic conditions, problems acquired during fetal development, traumatic sequelae of the birth process, and conditions occurring after delivery.

The illustrations are in color, and the overall quality of the photography is excellent. Among the few exceptions are the photographs of craniofacial dysostosis and of congenitally acquired herpes, which should be replaced by clearer examples.

The legends to the figures are written in simple, concise language, though one would have liked somewhat more extensive descriptions of some of the conditions. Among the minor errors or debatable statements in the book is the assertion that hemolytic disease due to Rh incompatibility is the most common cause of hydrops fetalis and of neonatal ascites. This is no longer so in those countries that have been using Rho immunoglobulin (Rhogam) routinely in women who are Rh-negative. The statement that damage to the brachial plexus in Erb's palsy involves particularly spinal nerve roots C3, C4, and C5, and the argument against the routine use of silver nitrate for prophylaxis of gonococcal ophthalmia are also debatable. Furthermore, most pediatric surgeons would regard gastroschisis as a separate entity, rather than the result of a ruptured omphalocele.

Although many neonatologists have extensive slide collections of their own, this atlas should provide a convenient and simple mechanism for presenting illustrative examples of clinical material during small group discussions. It will be a distinct asset to all those involved in neonatal care and in teaching students, nurses, and housestaff. Considering the modest price, it deserves to be part of the resource material available on all neonatal services.

NATHAN RUDOLPH, MD
Department of Pediatrics
SUNY-Downstate Medical Center
New York City 11203

THE DIABETIC'S BRAND-NAME FOOD EXCHANGE HANDBOOK. FOOD EXCHANGES FOR OVER 3,000 SUPERMARKET, GROCERY STORE, AND FAST-FOOD PRODUCTS.

By Andrea Barrett. 171 pp. Philadelphia, Running Press, 1984. $14.95 (hardcover)

Closer attention to dieting is required in order to translate the marked advances made by biochemical research into improved clinical results for patients with diabetes mellitus. It is now reasonable to achieve euglycemia in patients with insulin-dependent diabetes as well as weight reduction in patients with type II diabetes.

Two factors must be considered in all dietary treatment. First, compliance on the part of the patient is necessary. Recently expanded measures in health education have helped in achieving better results. Second is the goal of maintaining a near normal life style in the presence of the many inhibitions demanded by a treatment regimen for diabetes. This book can assist in attaining this objective. It can also lead to better compliance on the part of many patients.

Fast food and frozen dinners have become a prominent part of the American life style. The author of this book has collected and listed various brand name fast foods available in the marketplace. She presents an easy method for calculating suggested portions that facilitates substitutions into diets provided by the American Diabetes Association. No changes in prescribed diets are necessary, and no calculations of carbohydrate, protein, and fat content of the diet is required. For instance, I learned that one of my favorite foods in the frozen food counter, namely, the Celeste brand Sicilian Style Pizza (26-oz package) can be included in a regulated diet; one quarter of the pizza can be substituted for 2½ slices of bread, 2 oz of lean meat, and 2 allowed portions of fat. Also, Stouffer's short ribs of beef with vegetable gravy can be exchanged for 4 oz of lean meat, one-half portion of a vegetable, and 2½ pats of butter.

The book contains a vast amount of such information about commonly used fast foods. The products of Arby's, Burger King, McDonald's, Roy Rogers' and other eating establishments are well covered. The book contains a valuable section on available combination main dishes, frozen dinners, fish, shellfish, meats, and even vegetarian meat substitutes. Alcoholic beverages are also discussed. There is a helpful chapter dealing with various baked foods including pancakes and waffles, as well as pasta with sauces.

For nutritionists, the encyclopedic information in this book is a valuable asset. It is also a handy reference book for physicians who must answer questions about dieting from patients. The educated patient can easily use the information and can enjoy the freedom of eating foods that were previously forbidden and introducing more variability into his or her diet.

MILTON B. HANDELSMAN, MD
Long Island College Hospital
New York City 11201

SCHIZOPHRENIA: STRAIGHT TALK FOR FAMILIES AND FRIENDS

By Maryellen Walsh. 264 pp. New York, William Morrow & Co, 1985. $15.95 (clothcover)

This book is an informational guide and self-help publication for the families of the two million persons in this country suffering from schizophrenia. Written by a professional writer and researcher who is also a single mother of a schizophrenic son, it is clearly a labor of love, written, as the author candidly informs the reader, with the help of periodic escapes to the bathtub for long soaks with big packs of bubble gum and the latest issue of *Cosmopolitan*.

The book is neatly organized into traditional chapters covering what is known and not known about the disease. It discusses what life is like for the families of persons with schizophrenia and tells how to cope and how to organize for change. The author provides a liberal bibliography, as well as other helpful appendices, including the names and addresses of self-help groups and organizations for the mentally ill throughout the country, and a listing of involuntary commitment laws of all states. An index and note source chapter is also included.

Some of the author's material comes from questionnaires sent to families affiliated with the National Alliance for Mental Illness, most of whom seem as disaffected as she. The book reflects the hours of research put into the subject by a lay writer motivated by the most passionate of desires—to understand the catastrophe that happened to her and those she loves, and to do what she can to remedy the situation. By and large, it reflects with accuracy the state of knowledge today, although inevitably some errors and non-sequitors creep in. Her professional style—crisp, hard-hitting, short of sentence, hard of impact, with theories and syntax pared to the brevity and simplicity required for a sixth-grade level audience—enables the reader to sail through without impediment.

The book attempts to mobilize the eight million Americans (of the 160 million worldwide) who, as families of persons with schizophrenia, live in "the largest closet in the world." Mercilessly hammering at society for hiding its psychotic citizens, refusing to recognize them, deal with them, help them, adequately research their illness, she is full of anger, outrage, and rebellion with which she struggles, in vain, to deal with fairly. Perhaps it is too much to ask of a woman who embarked on her task from the most subjective of motives, and with so much to be mad about. Nevertheless, her contempt for the psychiatrists who have engineered what she believes to be so much needless misery is a bit much.

The author exhibits scorn for the psychoanalytic movement, which she perceives as blaming families for the illness of their children without adequate evidence, for flawed research, and for Freudian theory that exacerbates the patient's condition. She fails to consider, however, that few reputable analysts *do* consider analysis the treatment of choice for schizophrenia, and few would undertake anything but the most supportive therapy without concomitant neuroleptic medication. Her characterization of families by psychiatrists as "psychovermin" who are humiliated, punished, and cut off from their kids when they are most needed is intemperate. It reflects, however, the exquisite sensitivity of today's families to being blamed, and how important it is for the psychiatrist to avoid pouring salt on wounds.

Certainly the author raises issues and levels charges with much historic validity. However, sifting the truths from the half-truths, near-truths, and sometime truths is a major task in this disturbing book. Families who read it are not going to like psychiatrists very much. (But, says the author, they don't like them now.)

Her most helpful chapters concern coping. Her most interesting thoughts deal with suggestions for research, such as organizing conferences of multidisciplinary forces rather than the usual discussions limited to know-little psychiatrists, and suggestions for information-sharing through computerization of local support services. Her most important message is that no one knows a schizophrenic individual like the person who has lived with him 168 hours a week, and that a new psychiatric broom should not be allowed to sweep clean.

NAOMI BLUESTONE, MD
Great Neck, NY 11021

AN ATLAS OF CRITICAL CARE CHEST ROENTGENOGRAPHY

Edited by Lyle D. Victor, MD. 284 pp, illustrated. Rockville, MD, Aspen Systems Corp., 1985. $45.00 (hardcover)

The two stated purposes of this book are to summarize the authors' experience in the radiologic evaluation of critically ill patients, and as a reference for radiograph pattern recognition. The authors believe the book may be used as a resource for visual comparison when unfamiliar radiograph patterns are encountered on the chest film. Many authors have contributed to the text. Unfortunately, only the editors have identified their areas of expertise. It is not possible to judge the qualifications of the other authors.

The book is divided into ten chapters. Each chapter consists of a short text followed by multiple radiographs indexed to the text. In general, the radiographs are of high quality and are well labeled and captioned. The first two chapters are devoted to a brief review of pulmonary anatomy and radiograph fundamentals.

There is a section on abnormal roentgenographic patterns of localized and diffuse lung disease. The information presented here is sketchy and is easily found in standard chest radiology textbooks. Several chapters are devoted to commonly encountered disease entities and their iatrogenic complications in critically ill patients. These chapters serve as a basic guide for medical students and interns working in an intensive care unit.

The chapters on catheter and tube placement and complications of intubation and tracheostomy are useful for intensive care unit personnel as well as the beginning radiology resident. The last two chapters on abnormal roentgenography and neonatal chest roentgenology seem out of place and are confusing.

A problem with the book is its inadequate bibliography. Most chapters have only one or two references. This makes it difficult to obtain source information, especially on topics that are only briefly mentioned.

In general, the authors have succeeded in producing a book useful to medical students and interns working with critically ill patients. However, as a reference source for radiograph pattern recognition many other books are available that are more useful.

PAUL OSTROVSKY, MD
The Long Island College Hospital
New York City 11201

A CALCULUS OF SUFFERING. PAIN, PROFESSIONALISM, AND ANESTHESIA IN NINETEENTH-CENTURY AMERICA

By Martin S. Pernick. 421 pp, illustrated. New York, Columbia University Press, 1985. $35.00 (hardcover)

The safe and effective management of pain is the focus of modern anesthetic practice. Both physicians and patients expect medical and surgical procedures to be as free of pain as possible. Ideally, patients are expected to die with a minimum of pain. Yet in the nineteenth century, the advent of general anesthetic agents was seen as potentially inimical to the practice of good medicine. This excellent book carefully examines the conflicts created by the discovery of general anesthetic agents in the mid-19th century, a time when many medical practitioners thought that pain was both therapeutic and necessary for the patient's survival. General anesthesia, with its power to induce loss of consciousness and abolish pain, was seen as both a powerful impetus for change, and as a source of potential new dangers and abuses.

This work is carefully organized into three parts. Part one, entitled "Anesthesia and Medicine in Nineteeth-Century America," is an overview of 19th-century American medical thought. The moral and physical consequences of pain relief as seen by various schools of thought serve as the focus of discussion. Part two, "Why Not Everyone? The Meaning of Selective Anesthetization," examines how the various factions reconciled the discovery of general anesthesia and how the discovery helped shape medical practice and ideology as we know it today. Part three, "Who Received Anesthetics: Theory and Practice," addresses the problem of delineating actual 19th-century anesthetic practice.

The book is comprehensive, well organized, and the author's style is engaging. There are portions that could be more concise but, given the complexity of the topic, the book is generally easy reading. The author has clearly made exhaustive use of both resource persons and materials. This is apparent in part three where painstaking review of both hospital care records and the medical literature of the period yields a balanced and insightful view of realities of 19th-century anesthetic practice.

Those looking for a comprehensive history of anesthesia will be disappointed with this volume and would best direct their attention elsewhere. However, the book is "must" reading for those who desire insight into 19th-century American medical practice and social thought. Those interested in anesthesia will find that the book traces the genesis of some of the misconceptions, expectations, and fears about anesthesia that exist today.

It was the author's stated intention to demonstrate that a combination of social, ideologic, and technologic innovations in the mid-19th century brought about dramatic changes in medical ideology and practice. The book is successful in documenting the then growing concern about the alleviation of pain and suffering that was reflective of and affected by those innovations.

JAMES A. DINARDO, MD
Beth Israel Hospital
Boston, MA 02215

OBITUARIES

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Ruth M. Bakwin, MD, New York. Died July 31, 1985; age 87. She was a 1923 graduate of Cornell University Medical College. Dr Bakwin was a Diplomate of the American Board of Pediatrics and a member of the Academy of Medicine, the American Academy of Pediatrics, the American Medical Women's Association, the New York County Medical Society and the Medical Society of the State of New York.

Jacques Jacob Bardavid, MD, New York. Died July 29, 1985; age 74. He was a 1935 graduate of the Universite de Paris Faculte de Medecine. Dr Bardavid was a member of the American Academy of Family Practice, the New York County Medical Society and the Medical Society of the State of New York.

F. Albert Baumann, MD, Binghamton. Died August 6, 1985; age 75. He was a 1937 graduate of Cornell University Medical College. Dr Baumann was a Fellow of the American College of Surgeons and a member of the Academy of Medicine, the Broome County Medical Society and the Medical Society of the State of New York.

Daniel Noyes Brown, MD, Bedford. Died August 10, 1985; age 79. Dr Brown was a 1932 graduate of Columbia University College of Physicians and Surgeons. He was a Fellow of the American College of Physicians, a Diplomate of the American Board of Internal Medicine and a member of the Academy of Medicine, the Westchester County Medical Society and the Medical Society of the State of New York.

Harold Cohn, MD, Brooklyn. Died August 25, 1985; age 70. He was a 1940 graduate of the State University of New York Downstate Medical Center. Dr Cohn was a Fellow of the American College of Physicians, and a Diplomate of the American Board of Internal Medicine. His memberships included the New York Cardiological Society, the Clinical Society of the New York Diabetes Association, the American Thoracic Society, the Kings County Medical Society and the Medical Society of the State of New York.

John D'Arecca, MD, Binghamton. Died June 21, 1985; age 89. Dr D'Arecca was a 1924 graduate of the State University of New York Downstate Medical Center. He was a member of the Broome County Medical Society and the Medical Society of the State of New York.

Louis James DeBellis, MD, Bronx. Died July 18, 1985; age 34. Dr DeBellis was a 1977 graduate of Facultad de Medicina de la Universidad Autonoma de Guadalajara, Guadalajara Jalisco. He was a member of the Westchester County Medical Society and the Medical Society of the State of New York.

Patrick Henry DeCanio, MD, Kerhonkson. Died July 30, 1985; age 79. Dr De Canio was a 1932 graduate of the State University of New York Downstate Medical Center. He was a member of the Queens County Medical Society and the Medical Society of the State of New York.

Edward Leonard Fink, MD, Buffalo. Died June 17, 1985; age 73. He was a 1939 graduate of the State University of New York Downstate Medical Center. Dr Fink was a Fellow of the American College of Surgeons, and a Diplomate of the American Board of Urology. He was a member of the Erie County Medical Society and the Medical Society of the State of New York.

Samuel Barnett Frischberg, MD, Brooklyn. Died June 23, 1985; age 89. He was a 1925 graduate of the State University of New York Downstate Medical Center. Dr Frischberg was a Diplomate of the American Board of Dermatology, Inc., and a member of the American Academy of Dermatology, the Kings County Medical Society and the Medican Society of the State of New York.

John Frederick Gipner, MD, Rochester. Died July 29, 1985; age 89. He was a 1921 graduate of the University of Michigan Medical School, Ann Arbor. Dr Gipner was a Diplomate of the American Board of Ophthalmology and a member of the Academy of Medicine, the New York Ophthalmology Society, the American Academy of Ophthalmology and Otolaryngology, the American Ophthalmological Society, the Monroe County Medical Society and the Medical Society of the State of New York.

Iser Abraham Halpern, MD, Miami Beach, Florida. Died May 17, 1985; age 96. Dr Halpern was a 1917 graduate of First Moscow Medical Institute, Moscow. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Jeno Hartmann, MD, Kew Gardens. Died July 19, 1985; age 85. Dr Hartmann was a 1925 graduate of Schlesische-Friedrich-Wilhelms Universitat Medizinische Fakultat, Breslau. He was a member of the Queens County Medical Society and the Medical Society of the State of New York.

Stanley Cheney Hall, MD, Columbus, Michigan. Died August 6, 1985; age 86. He was a 1926 graduate of New York University College of Medicine. Dr

Hall was a Fellow of the American College of Surgeons and the American College of Obstetricians and Gynecologists, and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the New York Obstetrical Society, the American Fertility Society, the Kings County Medical Society and the Medical Society of the State of New York.

Benjamin Louis Feuerstein, MD, Brightwaters. Died July 30, 1985; age 79. He was a 1929 graduate of the New York University College of Medicine. Dr Feuerstein was a Fellow of the American College of Radiology, and a Diplomate of the American Board of Radiology. His memberships included the Academy of Medicine, the Radiological Society of North America, Inc., the American Radium Society, the Suffolk County Medical Society and the Medical Society of the State of New York.

George Klein, MD, Kew Gardens. Died July 8, 1985; age 90. He was a 1920 graduate of Johann-Wolfgang-Goethe-Universität Medizinische Fakultät, Frankfurt-Am-Main. Dr Klein was a Diplomate of the American Board of Dermatology, Inc., and a member of the Academy of Medicine, New York State Dermatological Society, Society for Investigative Dermatology, the New York County Medical Society and the Medical Society of the State of New York.

Harry J. Lesnick, MD, New Rochelle. Died August 14, 1985; age 86. Dr Lesnick was a 1923 graduate of the State University of New York Downstate Medical Center. He was a member of the Bronx Medical Society and the Medical Society of the State of New York.

Russell George Lindauer, MD, Verona Beach. Died July 1, 1985; age 63. He was a 1947 graduate of Temple University School of Medicine. Dr Lindauer was a Fellow of the American College of Pathologists, a Diplomate of the American Board of Pathology and held memberships in the New York Pathological Society, the American Society of Clinical Pathologists, the Madison County Medical Society and the Medical Society of the State of New York.

Charles Mandel, MD, Bronx. Died August 11, 1985; age 92. Dr Mandel was a 1926 graduate of the University of Nebraska College of Medicine, Omaha. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Albert Anthony Mazzeo, MD, Newburgh. Died July 19, 1985; age 69. Dr Mazzeo was a 1945 graduate of Jefferson Medical College of Philadelphia. He was a Fellow of the American Academy of Family Practice and a member of the Orange County Medical Society and the Medical Society of the State of New York.

Pasquale S. Milazzo, MD, Brooklyn. Died July 25, 1985; age 69. He was a 1940 graduate of the State University of New York Downstate Medical Center. Dr Milazzo was a member of the Kings County Medical Society and the Medical Society of the State of New York.

Wallace Bortle Nixdorf, MD, Oneida. Died August 12, 1985; age 69. Dr Nixdorf was a 1940 graduate of the State University of New York Upstate Medical Center. He was a member of the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc., the Madison County Medical Society and the Medical Society of the State of New York.

Sigismund Peller, MD, Walnut Creek, California. Died July 12, 1985; age 95. Dr Peller was a 1914 graduate of the Medizinische Fakultät Universität Wien. He was a member of the Academy of Medicine, the New York County Medical Society and the Medical Society of the State of New York.

Robert S. Priest, MD, Bronx. Died July 9, 1985; age 37. He was a 1975 graduate of New York Medical College, Flower and Fifth Avenue Hospitals. Dr Priest was a Diplomate of the American Board of Ophthalmology and his memberships included the New York Society for Clinical Ophthalmology the Academy of Medicine, the American Academy of Ophthalmology and Otolaryngology, the Bronx Medical Society and the Medical Society of the State of New York.

John J. Ranallo, Jr., MD, Yonkers. Died June 20, 1985; age 72. Dr Ranallo was a 1942 graduate of Hahnemann Medical College and Hospital of Philadelphia. He was a member of the Westchester County Medical Society and the Medical Society of the State of New York.

Peter Michael Saitta, MD, New York. Died July 18, 1985; age 79. Dr Saitta was a 1931 graduate of Georgetown University School of Medicine. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Albert Christy Santy, MD, New York. Died August 12, 1985; age 81. Dr Santy was a 1931 graduate of Cornell University Medical College. He was a Fellow of the American College of Physicians, a Diplomate of the American Board of Internal Medicine, and a member of the Academy of Medicine, the New York County Medical Society and the Medical Society of the State of New York.

Sydney Paul Schiff, MD, Liberty. Died July 10, 1985; age 67. He was a 1945 graduate of Queens University Faculty of Medicine, Kingston. Dr Schiff was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Surgery and a member of the Sullivan County Medical Society and the Medical Society of the State of New York.

Morris B. Schwartzfarb, MD, Lake Worth, Florida. Died July 3, 1985; age 86. He was a 1924 graduate of the New York University College of Medicine. Dr Schwartzfarb was a member of the Pan American Medical Association, the Bronx County Medical Society and the Medical Society of the State of New York.

Charles Shornstein, MD, Tuckahoe. Died July 21, 1985; age 79. He was a 1933 graduate of the University of St. Andrews School of Medicine, Dundee. Dr Shornstein was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Isidor Silbermann, MD, New York. Died August 15, 1985; age 86. He was a 1924 graduate of the Medizinische Fakultat der Universitat, Wien. Dr Silbermann was a Fellow of the American College of Physicians and a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the New York Neurological Society, the American Psychoanalytic Association, the American Psychiatric Association, the New York County Medical Society and the Medical Society of the State of New York.

Aaron Silver, MD, New York. Died July 11, 1985; age 75. Dr Silver was a 1935 graduate of the Boston University School of Medicine. He was a Fellow of the American College of Physicians and a Diplomate of the American Board of Internal Medicine. His memberships included the Academy of Medicine, the New York County Medical Society and the Medical Society of the State of New York.

Malcolm Goff Smilay, MD, Great Neck. Died August 15, 1985; age 58. He was a 1958 graduate of Medizinische Fakultat der Ludwig-Maximilians Universitat, München. Dr Smilay was a member of the New York State Society of Internal Medicine, the American Society of Internal Medicine, the Nassau County Medical Society and the Medical Society of the State of New York.

Sidney Solomon Steckel, MD, Yonkers. Died June 23, 1985; age 78. Dr Steckel was a 1933 Licentiate of the Royal College of Physicians of Edinburgh. He was a Fellow of the American College of Obstetricians and Gynecologists, a Diplomate of the American Board of Obstetrics and Gynecology, and a member of the Academy of Medicine, the Bronx County Medical Society and the Medical Society of the State of New York.

Emile G. Stoloff, MD, New York. Died August 19, 1985; age 87. He was a 1922 graduate of the New York University College of Medicine. Dr Stoloff was a Fellow of the American College of Physicians and a member of the Academy of Medicine, the American Psychoanalytic Association, the American Psychiatric Association, the New York County Medical Society and the Medical Society of the State of New York.

Lee Rowland Stoner, MD, Syracuse. Died July 1, 1985, age 68. Dr Stoner was a 1943 graduate of the State University of New York Upstate Medical Center, Syracuse. He was a Diplomate of the American Board of Otolaryngology, and a member of the American Academy of Otolaryngology, the American Laryngological, Rhinological and Otological Society, the Onondaga Medical Society and the Medical Society of the State of New York.

Lotte Strauss, MD, New York. Died July 4, 1985; age 72. She was a 1938 graduate of Facoltà di Medicina e Chirurgia Università di Siena. Dr Strauss was a Fellow of the American College of Pathologists and a Diplomate of the American Board of Pathology. Her memberships included the New York Pediatric Society, the New York Pathological Society, the American Association of Pathologists and Bacteriologists, the International Academy of Pathology, the New York County Medical Society and the Medical Society of the State of New York.

George Morris Stroll, MD, Shelter Island Heights. Died August 24, 1985; age 86. Dr Stroll was a 1928 graduate of the University of Virginia School of Medicine, Charlottesville. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Thomas F. Sullivan, MD, Suffern. Died July 10, 1985; age 60. Dr Sullivan was a 1950 graduate of Marquette University School of Medicine. He was a Fellow of the American College of Obstetricians and Gynecologists and a Fellow of the American College of Surgeons, a Diplomate of the American Board of Obstetrics and Gynecology, and a member of the Rockland Medical Society, and the Medical Society of the State of New York.

Moses Swick, MD, New York. Died August 8, 1985; age 85. He was a 1924 graduate of Columbia University College of Physicians and Surgeons. Dr Swick was a Fellow of the American College of Surgeons a Diplomate of the American Board of Urology, and a member of the Academy of Medicine, the American Urological Association, the New York County Medical Society and the Medical Society of the State of New York.

Abraham Joseph Tatelbaum, MD, Somers, Connecticut. Died August 3, 1985; age 78. Dr Tatelbaum was a 1932 graduate of the University of Rochester School of Medicine and Dentistry. He was a member of the Monroe County Medical Society and the Medical Society of the State of New York.

Howard Thomas Thompson, MD, Rochester. Died June 12, 1985; age 69. He was a 1942 graduate of the University of Rochester School of Medicine and Dentistry. Dr Thompson was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Urology, and a member of the Academy of Medicine, the American Urological Association, the Monroe County Medical Society and the Medical Society of the State of New York.

Bernard Bruce Wetchler, MD, New York. Died August 8, 1985; age 60. He was a 1947 graduate of New York Medical College, Flower and Fifth Avenue Hospital. Dr Wetchler was a Fellow of the American College of Surgeons and the American College of Gastroenterology, and a Diplomate of the American Board of Surgery. His memberships included the Academy of Medicine, the Pan American Medical Association, the New York Medical Society and the Medical Society of the State of New York.

Louis A. Wolfe, MD, New York. Died July 22, 1985; age 87. Dr Wolfe was a 1921 graduate of New York University College of Medicine. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Tamarath Knigin Yolles, MD, Stony Brook. Died July 29, 1985; age 66. Dr Yolles was a 1951 graduate of the New York University College of Medicine. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Israel Zeifman, MD, New York. Died August 17, 1985; age 59. He was a 1957 graduate of the Universite de Paris Faculte de Medecine. Dr Zeifman was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the American Psychoanalytic Association, the American Academy of Child Psychiatry, the New York County Medical Society and the Medical Society of the State of New York.

Attention MSSNY Delegates:

The 180th Annual Meeting of the Medical Society
of the State of New York will be held at the New York
Hilton Hotel, New York City, on March 6–9, 1986

ORGANIZED 1807

Nominating Committee, 1986

(The committee nominates Officers, Councilors, and Trustees of MSSNY, and Delegates to the American Medical Association.)

Member-at-Large—Allison B. Landolt, M.D., Westchester, *Chairman*
First District—David M. Benford, M.D., New York
Second District—Stanley A. Steckler, M.D., Suffolk
Third District—Thomas D. Pemrick, M.D., Rensselaer
Fourth District—Franklin A. De Peters, M.D., Warren
Fifth District—Robert M. George, M.D., Oneida
Sixth District—Irwin J. Rosenberg, M.D., Broome
Seventh District—James M. Flanagan, M.D., Wayne
Eighth District—Bruce E. Baker, M.D., Genesee
Ninth District—Seymour R. Stall, M.D., Dutchess
Member-at-Large—Victor J. Tofany, M.D., Monroe

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1983; 83:1089–1094). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), an abbreviated title of no greater than 40 spaces for use as a running foot, and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear on the text. When there are six or fewer authors, all should be listed; where there are seven or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

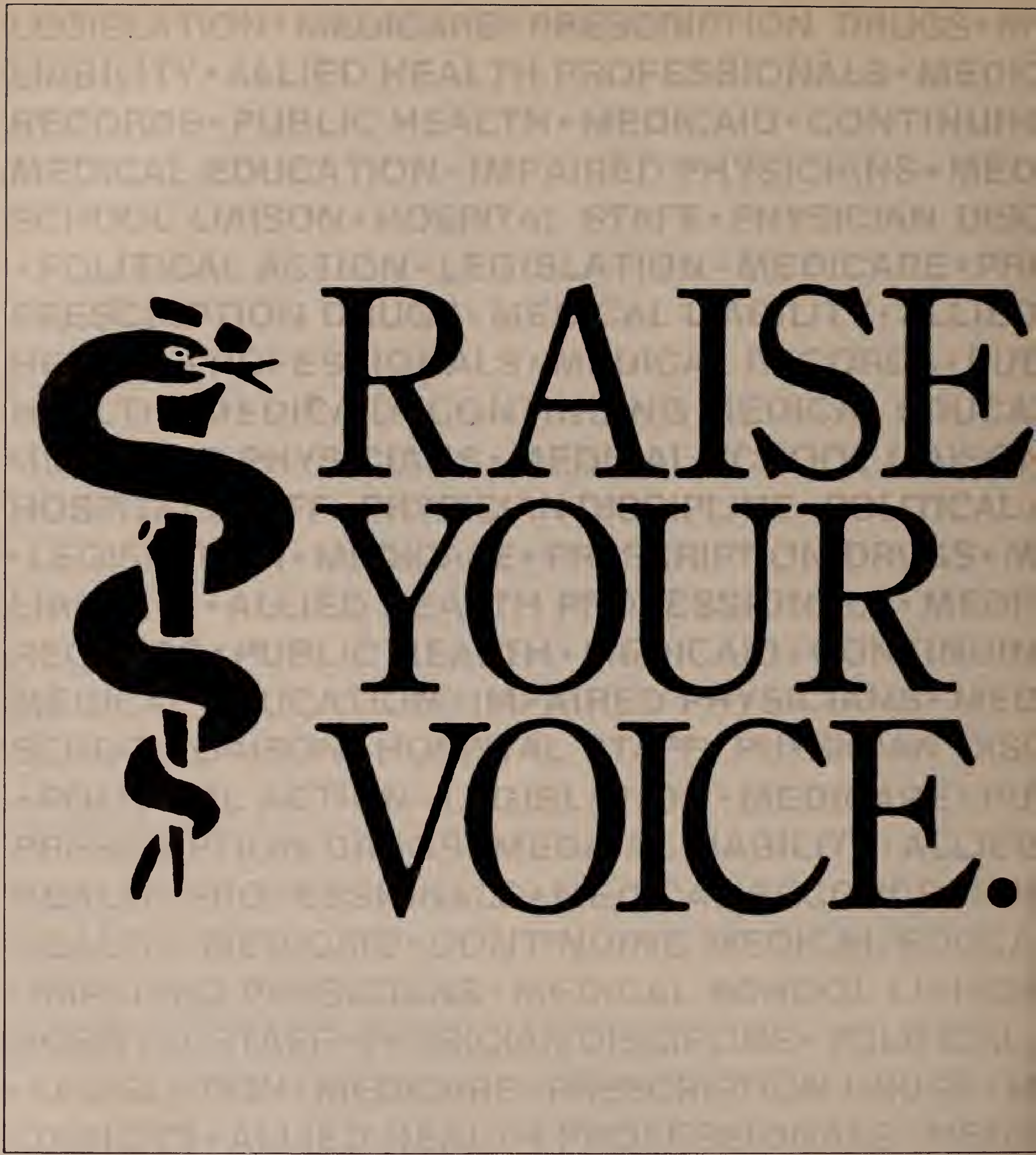
1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. McGuigan JE. Peptic ulcer. In: Petersdorf RG, Adams RD, Braunwald E, Isselbacher KJ, Martin JB, Wilson JD, eds. *Harrison's Principles of Internal Medicine.* New York, McGraw-Hill, 1983, pp 1697–1712.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Alan Blum, MD, Editor, *New York State Journal of Medicine*, P.O. Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

N Y M P A C



New York Medical Political Action Committee
An Arm of the Medical Society of the State of New York
420 Lakeville Road, Lake Success, NY 11042 • 516-488-6100

Political Action for Physician

Essential reading for every physician

NEW YORK STATE JOURNAL OF MEDICINE

JULY 1985 • Volume 85, Number 7

Table of Contents, page 279

THE WORLD CIGARETTE PANDEMIC

Part II

PRIMUM NON NOCERE?

The July issue of the **New York State Journal of Medicine** is the most comprehensive and disturbing ever published by a medical journal on the subject of smoking.

SEND A COPY TO COLLEAGUES AND MEDICAL STUDENTS in the US and abroad who might not normally receive the Journal. Or order an extra copy for your patients or yourself.

NOTE: the extra press run of The World Cigarette Pandemic Part 1 (December 1983 issue) sold out quickly. The issue has been updated and republished as *The Cigarette Underworld* ($9.95 plus $1.50 postage). Copies may be ordered from Lyle Stuart, Inc., 120 Enterprise Avenue, Secaucus, NJ 07094.

An extra press run has enabled the NEW YORK STATE JOURNAL OF MEDICINE to offer single or bulk copies of its recent issue on the world cigarette pandemic. As the official publication of the Medical Society of the State of New York since 1900, the *Journal* is one of the foremost independent peer-reviewed general medical journals in the country. Providing timely and stimulating commentary, challenging research papers, analysis of clinical trends, debate on topical issues, and in-depth coverage of medical news, the *Journal* makes important reading—and a welcome gift—to physicians and students in the United States or abroad. A free copy of the July issue will be included with each subscription.

Quantity	**Per Copy***
1	$7.50
2-9	$7.00
10-24	$6.50
25-49	$6.00
50-99	$5.50
100-149	$5.00
150-500	$4.00

ORDER FORM

Name____________________

Address____________________

City/State/Zip____________________

Please send________copies of July 1985 issue of the Journal.

Enter my subscription to the Journal. ☐ $25.00 U.S. & Canada; ☐ $30.00 Foreign

Total amount enclosed $__________ ***Note:** Domestic postage included. Please add New York state & local sales tax where applicable, or sales tax exemption certificate. Foreign postage additional.

Mail to: Circulation Department, **New York State Journal of Medicine**
420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

Classified Advertising

New York State Journal of Medicine

GENERAL INFORMATION

All classified advertisements are payable in advance, except for government agencies and advertising agencies; a 15% commission to recognized advertising agencies.

Frequency: Monthly
Mailing date: 15th of the month
Closing date: 30 days before mailing date

Circulation: 27,000 (mainly physicians & medical libraries)

DISPLAY ADVERTISING

	1 time	*6 time*	*12 time*	*Width*		*Depth*
1/4-page	$540	$480	$420	3-1/2″	×	4-7/8″
1/6-page	252	228	204	2-1/8″	×	4-7/8″
1/8-page	228	204	180	3-1/2″	×	2-3/8″
1/16-page	114	102	90	3-1/2″	×	1-1/8″
1/18-page	84	72	60	2-1/8″	×	1-1/2″

PARAGRAPH ADVERTISING

The first several words are set in bold face caps.

Rates: $35.00 per insertion for 50 words or less; additional words are 40 cents each. On request, Dept. numbers will be assigned by the Journal for replies to be forwarded to the advertiser. Dept. numbers are 50 cents per insertion. There is no frequency discount for paragraph ads.

Counting Words: Two initials, each abbreviation, figures consisting of 5 numerals or less, are counted as separate words. For replies, your name and address, or telephone number should appear at the end of the ad and be counted in the total number of words.

For further information, please call the Advertising Department at 516-488-6100.

CLASSIFIED ADVERTISING ORDER FORM

New York State Journal of Medicine
Attn: Classified Advertising
420 Lakeville Rd., P.O. Box 5404
Lake Success, N.Y. 11042

Date__________________

Name____________________________________

Address__________________________________

City/State/Zip_____________________________

Payment enclosed: $____________

Number of Insertions: __________

Assign Dept. Number: __________

Please make check payable to New York State Journal of Medicine.

FOREST HILLS **Woodhaven Blvd.**

NEW MEDICAL ARTS BLDG.
NEAR ST. JOHNS HOSPITAL

Suites available 550–4,000 sq. ft. Will alter to suit.

HERB JACOBS **Exclusive Broker**

(718) 454-4321

INTERNIST

Internist with W.C. Board S.J. rating needed full time for my clinics in Buffalo and Rochester to perform evaluations and examinations.

Kick the hassle of practice. Enjoy banker's hours and high income. Reply with C.V. to: H. Lewis, M.D., 3641 Quail Hollow, Bloomfield Hills, Michigan, 48013.

CRAIG PERSONNEL AGENCY, INC.

Medical Placement Specialists
Full Time, Part Time, Temporary
Medical Office Managers/Secretaries/
Assistants/RN's/Technicians/
Receptionists

Affiliated With Health Care Since 1964

40-18 Bell Blvd. (718) 229-3541
Bayside, N.Y. 11361
Manhattan: (212) 809-4141

Oceanside Living At Its Finest

Seabrook Island is resort living at its best on South Carolina's coastline.

Located off the coast of historic Charleston, this private residential resort features 2 championship golf courses, Har-Tru tennis, miles of beach, excellent stables and sports programs.

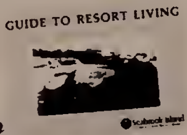
You'll discover homes and homesites situated on the fairways and tennis courts, the sea marsh and ocean, all in a splendidly private natural setting.

So come for a week and stay for a lifetime. **For information or reservations, call toll free 800-845-2475.**

Seabrook Island
Ocean Club

Box 32099, Charleston, South Carolina 29417
800-845-2475

Send for our free Guide to Resort Living



☐ Send your free Guide to Resort Living.
☐ Send vacation information

Name ____________________
Address ____________________
Phone ____________________

NYJM 1185

FOR PHYSICIANS AND SURGEONS WITH A PART-TIME EXPOSURE ONLY

Now offering Medical Malpractice Insurance on an ***occurrence basis*** at drastically reduced rates thru Frontier Insurance Co., Monticello, NY.

With the tail on claims made policies, you might lose your shirt.

Call or write for further information and application.

MEDICAL MQ QUADRANGLE

Medical Quadrangle, Inc.
P. O. Box 456
318 Bedford Road
Bedford Hills, New York 10507
914 666-0170
212 829-3654
"Specializing in Medical Liability Insurance"

Need A Temporary Physician?
During: vacations, CME's, illness, recruiting.
Want Free Time While You Practice Medicine?
Join CompHealth's Locum Tenens Physician Group.
For further information, call 412-741-3310.

CompHealth A Physician Group
WILSON ROSS, Regional Administrator
114 Centennial Ave., Sewickley, PA 15143

NATIONAL EMERGENCY SERVICES, INC. (N.E.S.)

Directorships, full- and part-time emergency department opportunities are available throughout New York State and the country for primary care physicians with ER experience. Enjoy a variety of practice settings, competitive remuneration, paid malpractice insurance, and flexible scheduling. For information and application materials contact the recruitment department at (516) 365-4010, (Outside N.Y. State 1-800-645-4848) or send C.V. to N.E.S., One Hollow Lane, Suite 304, Lake Success, N.Y. 11042.

Index to Advertisers

Ayerst Laboratories 2A,3A,4A,26A,27A,28A
Classified Advertising 5A,17A,18A,20A,24A
Craig Personnel 17A
Eastern Educational Programs, Inc. 5A
HealthWays 6A
Eli Lilly and Company .... 2nd Cover
Marion Laboratories 9A,10A
Medical Quadrangle 18A
A. H. Robins Company 7A,23A
Roche Laboratories .... 3rd & 4th Cover
Sandoz Company 19A,20A
Seabrook Island 17A
Stuart Pharmaceuticals 21A,22A
Upjohn Company 25A

ALZHEIMER'S DEMENTIA

Cure of the disease is still out of reach. In as devastating a condition as this, even the most modest relief of symptoms—or for that matter keeping them from getting worse or merely slowing their intensification—is a great contribution to patient and family.

HYDERGINE® LC (ergoloid mesylates) is indicated for patients over age sixty who manifest signs and symptoms of idiopathic mental decline. It appears that individuals who respond to HYDERGINE LC therapy are those who would be considered to suffer from some ill-defined process related to aging or to suffer from some underlying condition such as Alzheimer's dementia.

Before prescribing HYDERGINE therapy, the possibility that the patient's signs and symptoms arise from a potentially reversible and treatable condition should be excluded. In addition, because the presenting clinical picture may evolve to suggest an alternative treatment, the decision to use HYDERGINE therapy should be continually reviewed.

HYDERGINE® LC
(ergoloid mesylates)
liquid capsules, 1 mg

THE ONLY PRODUCT INDICATED FOR ALZHEIMER'S DEMENTIA.

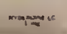
© 1985 Sandoz, Inc.

HYD-1085-13

For Brief Summary, please see following page.

HYDERGINE LC

(ergoloid mesylates) liquid capsules 1 mg



Indications: Symptomatic relief of signs and symptoms of idiopathic decline in mental capacity (i.e., cognitive and interpersonal skills, mood, self-care, apparent motivation) in patients over sixty. It appears that individuals who respond to HYDERGINE therapy are those who would be considered clinically to suffer from some ill-defined process related to aging or to have some underlying dementing condition, such as primary progressive dementia, Alzheimer's dementia, senile onset, or multi-infarct dementia. Before prescribing HYDERGINE® (ergoloid mesylates), the physician should exclude the possibility that signs and symptoms arise from a potentially reversible and treatable condition, particularly delirium and dementiform illness secondary to systemic disease, primary neurological disease, or primary disturbance of mood. Not indicated for acute or chronic psychosis regardless of etiology (see Contraindications).

Use of HYDERGINE therapy should be continually reviewed, since presenting clinical picture may evolve to allow specific diagnosis and specific alternative treatment, and to determine whether any initial benefit persists. Modest but statistically significant changes observed at the end of twelve weeks of therapy include: mental alertness, confusion, recent memory, orientation, emotional lability, self-care, depression, anxiety/fears, cooperation, sociability, appetite, dizziness, fatigue, bothersome(ness), and overall impression of clinical status.

Contraindications: Hypersensitivity to the drug; psychosis, acute or chronic, regardless of etiology.

Precautions: *Because the target symptoms are of unknown etiology, careful diagnosis should be attempted before prescribing HYDERGINE (ergoloid mesylates) preparations.*

Adverse Reactions: Serious side effects have not been found. Some transient nausea and gastric disturbances have been reported, and sublingual irritation with the sublingual tablets.

Dosage and Administration: 1 mg three times daily. Alleviation of symptoms is usually gradual and results may not be observed for 3-4 weeks.

How Supplied: HYDERGINE LC (liquid capsules); 1 mg, oblong, off-white, branded "HYDERGINE LC 1 mg" on one side, "△" other side. Packages of 100 and 500. (Encapsulated by R. P. Scherer, N.A., Clearwater, Florida 33518).

HYDERGINE (ergoloid mesylates) tablets (for oral use); 1 mg, round, white, embossed "HYDERGINE 1" on one side, "△" other side. Packages of 100 and 500.

Each liquid capsule or tablet contains ergoloid mesylates USP as follows: dihydroergocornine mesylate 0.333 mg, dihydroergocristine mesylate 0.333 mg, and dihydroergocryptine (dihydro-alpha-ergocryptine and dihydro-beta-ergocryptine in the proportion of 2:1) mesylate 0.333 mg, representing a total of 1 mg.

Also available: HYDERGINE sublingual tablets; 1 mg, oval, white, embossed "HYDERGINE" on one side, "78-77" other side. Packages of 100 and 1000. 0.5 mg, round, white, embossed "HYDERGINE 0.5" on one side, "△" other side. Packages of 100 and 1000.

HYDERGINE liquid; 1 mg/ml. Bottles of 100 mg with an accompanying dropper graduated to deliver 1 mg. [HYD—ZZ24—6 15 84]

Before prescribing, see package circular for full product information. HYD-1085-13

DORSEY PHARMACEUTICALS
Division of Sandoz, Inc • East Hanover, NJ 07936
A SANDOZ COMPANY

POSITIONS WANTED

PULMONARY INTERNIST, 30, ABIM certified, completing fellowship June 1986. Fully trained in all aspects of pulmonary medicine. Seeks hospital-based or subspecialty group practice affiliated with a teaching institution. Reply Harlan Weinberg, M.D., 337 N. Palm Drive, Beverly Hills, CA 90210, Apt. 8

GASTROENTEROLOGIST-ABIM, University-trained in all endoscopic procedures, ERCP, PEG, peritoneoscopy and consultative gastroenterology/hepatology. Seeking private practice associateship leading to partnership in the NYC/LI area. Available after completion of fellowship in July 1986. Reply Dept. 399 c/o NYSJM.

REAL ESTATE FOR SALE OR RENT

CENTRAL PARK WEST MEDICAL at 86th St. & CPW. PHYSICIANS—Time-sharing (staff, equipment & services provided) or your own exclusive office built to suit. PSYCHIATRIST—Beautiful ground floor soundproof office—exclusive or shared. N.Y.'s most elegant rental, beautiful lobby, security. Prime location on busy block near all transportation. (718) 229-8440.

BAY SHORE, Leasing new luxurious medical building. Located near Southside Hospital. Minimum suite 740 sq. ft. Large parking area, handicapped designed. Individual gas heat, a/c-electric. (516) 665-4064, or 665-4482.

WANTED: MEDICAL OFFICE BUILDINGS. As principals we are seeking "existing buildings"—fully tennanted by physicians, dentists and health care services. Contact: Paul Gellert, Gelco Associates, 155 West 68th St., N.Y., N.Y. 10023; (212) 724-7900.

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

MEDICAL BUILDING—Excellent N.Y.C. location. Free-standing 3200 sq. ft., ground floor. Adjacent to expanding hospital in Queens. Near all transportation, heavy traffic. Ideal for Medical Clinic or Group Practice. Excellent opportunity, Sale or Lease. Call (718) 526-3368, owner.

BROOKLYN, NY MIDWOOD SECTION—DOCTOR'S ROW. 2-family residence. Professional apt. with high income and fully renovated 7 room apt. for owner. Finished rentable basement and playroom for owner. Must see—one-of-a-kind. Asking $449,000. Kings Highway Realty. (718) 376-9300. An equal opportunity broker.

RICHMOND HILL, General Surgeon, Psychiatrist or other suitable specialist to share office with established Pediatrician and Internist. Additional office space also available for future expansion. Reasonable terms. Office located close to transportation and Medical Centers. Call (718) 849-3554.

WEEKLY SEMINARS

Most major ski areas, Club Med, Disney World, Cruising aboard Sailboats in the Virgin Islands or a Mississippi Paddlewheeler.

Topic: Medical/Legal Issues
Accredited ***Fee $175***

Current Concept Seminars, Inc. ***(since 1980)***
3301 Johnson St., Hollywood, FL 33021
(800) 428-6069

MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

1986 CME CRUISE/CONFERENCES on selected Medical Topics—Caribbean, Mexican, Hawaiian, Alaskan, Mediterranean. 7-12 days year-round. Approved for 20-24 CME Cat. 1 credits (AMA/PRA) & AAFP prescribed credit. Distinguished professors. Fly Roundtrip Free on Caribbean, Mexican, & Alaskan Cruises. Excellent group fares on finest ships. Registration limited. Pre-scheduled in compliance with IRS requirements. Information: Intenational Conferences, 189 Lodge Ave., Huntington Station, NY 11746. (516) 549-0869.

DOCTOR, YOU CAN'T BEAT the quality or the price! Holter Monitor Scanning Service. Physician owned, trained and supervised. No contracts to sign. We can arrange for lease/purchase of Holter equipment. New dual channel recorders with test cables $1,550. Now using UP Service for faster turn around time. DCG Interpretation (313) 879-8860.

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuarial certification and employee statements of participation—partnership agreements and professional incorporation—No insurance required—references upon request—Jack Wachstock, PC, 3000 Marcus Ave., Lake Success, N.Y. 11530 (516) 437-4848.

DOCTORS! State-of-the-Art Dual Channel Holter Recorders for only $995.00! Will arrange lease or purchase. Excellent scanning Service available. Only $50.00 for a scanned report. Fast, courteous and professional service. Call today (516) 938-6400, ask for Meryl or Diane.

ARE YOU UNDER INVESTIGATION BY THE STATE LICENSING AUTHORITIES? For consultation call Susan Kaplan, Attorney-at-Law, (212) 877-5998, concentrating in the vital legal problems affecting your professional license with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County.

Angina comes in many forms...

So does SORBITRATE®

(ISOSORBIDE DINITRATE)

Unsurpassed flexibility in nitrate therapy.

2.5 mg 5 mg 10 mg
Sublingual Tablets

5 mg 10 mg
Chewable Tablets

5 mg 10 mg 20 mg 30 mg 40 mg
Oral "Swallow" Tablets

40 mg
Sustained Action "Swallow" Tablets

© 1985 ICI AMERICAS INC.

See following page for brief summary of prescribing information.

SORBITRATE®
(ISOSORBIDE DINITRATE)

Please consult full prescribing information before use. A summary follows:

INDICATIONS AND USAGE: SORBITRATE (isosorbide dinitrate) is indicated for the treatment and prevention of angina pectoris. All dosage forms of isosorbide dinitrate may be used prophylactically to decrease frequency and severity of anginal attacks and can be expected to decrease the need for sublingual nitroglycerin.

The sublingual and chewable forms of the drug are indicated for acute prophylaxis of angina pectoris when taken a few minutes before situations likely to provoke anginal attacks Because of a slower onset of effect, the oral forms of isosorbide dinitrate are not indicated for acute prophylaxis

CONTRAINDICATIONS: SORBITRATE is contraindicated in patients who have shown purported hypersensitivity or idiosyncrasy to it or other nitrates or nitrites. Epinephrine and related compounds are ineffective in reversing the severe hypotensive events associated with overdose and are contraindicated in this situation.

WARNINGS: The benefits of SORBITRATE during the early days of an acute myocardial infarction have not been established If one elects to use organic nitrates in early infarction, hemodynamic monitoring and frequent clinical assessment should be used because of the potential deleterious effects of hypotension.

PRECAUTIONS: General: Severe hypotensive response, particularly with upright posture, may occur with even small doses of SORBITRATE The drug should therefore be used with caution in subjects who may have blood volume depletion from diuretic therapy or in subjects who have low systolic blood pressure (eg, below 90 mmHg) Paradoxical bradycardia and increased angina pectoris may accompany nitrate-induced hypotension. Nitrate therapy may aggravate the angina caused by hypertrophic cardiomyopathy

Marked symptomatic, orthostatic hypotension has been reported when calcium channel blockers and organic nitrates were used in combination Dose adjustment of either class of agents may be necessary.

Tolerance to this drug and cross-tolerance to other nitrates and nitrites may occur Tolerance to the vascular and antianginal effects of isosorbide dinitrate or nitroglycerin has been demonstrated in clinical trials, experience through occupational exposure, and in isolated tissue experiments in the laboratory The importance of tolerance to the appropriate use of isosorbide dinitrate in the management of patients with angina pectoris has not been determined However, one clinical trial using treadmill exercise tolerance (as an end point) found an 8-hour duration of action of oral isosorbide dinitrate following the first dose (after a 2-week placebo washout) and only a 2-hour duration of effect of the same dose after 1 week of repetitive dosing at conventional dosing intervals On the other hand, several trials have been able to differentiate isosorbide dinitrate from placebo after 4 weeks of therapy and, in open trials, an effect seems detectable for as long as several months.

Tolerance clearly occurs in industrial workers continuously exposed to nitroglycerin. Moreover, physical dependence also occurs since chest pain, acute myocardial infarction, and even sudden death have occurred during temporary withdrawal of nitroglycerin from the workers In clinical trials in angina patients, there are reports of anginal attacks being more easily provoked and of rebound in the hemodynamic effects soon after nitrate withdrawal The relative importance of these observations to the routine, clinical use of isosorbide dinitrate is not known However, it seems prudent to gradually withdraw patients from isosorbide dinitrate when the therapy is being terminated, rather than stopping the drug abruptly

Information for Patients: Headache may occur during initial therapy with SORBITRATE Headache is usually relieved by the use of standard headache remedies or by lowering the dose and tends to disappear after the first week or two of use.

Drug Interactions: Alcohol may enhance any marked sensitivity to the hypotensive effect of nitrates.

Isosorbide dinitrate acts directly on vascular smooth muscle, therefore, any other agent that depends on vascular smooth muscle as the final common path can be expected to have decreased or increased effect depending on the agent

Carcinogenesis, Mutagenesis, Impairment of Fertility: No long-term studies in animals have been performed to evaluate the carcinogenic potential of this drug A modified two-litter reproduction study in rats fed isosorbide dinitrate at 25 or 100 mg/kg/day did not reveal any effects on fertility or gestation or any remarkable gross pathology in any parent or offspring fed isosorbide dinitrate as compared with rats fed a basal-controlled diet

Pregnancy Category C: Isosorbide dinitrate has been shown to cause a dose-related increase in embryotoxicity (increase in mummified pups) in rabbits at oral doses 35 and 150 times the maximum recommended human daily dose There are no adequate and well-controlled studies in pregnant women SORBITRATE should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: It is not known whether this drug is excreted in human milk Because many drugs are excreted in human milk, caution should be exercised when SORBITRATE is administered to a nursing woman.

Pediatric Use: The safety and effectiveness of SORBITRATE in children has not been established

ADVERSE REACTIONS: Adverse reactions, particularly headache and hypotension, are dose-related In clinical trials at various doses, the following have been observed

Headache is the most common (reported incidence varies widely, apparently being dose-related, with an average occurrence of about 25%) adverse reaction and may be severe and persistent Cutaneous vasodilation with flushing may occur Transient episodes of dizziness and weakness, as well as other signs of cerebral ischemia associated with postural hypotension, may occasionally develop (the incidence of reported symptomatic hypotension ranges from 2% to 36%) An occasional individual will exhibit marked sensitivity to the hypotensive effects of nitrates and severe responses (nausea, vomiting, weakness, restlessness, pallor, perspiration, and collapse) may occur even with the usual therapeutic dose. Drug rash and/or exfoliative dermatitis may occasionally occur Nausea and vomiting appear to be uncommon. Case reports of clinically significant methemoglobinemia are rare at conventional doses of organic nitrates The formation of methemoglobin is dose-related and, in the case of genetic abnormalities of hemoglobin that favor methemoglobin formation, even conventional doses of organic nitrate could produce harmful concentrations of methemoglobin.

DOSAGE AND ADMINISTRATION: For the treatment of angina pectoris, the usual starting dose for sublingual SORBITRATE is 2.5 to 5 mg, for chewable tablets, 5 mg, for oral (swallowed) tablets, 5 to 20 mg, and for controlled-release forms, 40 mg

SORBITRATE should be titrated upward until angina is relieved or side effects limit the dose In ambulatory patients, the magnitude of the incremental dose increase should be guided by measurements of standing blood pressure

The initial dosage of sublingual or chewable SORBITRATE for prophylactic therapy in angina pectoris patients is generally 5 or 10 mg every 2 to 3 hours Adequate controlled clinical studies demonstrating the effectiveness of chronic maintenance therapy with these dosage forms have not been reported

SORBITRATE in oral doses of 10 to 40 mg given every 6 hours or in oral controlled-release doses of 40 to 80 mg given every 8 to 12 hours is generally recommended The extent to which development of tolerance should modify the dosage program has not been defined *The oral controlled-release forms of isosorbide dinitrate should not be chewed*

DOSAGE FORMS AVAILABLE: Sublingual Tablets (2.5, 5, 10 mg), Chewable Tablets (5, 10 mg); Oral Tablets (5, 10, 20, 30, 40 mg), Sustained Action Tablets (40 mg)

STUART PHARMACEUTICALS
Division of ICI Americas Inc.
Wilmington, DE 19897

STR-2282

ALBANY

Dec 5. **Cardiovascular Teaching Day.** Info: Office of Continuing Medical Education, St. Peter's Hospital, 315 South Manning Blvd, Albany, NY 12208. Tel: (518) 454-1333.

ONONDAGA

Dec 6–7. **36th Annual Postgraduate Course in Ophthalmology.** 24 Cat 1 Credits. Info: Program Coordinator, Office of Continuing Education, Upstate Medical Center, State University of New York, 750 East Adams St, Syracuse, NY 13210. Tel: (315) 473-4606.

AROUND THE NATION

CALIFORNIA

Dec 8–12. **Chest Radiology: Update and Review.** University of California San Diego, 24 Cat 1 Credits. Info: Dawne Ryals, Ryals and Associates, Box 610203, DFW Airport, TX 75261-0203. Tel: (214) 659-9590.

Dec 11–13. **Second International Research Symposium on Cellular Oncology.** Marquis Hotel, Palm Springs. 16 Cat 1 Credits. Info: Margaret Frederick, Assistant Director, Center of Health Education, 2810 Atlantic Ave, Long Beach, CA 90801. Tel: (213) 595-3823.

FLORIDA

Dec 27–29. **High Risk Obstetrics.** 12 Cat 1 Credits. *Dec 27–30.* **The First Thirty Minutes: The Boston City Hospital Approach to Medical and Pediatric Emergencies.** 15 Cat 1 Credits. Buena Vista Place at Disney World. Info: Department of Continuing Medical Education, Boston University School of Medicine, 80 East Concord St, Boston, MA 02118. Tel: (617) 638-4605.

WASHINGTON, DC

Dec 5–9. **Physicians in the 90s.** Info: American Medical Association, Hospital Medical Staff Services, 535 North Dearborn St, Chicago, IL 60610. Tel: (312) 645-4747.

FOR SEVERE COUGHS, CAN YOU REMEMBER THESE LETTERS?

You know Robitussin®, and the "DAC" formula gives you just what you need to manage those severe coughs complicated with nasal congestion . . . Decongestant And Codeine!

Robitussin®-DAC Ⓒ V

Each 5 ml (1 teaspoonful) contains:

Guaifenesin, USP	100 mg
Pseudoephedrine Hydrochloride, USP	30 mg
Codeine Phosphate, USP	10 mg

(Warning: May be habit forming)
Alcohol 1.4 percent

℞ ONLY

A-H-ROBINS
Pharmaceutical Division
Richmond, Virginia 23261-6609

PHYSICIANS IN RECOVERY

HELP!

The MSSNY wants the help of physicians who are recovering from chemical dependence (alcohol and other drugs) or mental illness. Other physicians attempting recovery need your guidance and support. The Committee for Physicians' Health (Physicians' Committee) can put you in touch. Please call or write.

We'll keep it confidential.

Barton A. Harris, M.D.
Medical Director
Committee for Physicians' Health
Medical Society of the
State of New York
P.O. Box 5404
Lake Sucess, NY 11042

516-488-7777

Medical Society of the State of New York
ANNUAL CME ASSEMBLY
MARCH 7-9, 1986
The New York Hilton, New York City
Combined With The Annual Meeting of
The House of Delegates
MARCH 6-9

MISCELLANEOUS—CONT'D

HAVE YOU BEEN CONVICTED OF A CRIME? YOUR MEDICAL LICENSE MAY BE AFFECTED! For consultation call Susan Kaplan, Attorney-at-Law, (212) 877-5998, concentrating in the vital legal problems affecting your professional license with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County.

ARCHITECTURAL DESIGN AND CONSTRUCTION: Designers, builders of custom professional offices. Consulting services available. Whether you're opening a new office, relocating, remodeling or expanding an existing office in New York City area and adjacent New Jersey counties. Donald Young, Inc. Tel: (718) 948-5191.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate, with no points, fees or charges of any kind. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions, announce the opening of their law office, Wood & Scher. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct preceedings, malpractice licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practioners. Wood & Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583, Telephone 914-723-3500.

MISCELLANEOUS—CONT'D

ARE YOU HAVING DIFFICULTIES GETTING YOUR MEDICAL LICENSE RESTORED? For consultation call Susan Kaplan, Attorney-at-Law, (212) 877-5998, concentrating in the vital legal problems affecting your professional license with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County.

BACK COPIES NEW YORK STATE JOURNAL OF MEDICINE

For a limited time, back copies may be purchased at the following rates plus postage.

1971-1980	**. . . $2.00**
1981-1983	**. . . 3.00**

Exception: for the special issue of December 1983 which costs $5.00.

Send your order to: Circulation Dept., New York State Journal of Medicine, 420 Lakeville Road, P.O. Box 5404, Lake Success, NY 11042.

Inquiries: (516) 488-6100, Circulation Dept.

New
Motrin® 800 TABLETS mg
ibuprofen

Extra strength
Convenience
Economy

Upjohn

The Upjohn Company
Kalamazoo, Michigan 49001 USA

© 1985 The Upjohn Company

J-5491 June 1985

Angina Protection with Benefits for a Lifetime

ONCE-DAILY CONTROL WITH HEART-SAVING BENEFITS

By reducing heart rate and cardiac contractility, INDERAL LA helps protect the heart from the potentially serious and debilitating consequences of ischemia. A highly effective antianginal agent for around-the-clock control of symptoms, INDERAL LA also provides cardiovascular protection for a sense of security in the years ahead.

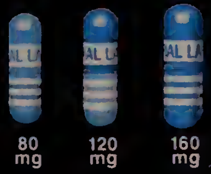
PROTECTION AND EXPERIENCE NO CALCIUM BLOCKER CAN MATCH

Unlike calcium blockers, INDERAL LA—either alone or with a nitrate—is recommended for early treatment of angina in the majority of patients. Equally important, INDERAL LA delivers the proven performance and safety profile of INDERAL tablets—confirmed by millions of patients during 16 years of clinical use.

START WITH 80 MG ONCE DAILY

Dosage may be increased to 160 mg once daily, as needed, to achieve optimal control. INDERAL LA should not be used in congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma. Please see next page for further details and brief summary of prescribing information.

ONCE-DAILY **INDERAL LA**
(PROPRANOLOL HCl) LONG ACTING CAPSULES



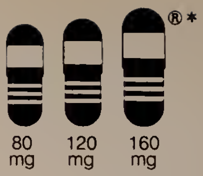
Ayerst.

The appearance of INDERAL LA capsules is a registered trademark of Ayerst Laboratories

JUST ONCE EACH DAY FOR SIMPLIFIED CORE THERAPY IN ANGINA

ONCE-DAILY INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES



Just once each day...

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. Inderal LA is formulated to provide a sustained release of propranolol hydrochloride Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules.

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

The mechanism of the antihypertensive effect of INDERAL has not been established. Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use. Effects on plasma volume appear to be minor and somewhat variable INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential The significance of the membrane action in the treatment of arrhythmias is uncertain

The mechanism of the antimigraine effect of propranolol has not been established. Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient But there are also situations in which sympathetic stimulation is vital For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

Propranolol is not significantly dialyzable

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock; 2) sinus bradycardia and greater than first degree block; 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)— PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e.g., dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA: Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes In these patients, it may be more difficult to adjust the dosage of insulin

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. *General:* Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL is not indicated for the treatment of hypertensive emergencies.

Beta adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

Clinical Laboratory Tests: Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Carcinogenesis, Mutagenesis, Impairment of Fertility Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

Pregnancy: Pregnancy Category C INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose

There are no adequate and well-controlled studies in pregnant women INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: INDERAL is excreted in human milk Caution should be exercised when INDERAL is administered to a nursing woman

Pediatric Use: Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

Cardiovascular: bradycardia; congestive heart failure; intensification of AV block; hypotension; paresthesia of hands; thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type.

Central Nervous System: lightheadedness; mental depression manifested by insomnia, lassitude, weakness, fatigue, reversible mental depression progressing to catatonia, visual disturbances, hallucinations; an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics

Gastrointestinal: nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

Allergic: pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory: bronchospasm.

Hematologic: agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained INDERAL LA should not be considered a simple mg for mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage of 640 mg may be required The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDERAL LA once daily The usual effective dose range is 160-240 mg once daily The dosage may be increased gradually to achieve optimum migraine prophylaxis If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use

*The appearance of INDERAL LA capsules is a registered trademark of Ayerst Laboratories

8833/384

Ayerst.
AYERST LABORATORIES.
New York, N.Y. 10017

> "...highly effective for both sleep induction and sleep maintenance"[3]

Sleep Laboratory Investigator
Pennsylvania

> "...onset of action is rapid...provides sleep with no rebound effect to agitate the patient the following day"[3]

Psychiatrist
California

> "...appears to have the best safety record of any of the benzodiazepines"[3]

Psychiatrist
California

After 15 years, the experts still concur about the continuing value of Dalmane (flurazepam HCl/Roche). It provides sleep that satisfies patients... and the wide margin of safety that satisfies you.

The recommended dose in elderly or debilitated patients is 15 mg. Contraindicated in pregnancy.

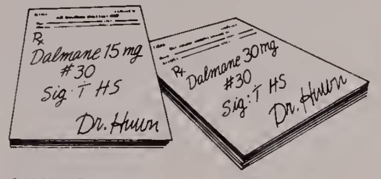
DALMANE®

flurazepam HCl/Roche Ⓒ

sleep that satisfies

15-mg/30-mg
capsules

References: **1.** Greenblatt DJ, Allen MD, Shader RI: *Clin Pharmacol Ther 21*:355-361, Mar 1977. **2.** Kales A, Kales JD: *J Clin Psychopharmacol 3*:140-150, Apr 1983. **3.** Tennant FS Jr: Treatment of patients with acute and chronic psychiatric conditions and insomnia, in *Hypnotics: A Clinical Update*, edited by Gripon EB, Princeton, NJ, Excerpta Medica, 1985, pp. 26-29. **4.** Kales J, *et al: Clin Pharmacol Ther 12*:691-697, Jul-Aug 1971. **5.** Kales A, *et al: Clin Pharmacol Ther 18*:356-363, Sep 1975. **6.** Kales A, *et al. Clin Pharmacol Ther 19*:576-583, May 1976. **7.** Kales A, *et al. Clin Pharmacol Ther 32*:781-788, Dec 1982. **8.** Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc 27*:541-546, Dec 1979. **9.** Dement WC, *et al. Behav Med*, pp 25-31, Oct 1978.



DALMANE®
flurazepam HCl/Roche Ⓒ

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits, in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended. Repeated therapy should only be undertaken with appropriate patient evaluation.

Contraindications: Known hypersensitivity to flurazepam HCl; pregnancy. Benzodiazepines may cause fetal damage when administered during pregnancy. Several studies suggest an increased risk of congenital malformations associated with benzodiazepine use during the first trimester. Warn patients of the potential risks to the fetus should the possibility of becoming pregnant exist while receiving flurazepam. Instruct patients to discontinue drug prior to becoming pregnant. Consider the possibility of pregnancy prior to instituting therapy.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Withdrawal symptoms rarely reported; abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase, and paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg recommended initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

ROCHE®

Roche Products Inc.
Manati, Puerto Rico 00701

#1 FOR SAFETY

Worldwide...a safety margin you can *count* on.

An exceptionally wide therapeutic margin that's confirmed by more than 15 years of use.[1-3] You're *more* than satisfied with the outstanding safety record of Dalmane.

And your patients are satisfied...they fall asleep quickly and sleep soundly until morning.[2-9] As always, caution patients about driving or drinking alcohol.

Please see references and summary of product information on reverse side

DALMANE®

flurazepam HCl/Roche ⓒⅣ

sleep that satisfies

Copyright © 1985 by Roche Products Inc. All rights reserved.

ROCHE®

NEW YORK STATE JOURNAL OF MEDICINE

STACKS

DECEMBER 1985 • Volume 85, Number 12

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

Contents

COMMENTARIES

Lactic dehydrogenase and malignancy 679
DERRY RIDGWAY, MD

Identifying cost effective diagnostic strategies 680
REX B. CONN, MD

The difficulties of doing research on cost effectiveness in health care 693
JOSEPH G. FELDMAN, DrPH

HISTORY

Linnaeus: right and wrong—On the 250th anniversary of his MD thesis and of his first edition of *Systema Naturae* 682
L. J. BRUCE-CHWATT, MD

RESEARCH PAPERS

Clinical significance of serum lactic dehydrogenase in chronic lymphocytic leukemia 685
TIN HAN, MD, L. J. EMRICH, MD, H. OZER, MD, P. A. REESE, MD, R. GAJERA, MD, G. A. GOMEZ, MD, E. S. HENDERSON, MD, M. L. BLOOM, MD, A. BHARGAVA, MD, J. FITZPATRICK, MD

The impact of technology on laboratory ordering strategies 690
CRAIG A. LEHMANN, MD, ALAN M. LEIKEN, PhD, DAVID F[illegible]

CPC

A 40-year-old man with chills, fever, weight loss, and a productive cough 694

CASE REPORTS

Intestinal fistula formation in a man with mycobacterial disease and AIDS 702
F. DUNCANSON, MD, J. PRABAKARAN, MD, S. SELVARAJ, MD, C. S. PITCHUMONI, MD

Disseminated cryptococcosis presenting as the adult respiratory distress syndrome 704
ELLIOTT N. PERLA, MD, SHLOMO MAAYAN, MD, STEVEN N. MILLER, MD, GITA RAMASWAMY, MD, HARVEY EISENBERG, MD

Pleural effusion due to communicating gastric ulcer 706
ROBERT D. BRANDSTETTER, MD, STEPHEN C. KLASS, MD, PHILIP GUTHERZ, MD, WALTER NEGLIA, MD, DONALD J. PINALS, MD

1985 INDEX 706a

MSSNY MEDICAL LIABILITY REFORM FUND HONOR ROLL 708

BOOK REVIEWS 710

OBITUARIES 714

MEDICAL MEETINGS AND LECTURES 4A

LIBRARY OF THE
COLLEGE OF PHYSICIANS
OF PHILADELPHIA

DEC 31 1985

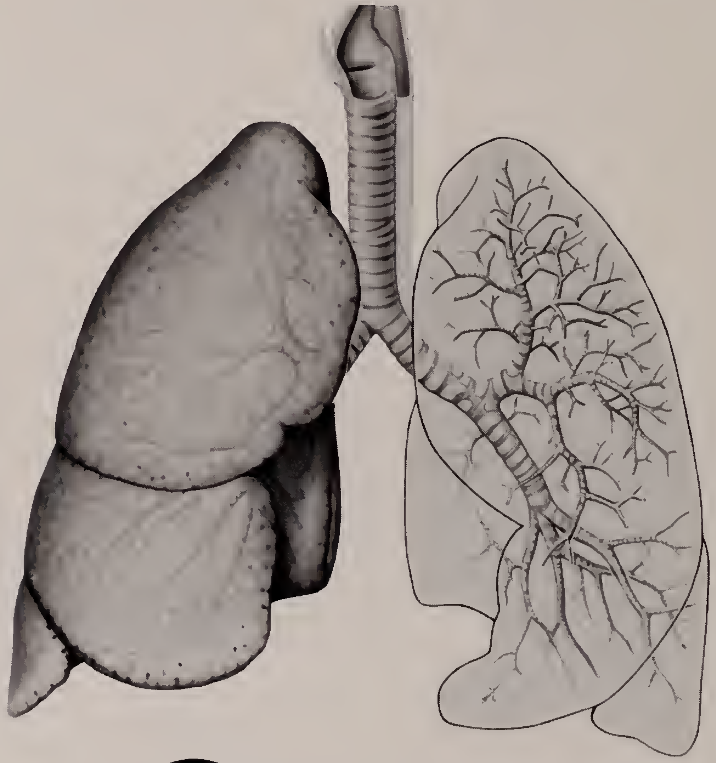
M. D. S.

Physician
85

3

1986 CME ASSEMBLY
Preliminary Program
See page 11A.

Consider the causative organisms…



Ceclor®
cefaclor
250-mg Pulvules® t.i.d.

offers effectiveness against the major causes of bacterial bronchitis

H. influenzae, *H. influenzae*, *S. pneumoniae*, *S. pyogenes*
(ampicillin-susceptible) (ampicillin-resistant)

Brief Summary Consult the package literature for prescribing information

Indications and Usage Ceclor* (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms

Lower respiratory infections including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae), Haemophilus influenzae* and *S pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor

Contraindication Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

Warnings IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy particularly to drugs

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins), therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics Such colitis may range in severity from mild to life-threatening

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C difficile* Other causes of colitis should be ruled out

Precautions *General Precautions* — If an allergic reaction to Ceclor* (cefaclor, Lilly) occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e g , pressor amines, antihistamines, or corticosteroids

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms Careful observation of the patient is essential If superinfection occurs during therapy, appropriate measures should be taken

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

Ceclor should be administered with caution in the presence of markedly impaired renal function Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur This has been observed with Benedict's and Fehling's solutions and also with Clinitest* tablets but not with Tes-Tape* (Glucose Enzymatic Test Strip, USP, Lilly)

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis

Usage in Pregnancy — Pregnancy Category B — Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor* (cefaclor, Lilly) There are, however, no adequate and well-controlled studies in pregnant women Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers — Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses Average levels were 0.18, 0 20, 0 21, and 0 16 mcg/ml at two, three, four, and five hours respectively Trace amounts were detected at one hour The effect on nursing infants is not known. Caution should be exercised when Ceclor is administered to a nursing woman.

Usage in Children — Safety and effectiveness of this product for use in infants less than one month of age have not been established

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below

Gastrointestinal symptoms occur in about 2 5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment Nausea and vomiting have been reported rarely

Hypersensitivity reactions have been reported in about 1 5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever) have been reported These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy No serious sequelae have been reported Antihistamines and corticosteroids appear to enhance resolution of the syndrome

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients)

Causal Relationship Uncertain — Transitory abnormalities in clinical laboratory test results have been reported Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician

Hepatic — Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40)

Hematopoietic — Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40)

Renal — Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200)

[061782R]

Note Ceclor* (cefaclor, Lilly) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever See prescribing information

© 1984, ELI LILLY AND COMPANY

Lilly

400441

Additional information available to the profession on request from
Eli Lilly and Company
Indianapolis Indiana 46285

Eli Lilly Industries, Inc.
Carolina Puerto Rico 00630

NEW YORK STATE JOURNAL OF MEDICINE

COMMITTEE ON PUBLICATIONS, LIBRARY, AND ARCHIVES

MILTON GORDON, MD, Chairman

PHILIP P. BONANNI, MD	FRANK C. MAXON, JR, MD
ELIZA H. CALDWELL, MD	RICHARD B. NOLAN, MD
URSULA A. MATULONIS (Medical Student)	JOHN T. PRIOR, MD

Editor	ALAN BLUM, MD
Deputy Editor and Book Review Editor	PASCAL JAMES IMPERATO, MD
Consulting Editor	JOHN T. FLYNN, MD
Consultant in Biostatistics	JOSEPH G. FELDMAN, DrPH
Production Manager	KATHLEEN WALSH TULLEY
Librarian and Administrative Assistant	ELLA ABNEY
Assistant Librarian	ELEANOR BURNS
Editorial Assistant	MILDRED ARFMANN
Secretary	KATHLEEN HUNTER SAGENDORPH
Advertising Director	ROBERT MILLER

ASSOCIATE EDITORIAL BOARD 1985

RANDALL BLOOMFIELD, MD	ROBERT LANDESMAN, MD
ROBERT D. BRANDSTETTER, MD	JAMES M. MORRISSEY, ESQ
JOHN S. DAVIS, MD	STEPHEN NORDLICHT, MD
BENNETT M. DERBY, MD	JOSEPH SCHLUGER, MD
ALFRED P. INGEGNO, MD	BJORN THORBJARNARSON, MD
FLORENCE KAVALER, MD	RODRIGO E. URIZAR, MD

NICHOLAS J. VIANNA, MD

Microform copies. This publication is available on microform film. Address orders to University Microfilms, 300 North Zeeb Road, Ann Arbor, MI 48106.

Advertising representatives. Synergistic Media Sales, 50 East 42nd St., Suite 504, New York, N.Y. 10017. Telephone: (212) 682-2200.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

Daniel F. O'Keeffe, MD,	*President*
Allison B. Landolt, MD,	*Past-President*
John A. Finkbeiner, MD,	*President-Elect*
Samuel M. Gelfand, MD,	*Vice-President*
John H. Carter, MD,	*Secretary*
George Lim, MD,	*Assistant Secretary*
Charles D. Sherman, Jr., , MD,	*Treasurer*
Morton Kurtz, MD,	*Assistant Treasurer*
Charles N. Aswad, MD,	*Speaker*
Seymour R. Stall, MD,	*Vice-Speaker*

Councilors

Term Expires 1986	Robert A. Mayers, MD, Westchester
	Thomas D. Pemrick, MD, Rensselaer
	Ralph E. Schlossman, MD, Queens
	Stanley A. Steckler, MD, Suffolk
	Robert J. Newborn, Albany *(Councilor representing the medical student membership)*
Term Expires 1987	David M. Benford, MD, New York
	Duane M. Cady, MD, Onondaga
	James M. Flanagan. MD, Wayne
	M. Theodore Tanenhaus, MD, Kings
Term Expires 1988	James H. Cosgriff, Jr, MD, Erie
	Richard A. Hughes, MD, Warren
	Antonio F. LaSorte, MD, Broome
	Sidney Mishkin, MD, Nassau

Trustees

G. Rehmi Denton, MD, Albany, *Chairman*
Ralph M. Schwartz, MD, New York
Edgar P. Berry, MD, New York
Richard D. Eberle, MD, Onondaga
Joseph F. Shanaphy, MD, Richmond
Victor J. Tofany, MD, Monroe
Bernard J. Pisani, MD, New York

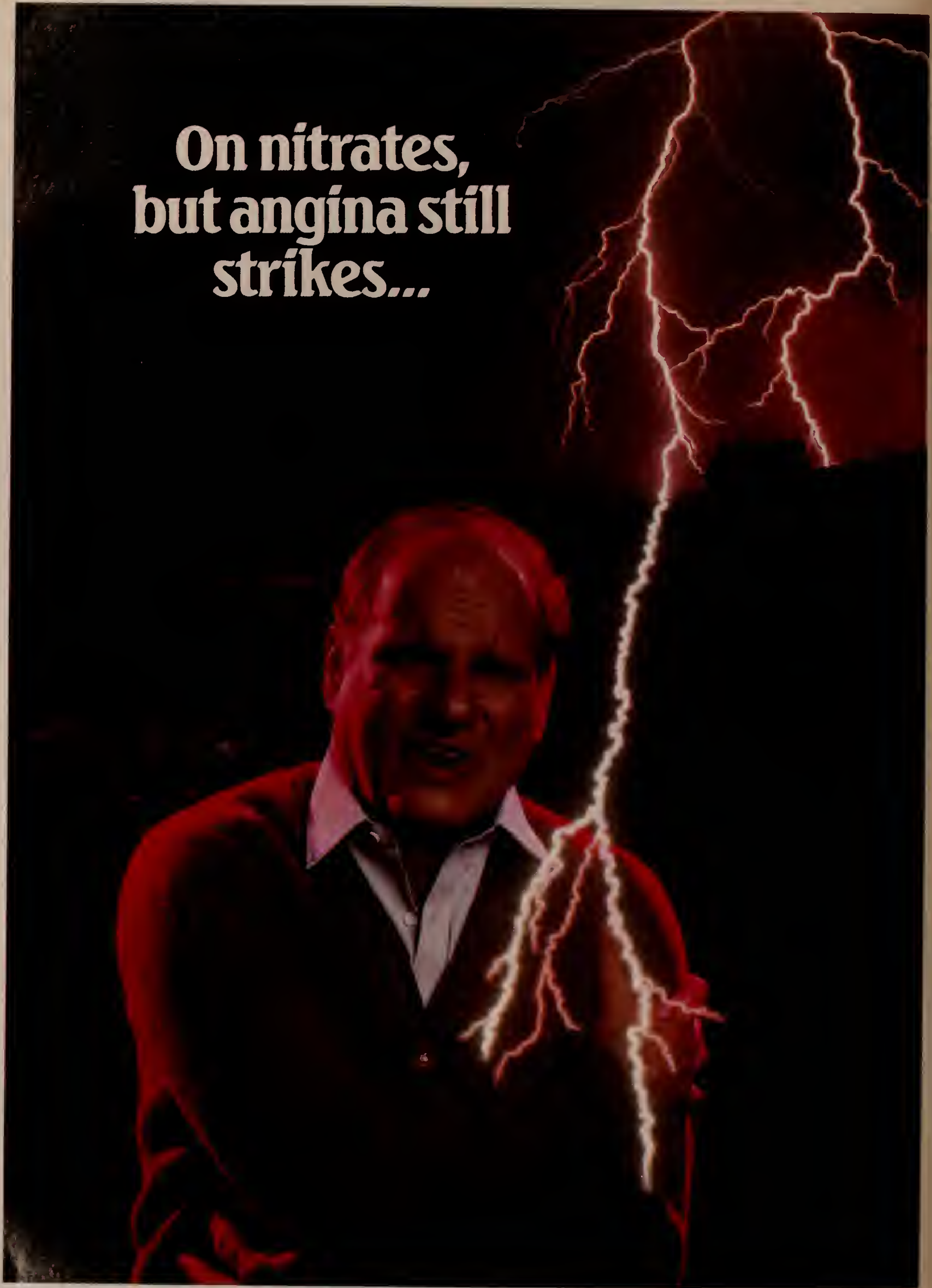
Executive Vice-President	George J. Lawrence, Jr, MD
Deputy Executive Vice-President	ROBERT J. O'CONNOR, MD
Executive Vice-President Emeritus	HENRY I. FINEBERG, MD
Executive Associate	IRMA A. ERICKSON
Director, Division of Scientific Publications	ALAN BLUM, MD
Director, Division of Management Services	GEORGE FORREST, JR
Assistant Director, Division of Governmental Affairs	JERRY S. HOFFMAN, ESQ
Director, Division of Communications	EDWARD HYNES
Director, Division of Insurance and Membership Benefits	BERNARD M. JACKSON
Director, Division of Membership	EUNICE SKELLY
Director, Division of Finance	PHILIP G. KEATING
Director, Division of Scientific Activities	PETER B. FARNSWORTH, MD
Director, Division of Advertising-Sales and Meeting Services	ROBERT MILLER
General Counsel	DONALD R. MOY, JD
Director, Division of Computer Services	IVAN H. NAJMAN
Acting Director, Division of Medical Services	MARIO MENGHINI
Director, Division of Long-Range Planning	DEBORAH R. SIEGEL
Consultant	EDWARD SIEGEL, MD

The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York. Editorial material published in the *Journal* is covered by copyright. Material may be photocopied for noncommercial scientific or educational use only. Special arrangements and permission are required from the author(s) of the particular article and from the editor for any other purpose. The editors of the *Journal* assume no responsibility for opinions and claims expressed in the articles contributed by individual authors.

SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $25.00 ($30.00 for all other countries). The single copy rate is $4.00 Domestic/Canada, and $5.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

EDITORIAL, CIRCULATION, and ADVERTISING OFFICES are located at 420 Lakeville Road, PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, 420 Lakeville Road, Lake Success, New York 11042.


On nitrates,
but angina still
strikes...

After a nitrate, add ISOPTIN®
(verapamil HCl/Knoll)

To protect your patients, as well as their quality of life, add Isoptin instead of a beta blocker.

First, Isoptin not only reduces myocardial oxygen demand by reducing peripheral resistance, but also increases coronary perfusion by preventing coronary vasospasm and dilating coronary arteries — both normal and stenotic. These are antianginal actions that no beta blocker can provide.

Second, Isoptin spares patients the beta-blocker side effects that may compromise the quality of life.

With Isoptin, fatigue, bradycardia and mental depression are rare. Unlike beta blockers, Isoptin can safely be given to patients with asthma, COPD, diabetes or peripheral vascular disease. Serious adverse reactions with Isoptin are rare at recommended doses; the single most common side effect is constipation (6.3%).

Cardiovascular contraindications to the use of Isoptin are similar to those of beta blockers: severe left ventricular dysfunction, hypotension (systolic pressure <90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no artificial pacemaker is present) and second- or third-degree AV block.

So, the next time a nitrate is not enough, add Isoptin... for more comprehensive antianginal protection without side effects which may cramp an active life style.

ISOPTIN. Added antianginal protection without beta-blocker side effects.

Please see brief summary on following page.

ISOPTIN®
(verapamil HCl/Knoll)
80 mg and 120 mg scored, film-coated tablets

Contraindications: Severe left ventricular dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (except in patients with a functioning artificial ventricular pacemaker), 2nd- or 3rd-degree AV block. **Warnings:** ISOPTIN should be avoided in patients with severe left ventricular dysfunction (e.g., ejection fraction < 30% or moderate to severe symptoms of cardiac failure) and in patients with any degree of ventricular dysfunction if they are receiving a beta blocker. (See *Precautions.*) Patients with milder ventricular dysfunction should, if possible, be controlled with optimum doses of digitalis and/or diuretics before ISOPTIN is used. (Note interactions with digoxin under *Precautions.*) ISOPTIN may occasionally produce hypotension (usually asymptomatic, orthostatic, mild and controlled by decrease in ISOPTIN dose). Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported. Such elevations may disappear even with continued treatment; however, four cases of hepatocellular injury by verapamil have been proven by rechallenge. Periodic monitoring of liver function is prudent during verapamil therapy. Patients with atrial flutter or fibrillation and an accessory AV pathway (e.g. W-P-W or L-G-L syndromes) may develop increased antegrade conduction across the aberrant pathway bypassing the AV node, producing a very rapid ventricular response after receiving ISOPTIN (or digitalis). Treatment is usually D.C.-cardioversion, which has been used safely and effectively after ISOPTIN. Because of verapamil's effect on AV conduction and the SA node, 1° AV block and transient bradycardia may occur. High grade block, however, has been infrequently observed. Marked 1° or progressive 2° or 3° AV block requires a dosage reduction or, rarely, discontinuation and institution of appropriate therapy depending upon the clinical situation. Patients with hypertrophic cardiomyopathy (IHSS) received verapamil in doses up to 720 mg/day. It must be appreciated that this group of patients had a serious disease with a high mortality rate and that most were refractory or intolerant to propranolol. A variety of serious adverse effects were seen in this group of patients including sinus bradycardia, 2° AV block, sinus arrest, pulmonary edema and/or severe hypotension. Most adverse effects responded well to dose reduction and only rarely was verapamil discontinued. **Precautions:** ISOPTIN should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacologic effects. Studies in a small number of patients suggest that concomitant use of ISOPTIN and beta blockers may be beneficial in patients with chronic stable angina. Combined therapy can also have adverse effects on cardiac function. Therefore, until further studies are completed, ISOPTIN should be used alone, if possible. If combined therapy is used, close surveillance of vital signs and clinical status should be carried out. Combined therapy with ISOPTIN and propranolol should usually be avoided in patients with AV conduction abnormalities and/or depressed left ventricular function. Chronic ISOPTIN treatment increases serum digoxin levels by 50% to 70% during the first week of therapy, which can result in digitalis toxicity. The digoxin dose should be reduced when ISOPTIN is given, and the patients should be carefully monitored to avoid over- or under-digitalization. ISOPTIN may have an additive effect on lowering blood pressure in patients receiving oral antihypertensive agents. Disopyramide should not be given within 48 hours before or 24 hours after ISOPTIN administration. Until further data are obtained, combined ISOPTIN and quinidine therapy in patients with hypertrophic cardiomyopathy should probably be avoided, since significant hypotension may result. Clinical experience with the concomitant use of ISOPTIN and short- and long-acting nitrates suggest beneficial interaction without undesirable drug interactions. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. *Pregnancy Category C*: There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor and delivery only if clearly needed. It is not known whether verapamil is excreted in breast milk; therefore, nursing should be discontinued during ISOPTIN use. **Adverse Reactions:** Hypotension (2.9%), peripheral edema (1.7%), AV block: 3rd degree (0.8%), bradycardia: HR < 50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%), elevations of liver enzymes have been reported. (See *Warnings.*) The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain: ecchymosis, bruising, gynecomastia, psychotic symptoms, confusion, paresthesia, insomnia, somnolence, equilibrium disorder, blurred vision, syncope, muscle cramp, shakiness, claudication, hair loss, macules, spotty menstruation. **How Supplied:** ISOPTIN (verapamil HCl) is supplied in round, scored, film-coated tablets containing either 80 mg or 120 mg of verapamil hydrochloride and embossed with "ISOPTIN 80" or "ISOPTIN 120" on one side and with "KNOLL" on the reverse side. Revised August, 1984. 2385

knoll **KNOLL PHARMACEUTICAL COMPANY**
30 NORTH JEFFERSON ROAD, WHIPPANY, NEW JERSEY 07981

2406

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meetings and lectures unless the information is submitted at least three months prior to the event.*

JANUARY 1986 AROUND THE STATE

MANHATTAN

Jan 3–4. **First Annual Infection/Immunology in Obstetrics and Gynecology.** 1 Cat 1 Credit. Info: Program Coordinator, c/o C.R.I.S.I.S., P.O. Box 4853, New York, NY 10185. Tel: (212) 696-0654.

Jan 8. **Internal Medicine Board Review.** Info: Registration Office, NYU Postgraduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Jan 9–11. **Surgical Courses in Lens Implantation.** Info: Ruth Green, Administrator, Ophthalmological Foundation of America, 823 United Nations Plaza, New York, NY 10017. Tel: (212) 986-4848.

Jan 21–24. **Bioregulatory Functions of Thrombin.** Barbizon-Plaza Hotel. Info: Conference Director, The New York Academy of Sciences, 2 East 63 St, New York, NY 10021.

BROOKLYN

Jan 8. **Bone Tumors: Osteogenic and Ewings Sarcoma.** *Jan 15.* **Endocrine Sequelae of Cancer Therapy.** *Jan 22.* **Adjuvant Chemotherapy of Breast Cancer—Current Status and Practice Implications.** *Jan 29.* **Chromosomal Basis of Human Cancer.** Info: Division of Hematology/Oncology, Department of Medicine, Interfaith Medical Center, 555 Prospect Pl, Brooklyn, NY 11238. Tel: (718) 240-1211.

NASSAU

Jan 8. **Human Teratogens: Environmental Factors Which Cause Birth Defects.** *Jan 22.* **Peripheral Nerve and Muscle**

(Continued on p 8A)

REGISTER NOW — ONLY ANNOUNCEMENT!

— 1986 —

13TH ANNUAL INTERSPECIALTY™
and
INTERNATIONAL OCULOPLASTIC SOCIETY CONGRESS

CASA DE CAMPO, DOMINICAN REPUBLIC
"The Ultimate Caribbean Resort"

FEBRUARY 15-22, 1986
Washington's Birthday Week

CONCURRENT PROGRAMS

	FACIAL PLASTIC SURGERY Room "A" 9:00 - 1:00 Program Chairmen	**OPHTHALMIC SURGERY** Room "B" 9:00 - 1:00 Program Chairmen
Monday, Feb 17	Cosmetic Facial Surgery NORMAN J. PASTOREK, M.D. New York, New York ANTONIO JORGE MESSINA, M.D. Santo Domingo, Dominican Republic	Cataract & IOL Surgery K. BUOL HESLIN, M.D. New York, New York
Tuesday, Feb 18	Management of Facial Skin Cancer MICHAEL J. ALBOM, M.D. New York, New York	Cataract & IOL Surgery DONALD L. PRAEGER, M.D. New York, New York
Wednesday, Feb 19	Reconstructive Facial Surgery GREGORY E. RAUSCHER, M.D. Hackensack, New Jersey	Retinal & Vitreal Surgery J. GRAHAM DOBBIE, M.D. Chicago, Illinois
Thursday, Feb 20	Oculoplastic Surgery PIERRE GUIBOR, M.D. New York, New York SANFORD D. HECHT, M.D. Boston, Massachusetts	Out-Patient Surgery HUNTER R. STOKES, M.D. Florence, South Carolina
Friday, Feb 21	Ocular Adnexal & Facial Cancer Surgery EUGENE O. WIGGS, M.D. Denver, Colorado	Keratorefractive Surgery HERBERT L. GOULD, M.D. New York, New York

Seminars ★ Exhibits ★ Panels ★ CME Credit ★ Discussion ★ Social Program

TRAVEL PACKAGES AVAILABLE

Name ______________________________

Street ______________________________

City, State, Zip ______________________________

____

Telephone ( ) ______________________________

____

Home Phone - For Emergency Use Only

Specialty: ☐ PLAS ☐ DERM ☐ OPH ☐ OTO ☐ O/MAX ☐ OTHER

CLIP & MAIL TO:

Congress Administrator
c/o PIERRE GUIBOR, M.D.
630 Park Avenue
New York, New York 10021
(800) 223-4500 (212) 734-1010

REGISTRATION FEE: $350

OPTIONAL SPOUSE/GUEST FEE: $150

MAKE CHECK PAYABLE TO:
The International Oculoplastic Society, Inc.

Classified Advertising

PHYSICIANS WANTED

PHYSICIAN-NY, Metropolitan areas. Air National Guard Units are seeking qualified physicians. Flexible active duty time, rewarding work, training opportunities for flying, travel, conferences, CME credits. Retirement benefits and insurance. Serve your state, country and community. For more information call 106th USAF Clinic, Suffolk Cty Air National Guard Base, Westhampton Bch., NY 11978. (516) 288-4200 Ext. 247.

INTERNAL MEDICINE, large practice outside Buffalo, New York for sale. For more information send C.V. to: Health Care Personnel Consulting, Inc., 400 GSB Building, One Belmont Avenue, Bala Cynwyd, PA 19004 (215) 667-8630.

SURGERY: GENERAL PRACTICE AVAILABLE—this GP/GS combination practice in upstate New York is a unique opportunity for the physician interested in a broad practice. Send C.V. to: Health Care Personnel Consulting, Inc., 400 GSB Building, One Belmont Avenue, Bala Cynwyd, PA 19004 (215) 667-8630.

PHYSICIANS WANTED—CONT'D

FAMILY PRACTITIONER—Established medical group needs a family practitioner. Located in the sun-belt and no state tax. Guaranteed salary and early partnership for the conscientious person. Contact clinic administrator 10737 Gateway West, El Paso, Texas 79935 or call (915) 592-0201.

ARIZONA BASED PHYSICIAN RECRUITMENT firm has opportunites Coast to Coast. "Professionals working with Professionals". Over 13 years experience. Call (602) 795-7474; or send CV to: Mitchell & Associates, Inc., 2761 N. Country Club Rd., Suite 202, Tucson, AZ 85716.

FAMILY PRACTICE—Hartford area Board Certified Family Physician seeks BE/BC associate to join well-established, expanding practice; partnership available. Attractive community of 60,000 located 1/2 hour from Hartford. A modern 250-bed hospital is conveniently located to the office, coverage is readily available, no obstetrics. First year financial and benefits package available. Contact: Anne Sondek, Four-NY Vermont Street, Buffalo, NY 13213, (716) 884-3700.

FAMILY PRACTICE, OB-GYN, PEDIATRICS. Small physician owned multi-specialty clinic, in Northern Indiana is expanding staff. Beautiful lake community of 10,000, near metropolitan centers. Salary plus bonus, mal practice paid plus other benefits. Please contact Kenneth E. Hoff, M.D., (219) 223-4337.

PHYSICIANS WANTED—CONT'D

EAST RANGE CLINICS, LTD. seeks physicians in the following specialties: Orthopedic Surgery, Ophthalmology and Internal Medicine (with special interest in cardiology, pulmonary medicine, or intensive care). Opportunity to join established practice with progressive multi-specialty group of 27 physicians, unlimited opportunity for outdoor recreation. Contact Gary Lishinski, Administrator, East Range Clinics, Ltd., 910 North Sixth Avenue, Virginia, MN 55792, (218) 741-0150.

GASTROENTEROLOGIST needed to join busy, strictly GI practice suburban Washington, DC. BC/BE. Must be warm, self-assured and outgoing, and possess manometric and advanced endoscopic skills. Send CV and cover letter to Dept. 401 c/o NYSJM.

BOARD CERTIFIED OB/GYN/FERTILITY PHYSICIAN NEEDED: established practice. Physician looking for an associate for a buy out situation. Office situated in beautiful suburban area, 26 miles north of Manhattan. Good hospital affiliation. Excellent opportunity for the right physician. For additional information, please send CV to: Mary Ann Hamburger, Mary Ann Hamburger Associates, 74 Hudson Avenue, Maplewood, New Jersey 07040.

PRACTICES AVAILABLE

SELLING PRACTICES IS OUR ONLY BUSINESS—Want to maximize the return on your investment? Before you buy or sell, call for a consultation to assure the best financial and transfer terms. We guide you through the entire sales process from initial meeting to closing. Serving 100 mile radius of New York City. Countrywide Business Brokerage, Inc., 319 East 24th Street, Suite 23-G, New York, NY 10010 (212) 686-7902.

MEDICAL PRACTICE SALES and Appraisals. We specialize in the valuation and selling of medical practices. If interested in buying or selling a medical practice, contact our Brokerage Division at: The Health Care Group, 400 GSB Building, Bala Cynwyd, PA 19004. (215) 667-8630.

INTERNAL MEDICINE—Active, long-established solo practice for sale. Nassau county; home-office fully equipped for 1 or 2 physicians, within minutes of several hospitals. Phone (516) 747-3072.

INTERNIST OR GENERAL PRACTITIONER—Unique opportunity exist to establish a practice in a fast-growing community of 30,000+ with low overhead structure. Area located near the ocean and within two hours of Philadelphia and Baltimore. Opportunity available immediately. Send inquiries to J. Stokes, P.O. Box 809, Milford, Delaware 19963.

VIABLE FAMILY PRACTICE FOR SALE—North Shore of Long Island. Home office, well equipped with X-Ray, EKG, Lab, etc. Call (516) 427-2180.

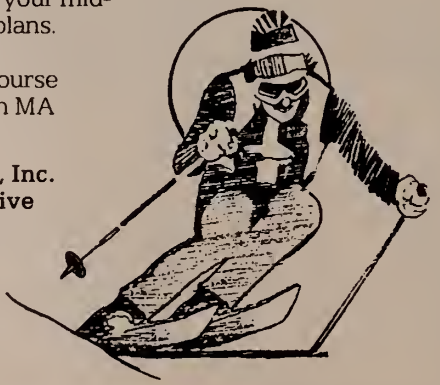
Tax-deductible Seminars At Five Vermont Ski Areas

Register now for **4-session course on medical malpractice** now offered throughout the season at Mt. Snow, Stratton-Bromley, Killington, Sugarbush and Stowe.

Flexible scheduling adjusts to your mid-week and/or weekend vacation plans. Tuition only $140.

For brochure with complete course description call 800/447-1175 (in MA 413/525-4785).

Eastern Educational Programs, Inc.
Benton Building, 15 Benton Drive
East Longmeadow, MA 01028



Added Control for
America's Coughs

Robitussin

Robitussin and codeine
Each 5 ml (1 teaspoonful) contains:
Guaifenesin, USP .... 100 mg
Codeine Phosphate, USP .... 10 mg
(*Warning. May be habit forming*)
Alcohol 3.5 percent
In a palatable, aromatic syrup

A-C® Ⓥ

The expectorant action of guaifenesin plus the Advantage of Codeine!

A·H·ROBINS
Pharmaceutical Division, Richmond, Virginia 23261-6609
©A.H. Robins Company 1985

MEDICAL & SURGICAL PROBLEMS IN WORKERS' COMPENSATION

Sponsored by: The American Academy of Legal & Industrial Medicine, Inc.

Co-sponsored by: New York University Post-Graduate Medical School

March 12–13, 1986, Sheraton Centre Hotel, 811 Seventh Ave & 53rd St., NYC, NY

Accreditation: As an organization accredited for continuing medical education, the New York University Post-Graduate Medical School certifies that this continuing medical education offering meets the criteria for 14 credit hours in Category 1 of the Physician's Recognition Award of the American Medical Association.

WEDNESDAY, MARCH 12TH:
Aspects of Stress in the Workplace
Moderator: Morton Gluck, MD
The legal, medical and insurance aspects of stress.
Third Party Payments
Moderator: Robert Katz, MD
Alternate methods of health care delivery and their impact on Worker's Compensation.

THURSDAY, MARCH 13TH:
Exercise in Illness and Health: Role of Rehabilitation
Moderator: Erwin Gonzalez, MD
Endurance and strength exercise; the arthritic joint; re-learning control of the injured brain.
Cervical Radiculopathy and Its Ramifications
Moderator: Otto Kestler, MD
The orthopedic, neurosurgical and hand surgeon's aspects

***For information and application please write:* Mathew H.M. Lee, M.D., F.A.C.P., Program Chairman, American Academy of Legal & Industrial Medicine, Inc., P.O. Box 10, Island Station, Roosevelt Island, NY 10044. Phone (212) 750-6781.**

Leave your mark on life.

By leaving even the smallest legacy to the American Cancer Society in your will, you can leave a loving and lasting impression on life. And giving life is the greatest way of leaving your mark on it.

AMERICAN CANCER SOCIETY®

For more information, call your local ACS Unit or write to the American Cancer Society, 4 West 35th Street, New York, NY 10001.

Disease. Info: Medical Education Department, Mercy Hospital, 1000 North Village Ave, Rockville Centre, NY 11570. Tel: (516) 255-2260.

Jan 15. **7th Annual Internal Medicine Board Review.** *Jan 17.* **A Day of Pediatric Radiology for the Clinician.** 7 Cat 1 Credits. Info: Ann J. Boehme, Associate Director for Continuing Education, Long Island Jewish Medical Center/Queens Hospital Center Affiliation, New Hyde Park, NY 11042. (718) 470-8650.

Jan 24. **Ultrasonic Treatment for Renal Stones.** Info: Medical Staff Office, Long Beach Memorial Hospital, 455 East Bay Dr, Long Beach, NY 11561. (516) 432-8000, ext. 2275.

SUFFOLK

Jan 7. **Infectious Disease: Office Diagnosis.** *Jan 14.* **Child Abuse.** *Jan 21.* **Neurology: Evaluation of the Comatose Patient.** Info: Arthur J. Schwager, MD, Director, Continuing Pediatric Medical Education, Huntington Hospital, Park Ave, Huntington, NY 11743. Tel: (516) 427-0530.

WESTCHESTER

Jan 22. **The Coming Changes in the Delivery of Health Care.** 3 ½ Cat 1 Credits. Info: Toni Calabrese Boelsen, Director, Public Relations, White Plains Hospital Medical Center, Davis Ave, White Plains, NY 10601. Tel: (914) 681-1118.

ROCKLAND

Jan 7. **Doppler Echocardiography.** 1 Cat 1 Credit. *Jan 14.* **Interventional Cardiology.** 1 Cat 1 Credit. Info: Ms. Myrna Hahn, Nyack Hospital, North Midland Ave, Nyack, NY 10960. (914) 358-6200, ext. 2509.

AROUND THE NATION

CALIFORNIA

Jan 11. **New Applications of Calcium Channel Blockers.** 4 Cat 1 Credits. Info: Extended Programs in Medical Education. Registration Office, Room 569-U. University of California, San Francisco, CA 94143-0958. Tel: (415) 476-5808.

(*Continued on p 36A*)

BALANCED CALCIUM CHANNEL BLOCKADE!

CARDIZEM®
(diltiazem HCl)
balances potent coronary vasodilation with a low incidence of side effects

Low incidence of side effects
CARDIZEM® (diltiazem HCl) produces an incidence of adverse reactions not greater than that reported with placebo therapy, thus contributing to the patient's sense of well-being.

*Cardizem is indicated in the treatment of angina pectoris due to coronary artery spasm and in the management of chronic stable angina (classic effort-associated angina) in patients who cannot tolerate therapy with beta-blockers and/or nitrates or who remain symptomatic despite adequate doses of these agents.

References:

1. Strauss WE, McIntyre KM, Parisi AF, et al: Safety and efficacy of diltiazem hydrochloride for the treatment of stable angina pectoris: Report of a cooperative clinical trial. Am J Cardiol 49:560-566, 1982.
2. Pool PE, Seagren SC, Bonanno JA, et al: The treatment of exercise-inducible chronic stable angina with diltiazem: Effect on treadmill exercise. Chest 78 (July suppl):234-238, 1980.

Reduces angina attack frequency*
42% to 46% decrease reported in multicenter study.[1]

Increases exercise tolerance*
In Bruce exercise test,[2] control patients averaged 8.0 minutes to onset of pain; Cardizem patients averaged 9.8 minutes ($P<.005$).

CARDIZEM®
(diltiazem HCl)

**THE BALANCED
CALCIUM CHANNEL BLOCKER**

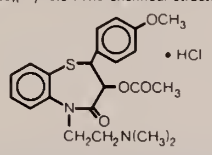
Please see full prescribing information on following page. 2/84

PROFESSIONAL USE INFORMATION

cardizem®
(diltiazem HCl)
30 mg and 60 mg tablets

DESCRIPTION

CARDIZEM® (diltiazem hydrochloride) is a calcium ion influx inhibitor (slow channel blocker or calcium antagonist). Chemically, diltiazem hydrochloride is 1,5-Benzothiazepin-4(5H)one,3-(acetyloxy)-5-[2-(dimethylamino)ethyl]-2,3-dihydro-2-(4-methoxyphenyl)-, monohydrochloride,(+) -cis-. The chemical structure is:



Diltiazem hydrochloride is a white to off-white crystalline powder with a bitter taste. It is soluble in water, methanol, and chloroform. It has a molecular weight of 450.98. Each tablet of CARDIZEM contains either 30 mg or 60 mg diltiazem hydrochloride for oral administration.

CLINICAL PHARMACOLOGY

The therapeutic benefits achieved with CARDIZEM are believed to be related to its ability to inhibit the influx of calcium ions during membrane depolarization of cardiac and vascular smooth muscle.

Mechanisms of Action. Although precise mechanisms of its antianginal actions are still being delineated, CARDIZEM is believed to act in the following ways:

1. Angina Due to Coronary Artery Spasm: CARDIZEM has been shown to be a potent dilator of coronary arteries both epicardial and subendocardial. Spontaneous and ergonovine-induced coronary artery spasm are inhibited by CARDIZEM.
2. Exertional Angina: CARDIZEM has been shown to produce increases in exercise tolerance, probably due to its ability to reduce myocardial oxygen demand. This is accomplished via reductions in heart rate and systemic blood pressure at submaximal and maximal exercise work loads.

In animal models, diltiazem interferes with the slow inward (depolarizing) current in excitable tissue. It causes excitation-contraction uncoupling in various myocardial tissues without changes in the configuration of the action potential. Diltiazem produces relaxation of coronary vascular smooth muscle and dilation of both large and small coronary arteries at drug levels which cause little or no negative inotropic effect. The resultant increases in coronary blood flow (epicardial and subendocardial) occur in ischemic and nonischemic models and are accompanied by dose-dependent decreases in systemic blood pressure and decreases in peripheral resistance.

Hemodynamic and Electrophysiologic Effects. Like other calcium antagonists, diltiazem decreases sinoatrial and atrioventricular conduction in isolated tissues and has a negative inotropic effect in isolated preparations. In the intact animal, prolongation of the AH interval can be seen at higher doses.

In man, diltiazem prevents spontaneous and ergonovine-provoked coronary artery spasm. It causes a decrease in peripheral vascular resistance and a modest fall in blood pressure and, in exercise tolerance studies in patients with ischemic heart disease, reduces the heart rate-blood pressure product for any given work load. Studies to date, primarily in patients with good ventricular function, have not revealed evidence of a negative inotropic effect; cardiac output, ejection fraction, and left ventricular end diastolic pressure have not been affected. There are as yet few data on the interaction of diltiazem and beta-blockers. Resting heart rate is usually unchanged or slightly reduced by diltiazem.

Intravenous diltiazem in doses of 20 mg prolongs AH conduction time and AV node functional and effective refractory periods approximately 20%. In a study involving single oral doses of 300 mg of CARDIZEM in six normal volunteers, the average maximum PR prolongation was 14% with no instances of greater than first-degree AV block. Diltiazem-associated prolongation of the AH interval is not more pronounced in patients with first-degree heart block. In patients with sick sinus syndrome, diltiazem significantly prolongs sinus cycle length (up to 50% in some cases).

Chronic oral administration of CARDIZEM in doses of up to 240 mg/day has resulted in small increases in PR interval, but has not usually produced abnormal prolongation. There were, however, three instances of second-degree AV block and one instance of third-degree AV block in a group of 959 chronically treated patients.

Pharmacokinetics and Metabolism. Diltiazem is absorbed from the tablet formulation to about 80% of a reference capsule and is subject to an extensive first-pass effect, giving an absolute bioavailability (compared to intravenous dosing) of about 40%. CARDIZEM undergoes extensive hepatic metabolism in which 2% to 4% of the unchanged drug appears in the urine. In vitro binding studies show CARDIZEM is 70% to 80% bound to plasma proteins. Competitive ligand binding studies have also shown CARDIZEM binding is not altered by therapeutic concentrations of digoxin, hydrochlorothiazide, phenylbutazone, propranolol, salicylic acid, or warfarin. Single oral doses of 30 to 120 mg of CARDIZEM result in detectable plasma levels within 30 to 60 minutes and peak plasma levels two to three hours after drug administration. The plasma elimination half-life following single or multiple drug administration is approximately 3.5 hours. Desacetyl diltiazem is also present in the plasma at levels of 10% to 20% of the parent drug and is 25% to 50% as potent a coronary vasodilator as diltiazem. Therapeutic blood levels of CARDIZEM appear to be in the range of 50 to 200 ng/ml. There is a departure from dose-linearity when single doses above 60 mg are given, a 120-mg dose gave blood levels three times that of the 60-mg dose. There is no information about the effect of renal or hepatic impairment on excretion or metabolism of diltiazem.

INDICATIONS AND USAGE

1. **Angina Pectoris Due to Coronary Artery Spasm.** CARDIZEM is indicated in the treatment of angina pectoris due to coronary artery spasm. CARDIZEM has been shown effective in the treatment of spontaneous coronary artery spasm presenting as Prinzmetal's variant angina (resting angina with ST-segment elevation occurring during attacks).
2. **Chronic Stable Angina (Classic Effort-Associated Angina).** CARDIZEM is indicated in the management of chronic stable angina. CARDIZEM has been effective in controlled trials in reducing angina frequency and increasing exercise tolerance.

There are no controlled studies of the effectiveness of the concomitant use of diltiazem and beta-blockers or of the safety of this combination in patients with impaired ventricular function or conduction abnormalities.

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt). Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, patients receiving CARDIZEM have exhibited reversible acute hepatic injury as evidenced by moderate to extreme elevations of liver enzymes. (See PRECAUTIONS and ADVERSE REACTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS).

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, exercise caution when CARDIZEM is administered to a nursing woman if the drug's benefits are thought to outweigh its potential risks in this situation.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences, as well as their frequency of presentation, are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%), AV block (1.1%). In addition, the following events were reported infrequently (less than 1%) with the order of presentation corresponding to the relative frequency of occurrence.

Cardiovascular:	Flushing, arrhythmia, hypotension, bradycardia, palpitations, congestive heart failure, syncope.
Nervous System:	Paresthesia, nervousness, somnolence, tremor, insomnia, hallucinations, and amnesia.
Gastrointestinal:	Constipation, dyspepsia, diarrhea, vomiting, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH.
Dermatologic:	Pruritus, petechiae, urticaria, photosensitivity.
Other:	Polyuria, nocturia.

The following additional experiences have been noted:

A patient with Prinzmetal's angina experiencing episodes of vasospastic angina developed periods of transient asymptomatic asystole approximately five hours after receiving a single 60-mg dose of CARDIZEM.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: erythema multiforme; leukopenia; and extreme elevations of alkaline phosphatase, SGOT, SGPT, LDH, and CPK. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

OVERDOSAGE OR EXAGGERATED RESPONSE

Overdosage experience with oral diltiazem has been limited. Single oral doses of 300 mg of CARDIZEM have been well tolerated by healthy volunteers. In the event of overdosage or exaggerated response, appropriate supportive measures should be employed in addition to gastric lavage. The following measures may be considered:

Bradycardia	Administer atropine (0.60 to 1.0 mg). If there is no response to vagal blockade, administer isoproterenol cautiously.
High-Degree AV Block	Treat as for bradycardia above. Fixed high-degree AV block should be treated with cardiac pacing.
Cardiac Failure	Administer inotropic agents (isoproterenol, dopamine, or dobutamine) and diuretics.
Hypotension	Vasopressors (eg, dopamine or levarterenol bitartrate).

Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

The oral/LO_{50}'s in mice and rats range from 415 to 740 mg/kg and from 560 to 810 mg/kg, respectively. The intravenous LD_{50}'s in these species were 60 and 38 mg/kg, respectively. The oral LD_{50} in dogs is considered to be in excess of 50 mg/kg, while lethality was seen in monkeys at 360 mg/kg. The toxic dose in man is not known, but blood levels in excess of 800 ng/ml have not been associated with toxicity.

DOSAGE AND ADMINISTRATION

Exertional Angina Pectoris Due to Atherosclerotic Coronary Artery Disease or Angina Pectoris at Rest Due to Coronary Artery Spasm. Dosage must be adjusted to each patient's needs. Starting with 30 mg four times daily, before meals and at bedtime, dosage should be increased gradually (given in divided doses three or four times daily) at one- to two-day intervals until optimum response is obtained. Although individual patients may respond to any dosage level, the average optimum dosage range appears to be 180 to 240 mg/day. There are no available data concerning dosage requirements in patients with impaired renal or hepatic function. If the drug must be used in such patients, titration should be carried out with particular caution.

Concomitant Use With Other Antianginal Agents:

1. **Sublingual NTG** may be taken as required to abort acute anginal attacks during CARDIZEM therapy.
2. **Prophylactic Nitrate Therapy** — CARDIZEM may be safely coadministered with short- and long-acting nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.
3. **Beta-blockers.** (See WARNINGS and PRECAUTIONS.)

HOW SUPPLIED

Cardizem 30-mg tablets are supplied in bottles of 100 (NDC 0088-1771-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1771-49). Each green tablet is engraved with MARION on one side and 1771 engraved on the other. CARDIZEM 60-mg scored tablets are supplied in bottles of 100 (NDC 0088-1772-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1772-49). Each yellow tablet is engraved with MARION on one side and 1772 on the other.

Issued 4/1/84

Another patient benefit product from
PHARMACEUTICAL DIVISION
MARION
LABORATORIES, INC.
KANSAS CITY, MISSOURI 64137

Lactic dehydrogenase and malignancy

The observation that some patients with cancer have elevated serum levels of the glycolytic enzyme lactic dehydrogenase (LDH) was made 31 years ago.[1] During the ensuing three decades, elevated serum LDH levels have been correlated with disease activity for a great variety of carcinomas, soft tissue and bone sarcomas, and hematopoietic and lymphoid cancers; however, a convincing explanation for enzyme elevation has not yet been offered. Proposed mechanisms that assume the excess to be tumor-derived must invoke increased production or release by the tumor cells. Direct correlation of serum LDH with other measures of tumor burden such as observable tumor size, presence of bone marrow involvement, or severity of systemic symptoms, has been possible only for some tumors.[2,3] It appears that elevated serum LDH cannot be explained by simple increases in the number of rapidly proliferating cancer cells. Since neoplastic cells show a relative dependence on glycolytic pathways for energy derivation, it has been suggested that tumor-specific characteristics of metabolism may determine the increased production of glycolytic enzymes, including LDH. However, the measurement of intracellular LDH from a variety of tumors has failed to demonstrate a consistent elevation, even when serum levels are abnormal.[4]

Further, the examination of serum isoenzyme patterns has confused rather than clarified the issue. LDH is a tetrameric enzyme made up of two different subunits in various proportions. The five resulting isoenzymes (labelled LDH 1-5) migrate at different rates on agarose gel. Different tissues are associated with characteristic LDH isoenzyme patterns, and in at least one tissue an embryologic evolution of isoenzyme patterns has been documented.[5] It might be expected that the isoenzyme pattern of serum LDH in patients with cancer would reflect the tissue of origin of the tumor. Instead, with rare exceptions,[6] the isoenzyme pattern in patients with cancer and elevated LDH is "nonspecific." This puzzling and unhelpful state of affairs prevails even when the isoenzyme pattern of intracellular LDH from tumor cells resembles a distinctive pattern found in the normal tissue from which the tumor originates.[5] Since neither increased production nor release of LDH by tumor cells can be documented, it has been proposed that secondary damage to normal tissue may account for a large part of elevated LDH. However, attempts to correlate the striking elevations of LDH seen in some patients with other evidence of tissue damage (such as measures of hepatic dysfunction or hemolysis) have not supported this hypothesis.

Although a convincing physiologic explanation of the high serum LDH levels in patients with cancer remains elusive, clinical oncologists have attempted to take advantage of this metabolic peculiarity for diagnosis and therapy. In no tumor system has this effort been pursued more intensively than in cancers of lymphoid origin. In this issue of the *Journal*, Han and coworkers[7] at Roswell Park Memorial Institute offer evidence that serum LDH levels correlate with the clinical stage at diagnosis for patients with chronic lymphocytic leukemia (CLL), and may be used as a predictor of survival time. Further, increasing LDH is shown to be a harbinger of progressive disease. CLL now joins Hodgkin's disease, non-Hodgkin's lymphomas, Burkitt's lymphoma, diffuse histiocytic lymphomas, diffuse poorly-differentiated lymphomas, and immunoblastic sarcoma as lymphoid malignancies for which the serum LDH level has prognostic inportance.[2,8] For each of these, the level of serum LDH at diagnosis is strongly correlated with extent of the disease. Whatever the explanation of high serum LDH levels in these patients, it appears to be related both to bulk and to the extent of the tumor. Most published series of patients with these tumors have failed to demonstrate a prognostic significance of LDH levels independent of standard prognostic indicators, and Han and associates show suggestive—although not statistically significant—prognostic importance of serum LDH for patients with CLL after standard staging procedures. In the clinical setting, the measurement of serum LDH can serve as a readily-available and inexpensive first approximation for prognostic grouping; it may even serve as an acceptable substitute for more costly or burdensome staging evaluations.

Conspicuous by their absence from the list presented above are the lymphoid cancers with lymphoblastic features: acute lymphoblastic leukemia (ALL) and non-Hodgkin's lymphomas with diffuse lymphoblastic histology. A correlation between serum LDH at diagnosis and standard prognostic characteristics of ALL is reported only rarely in the literature.[3,9,10] At Doernbecher Memorial Hospital for Children, serum LDH was determined in 104 children with ALL referred on the day of diagnosis for a five year period beginning in 1977. The LDH values ranged from 208 IU/L to 16,848 IU/L; 89 children (85%) had values greater than 300 IU/L. Levels for children who died or suffered a bone marrow relapse (mean = 1,503 ± 1,894, n = 38), or for children dying during the first month of therapy (mean = 1,812 ± 2,447, n = 14), were not different from the levels of the whole group (mean = 1,398 ± 2,209).

Although patients with diffuse lymphoblastic lymphomas are included in studies of non-Hodgkin's lymphomas demonstrating prognostic significance of serum LDH at diagnosis, no report has shown prognostic importance for serum LDH with this specific lymphoma subgroup. Among 15 patients with diffuse lymphoblastic lymphoma seen here in the last eight years, serum LDH has been a poor predictor of outcome. In contrast to other lymphoid malignancies, elevations in serum LDH observed in patients with lymphoblastic disease have no apparent prognostic implications.

An additional proposal for the diagnostic use of serum LDH levels in patients with lymphoid cancer rests on the observation of increased LDH levels in patients with ALL.[11] Thirty-one (30%) of 104 Oregon patients had values greater than 1,000 IU/L. Since levels this high rarely occur in patients with acute nonlymphoblastic leukemia (ANLL), the observation of very high serum LDH might assist the clinician in distinguishing ALL from ANLL.

In this age of multichannel serum chemistry analyses, the clinician will encounter an occasional patient with an unexpected and unexplained elevation of serum LDH. How should one proceed? The steady stream of reports about elevated serum LDH in patients with various malignant diseases may tempt the physician to search for an occult tumor; indeed, this course has been formally endorsed.[12] The outcome of such searches would be interesting and useful; perhaps this is the next logical chapter in the unfolding story of serum LDH and malignant disease.

DERRY RIDGWAY, MD
Assistant Professor
Pediatric Hematology/Oncology
Department of Pediatrics
The Oregon Health Sciences University
Portland, OR 97201

1. Hill BR, Levi C: Elevation of serum component in neoplastic disease. *Cancer Res* 1954; 14:513-515.

2. Hagberg H, Siegbahn A: Prognostic value of serum lactic dehydrogenase in non-Hodgkin's lymphoma. *Scand J Haematol* 1983; 31:49-56.

3. Suarez CR, Andreeff M, Miller DR: Serum LDH values in childhood acute leukemias and non-Hodgkin's lymphoma. *Med Pediatr Oncol* 1984; 12:89-92.

4. Ho AD, Fiehn W, Hunstein W: Intracellular lactic dehydrogenase and phosphohexose isomerase activity in leukaemia and malignant lymphoma. *Br J Haematol* 1982; 50:637-645.

5. Blatt J, Spiegel RJ, Papadopoulos NM, Lazarou SA, Magrath IT, Poplack DG: Lactate dehydrogenase isoenzymes in normal and malignant human lymphoid cells. *Blood* 1982; 60:491-494.

6. Otsu N, Hirata M, Miyazawa K, Tuboi S: Abnormal lactate dehydrogenase isoenzyme in serum and tumor tissue of a patient with neuroblastoma. *Clin Chem* 1985; 31:318-320.

7. Han T, Emrich LJ, Ozer H, et al: Clinical significance of serum lactate dehydrogenase in chronic lymphocytic leukemia. *NY State J Med* 1985; 85:685-690.

8. Schilling RF, McKnight B, Crowley JJ: Prognostic value of serum lactic dehydrogenase level in Hodgkin's disease. *J Lab Clin Med* 1982; 99:382-387.

9. Ho AD, Fiehn W, Hunstein W: Plasma and intracellular levels of lactate dehydrogenase, phosphohexose isomerase and lysozyme activity in acute leukemia. *Blut* 1984; 49:19-28.

10. Blatt J, Reaman G, Poplack DG: Current concepts. Biochemical markers in lymphoid malignancy. *N Engl J Med* 1980; 303:918-922.

11. Kornberg A, Polliack A: Serum lactic dehydrogenase (LDH) levels in acute leukemia: marked elevations in lymphoblastic leukemia. *Blood* 1980; 56:351-355.

12. Rotenberg Z, Weinberger I, Fuchs Y, Erdberg A, Davidson E, Agmon J: Elevation of serum lactic dehydrogenase levels as an early marker of occult malignant lymphoma. *Cancer* 1984; 54:1379-1381.

Identifying cost effective diagnostic strategies

The identification of the most cost effective diagnostic testing strategies is an attractive approach to reducing the cost of hospital care. In this issue of the *Journal*, Lehmann and associates describe an analysis of laboratory test utilization by the same group of resident physicians during rotations at two different hospitals. One of their conclusions is that providing individual chemical tests as required rather than providing only large panels of chemical tests may result in significantly higher costs. Unfortunately, the design of the study is flawed and the conclusions drawn may be spurious.

The study involved patients with three clearly-identifiable clinical conditions—myocardial infarction, congestive heart failure, and chronic airway obstruction—in the two hospitals. Hospital A was a 250-bed tertiary care facility in which approximately one third of the patients were women and two thirds were men. Hospital B was a 340-bed primary care facility in which all of the patients studied were men. The patient populations in the two hospitals cannot be considered identical.

The automated instruments in the chemistry laboratories of the two hospitals were quite different. Hospital A utilized a Beckman Astra 8 for the measurement of seven frequently ordered analytes, and a Boehringer Mannheim Hitachi on which 16 other analytes were measured providing a total of 23 different chemical tests. Both of these instruments are discrete sample analyzers on which any test or group of tests desired may be performed as requested. Hospital B utilized a Technicon SMAC that provides a panel of 20 chemical tests, and an SMA 6/60 that provides six chemical tests. The tests on the SMA 6/60 are completely duplicated on the SMAC. Thus there are a total of 20 different chemical tests available, and there would be no reason for ordering both the small panel and the large panel in hospital B. It is likely that some of the tests available on the Hitachi in hospital A but not available on the SMAC such as direct bilirubin, magnesium, and gamma glutamyl transferase were performed in hospital B by other methods, but these tests were not tabulated.

Although the authors state that the biochemical panels available in the two hospitals "were essentially the same," this is incorrect. In hospital A, if a physician wished to have the general chemical panel from the Hitachi plus glucose, electrolytes, BUN, and creatinine, it would be necessary to request both panels for a total of 23 tests. In contrast, there would be no point in ordering both the SMAC panel and the SMA 6/60 panel in hospital B since this would duplicate six tests. It is likely that this is the reason why more of the small panels were requested in hospital A than in hospital B.

In hospital A, any test or combination of tests could be requested at any time. In contrast, only the six tests on the SMA 6/60 were available at all times in hospital B; the SMAC panel of 20 tests was available only Monday through Friday, 8:00 AM to 5:00 PM. It appears logical to assume that one approach to decreasing laboratory utilization is simply to make some tests or panels unavailable. Another consideration is that it is unlikely that the medical staff in an acute care hospital would find it acceptable to limit their requests to those tests on the SMA 6/60 from Friday afternoon until Monday morning. It follows that some of the tests on the SMAC panel available only Monday through Friday from 8:00 AM to 5:00 PM were performed individually by other methods during the evening, during the night, and on weekends. Valid assessment of test utilization obviously requires tabulation of these individual tests; they were no doubt done and they should have been counted. Furthermore, it is likely that these tests were performed on a DuPont ACA, a superb instrument, but one with very high reagent costs that would affect the results of the authors' cost analysis.

Both the SMAC and the SMA 6/60 are continuous flow instruments that when in operation continuously pump reagents regardless of whether any patient samples are being analyzed. The SMAC processes 90 samples per hour, and if all 20 tests are requested it is without question an inexpensive approach to providing them. The Hitachi and Astra 8 are both discrete sample analyzers that may be maintained in the standby mode and use reagents only when samples are being analyzed. Most importantly, the discrete sample analyzers permit the performance of a single test or any combination of tests desired.

The conclusion that more laboratory tests were performed in hospital A than in hospital B is derived from the incomplete data presented. The authors considered the possibility that patients in hospital A had more severe illnesses and therefore required more laboratory work than those in hospital B. The results were different than anticipated: patients in hospital B had more complications (3.38 versus 2.26) and a longer length of stay (14.08 versus 11.89 days) than patients in hospital A. One might argue that the greater availability of laboratory services in hospital A, and therefore higher utilization, led to better patient care, shorter hospital stays, and fewer complications.

Overall, the data presented in this article are inadequate to make a valid judgment as to whether panel testing is more cost effective than obtaining only those tests that are required. Perhaps a more important question to ask is why a 20-test SMAC profile was performed on the average every other day for patients in hospital B and the 16-test Hitachi profile performed with equal frequency for patients with myocardial infarctions in hospital A even when individual tests could be ordered as necessary. Many of the tests in the large profiles in both hospitals might be useful in the initial assessment of the patient; however, repetitious measurement of triglycerides, cholesterol, total protein, albumin, or uric acid probably added little if anything to the quality of care of the patients.

The SMA-12, predecessor to the SMAC, was introduced approximately 20 years ago. At that time, it was less expensive to perform the 12-test profile than to perform two or three individual tests by manual methods. Indeed, many samples were put on the SMA-12 and all results were reported even though the physician might have requested only one or two of the group. The result is that we have trained an entire generation of physicians to equate chemical testing with an "SMA" or a "SMAC." With the advent of discrete sample analyzers, on which every additional test incurs an incremental cost, this approach to diagnostic testing is rapidly becoming obsolete.

Physicians still write orders for SMA-12s and SMACs, both trademarks of the Technicon Corporation, even though the tests may be performed on an entirely different instrument. It is easier and requires much less thought than selecting individual tests. An amusing experiment to try is to ask a group of physicians to name the 12 tests on an SMA-12 profile or the 20 tests on a SMAC profile. Most of them will be unable to recall all of the tests, and many of those who can will have to write down the tests as they are recalled. An even smaller number of physicians who write orders for SMAs and SMACs will be able to recall what the abbreviations represent (SMA 12/60 = Sequential Multiple Analysis; 12 tests/60 samples per hour; SMAC = Sequential Multiple Analysis plus Computer).

The basic problem of cost effective management in the laboratory is not the cost of panels versus individual tests because today's technology provides a cost-effective solution to individual testing as well as custom-designed panels for specific purposes. In order to solve the problem, physicians have to stop repetitious ordering of large chemical panels and select only those tests that will provide the information required to care for their patients.

REX B. CONN, MD
Professor of Pathology and Laboratory Medicine
Emory University School of Medicine
Atlanta, GA 30322

See also p 693.

Linnaeus: right and wrong

On the 250th anniversary of his MD thesis and of his first edition of *Systema Naturae*

L.J. Bruce-Chwatt, MD

Founded in 1739 "for the promotion of useful sciences," the Royal Swedish Academy of Sciences is situated in Frascati, at the periphery of Stockholm, in a 19th century mansion. One of the founders of the institution was Carl Linnaeus, considered today to be the father of systematic nomenclature of the living world and one of the most creative intellects of the 18th century (Fig 1). Together with other Swedish scientists of that and later periods—Klingenstierna the mathematician, Rosenstein the pediatrician, de Geer the entomologist, Swedenborg the philosopher, Celsius the astronomer, Sheele the chemist, and Thunberg the botanist—these remarkable men and their followers linked the Royal Swedish Academy of Sciences with the Royal Society of London and the Académie des Sciences in Paris.

Much has been written about Linnaeus' life and his botanical work but only recently has attention been drawn to his medical practice and early involvement in the study of intermittent fevers, or agues.[1-7] Garnham[2] is credited with completing the first English translation of Linnaeus' MD thesis, *New Hypothesis on the Cause of Intermittent Fevers*, and the description of events related to it.

Born in 1707, Carl Linnaeus was the son of a curate in Rashult, a small town in southern Sweden. He chose to study medicine first in Lund and then at Uppsala. However, Linnaeus never completed his doctorate that would allow him to practice, because he was given charge of a botanical garden and began the classification of plants according to their reproductive organs. With the support of the governor of Umea, Linnaeus went on two botanical expeditions to Lappland in 1732.

In spite of his interest in botany, Linnaeus felt that without a medical degree his future would be uncertain. However, Linnaeus had other reasons for wanting to practice medicine. In 1734 he met a young woman, the daughter of a physician, whom he wished to marry. Her father, however, would not consent to the marriage unless Linnaeus obtained the right to practice medicine. Fortunately, during his previous travels, Linnaeus had observed many cases of intermittent fever and felt that this would be a good subject for his thesis that he had already drafted.

In 1735 Linnaeus went to Holland, where educational opportunities were abundant and where he could meet many influential men. Moreover, this was a country with the richest collection of tropical flora that had been brought from the Indies. Linnaeus decided to attend Harderwijk, approximately 35 miles from Amsterdam, where a medical degree could be obtained rapidly and at little expense. The day after his arrival in Harderwijk Linnaeus passed a perfunctory examination and was given permission to print his dissertation. A week later he defended his thesis in a formal disputation before the University staff

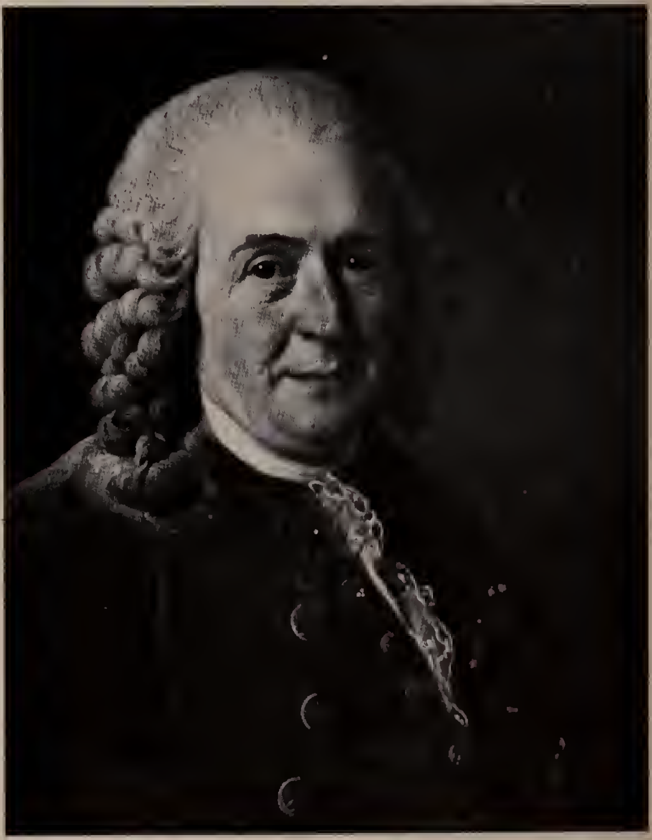


FIGURE 1. Carl Linnaeus (1707–1778), one of the founders and first president of the Royal Swedish Academy of Sciences. From a portrait in 1775 by A. Roslin. Courtesy of the Royal Swedish Academy of Sciences.

Address correspondence to Dr Bruce-Chwatt, Wellcome Museum of Medical Science, London, England.

and received his diploma the following week. Linnaeus then left Harderwijk and went to Leyden to attend the lectures of Boerhaave, the most famous Dutch physician and scientist of that time. While in Leyden, Linnaeus met Johan Gronovius, a physician and botanist, and showed him the manuscript of a small essay entitled, *Systema Naturae.* Gronovius immediately recognized it to be an original work of major importance and arranged for its publication.

Linnaeus spent the next three years in Holland and published nine books while cataloguing various botanical collections. In 1738 he returned to Sweden, where his reception was muted and his future uncertain. However, he married the physician's daughter and decided "to forsake Flora for Aesculapius" by starting a medical practice. He soon built up a profitable clientele by dispensing mercury to patients suffering from syphilis and gonorrhea. In 1741, however, Linnaeus requested of his patron, Prince Tessin, that he be released from general practice to become a professor of medicine at Uppsala. He then spent the rest of his life teaching and writing. His writings included such topics as dietetics, health precepts, ergot poisoning, leprosy, scurvy, and the use of medicinal plants. His compendium on pharmacology greatly improved the methods of drug prescription in Sweden.

However, there was a somber side to the latter part of his life. In 1758 Linnaeus passed through a period of bitterness and depression. He regretted the loss of his former lucrative practice, and he mistrusted his wife and children. In 1774 Linnaeus suffered a stroke and his physical and mental condition deteriorated markedly until his death in 1778.

The first edition of *Systema Naturae* consisted of the accumulated biological knowledge of two centuries of European geographic expansion. By creating an orderly classification of plants Linnaeus' work identified each species by using a universal binomial method. Without such a classification system the organization of new botanic and zoologic findings, brought about by the discovery of the New World, and due to British, Dutch, and French travels to the East, would have been impossible. Linnaeus is also responsible for defining the terms taxonomic genera and species. His descriptive style is a model of brevity and precision. He was no armchair philosopher, as his hypothesis of the cause of ague may suggest, but a full-blooded field worker, a lover of nature, and an inspiring personality.

As the number of discoveries accumulated, Linnaeus modified his *Systema Naturae* to adapt his classification to the growing wealth of knowledge. His early belief that all existing species were immutable products of an original creation gradually changed, and in the last edition of his book he admitted that there may be no limit to the possibilities of interbreeding and hybridization, although this was still far away from the evolutionary position.

In 1735 Linnaeus created a new hypothesis on the cause of intermittent fever; in the early 19th century these fevers were generally known as malaria. Linnaeus' hypothesis stated that ague was not caused by miasma, as had generally been believed, but by particles of clay that was common in the marshy soil of many areas of the country. "The smooth clay particles are present in both food and drink and, on ingestion, get into the blood where they eventually lodge in the smallest vessels and produce the disease." This affirmation is followed by a number of unverified comments on the symptoms of ague. Although he attempted to defend this theory from subsequent attacks, Linnaeus eventually admitted that he was in error.

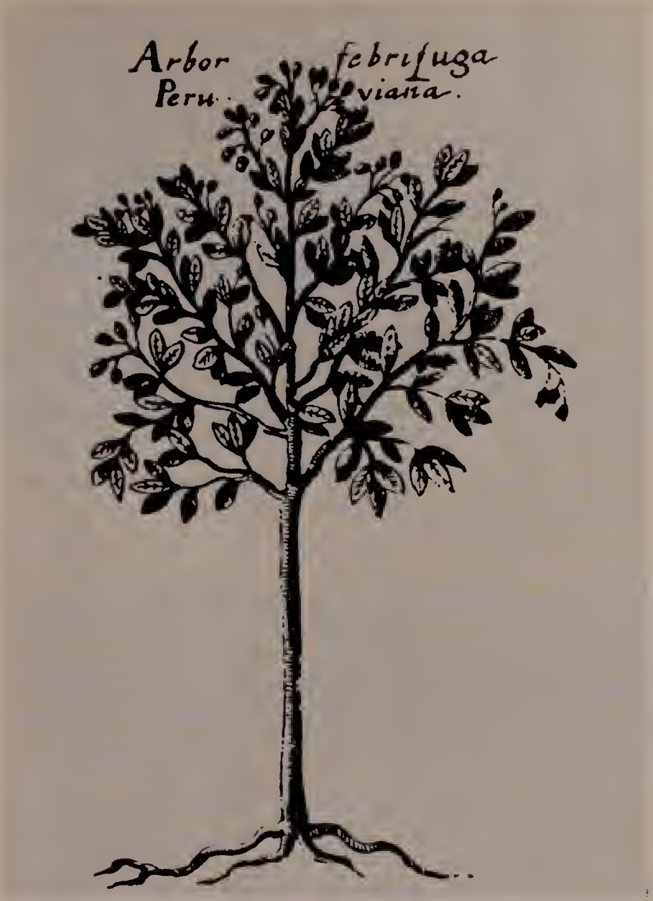
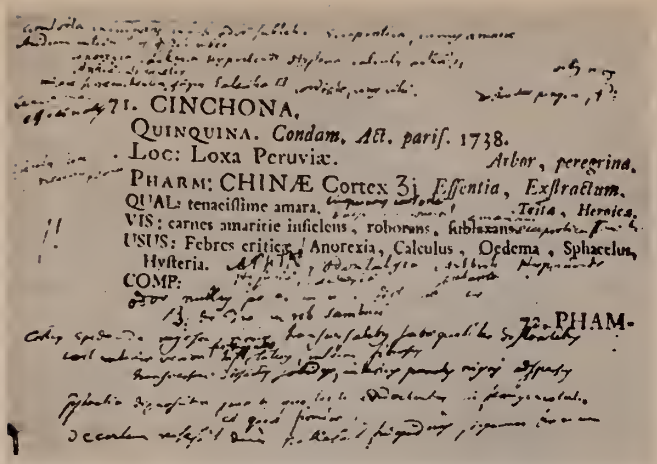


FIGURE 2. Drawing of the Cinchona tree, published in John Jonston's *Dendrographias* of 1662. Reproduced from [7]. Courtesy of the Wellcome Institute for the History of Medicine.

Intermittent fever or ague were common in southern Sweden in the 18th century. In 1754 Linnaeus described an outbreak of tertian ague in Uppsala when 300 people fell ill, including his wife. In a paper entitled *Febris Uppsaliensis* published in 1757, Linnaeus did not refer to clay particles, but advocated the removal of stagnant water from the city limits. It appears that he came down with an attack of ague in 1764, and used powdered Peruvian bark for treatment. However, his interest in this new remedy, brought from the New World, was not purely medical.

In 1749 Linnaeus gave the generic name of *Cinchona* to the fever bark tree in his *Materia Medica* (Fig 2).[8] In collecting all the information he could gather, Linnaeus read La Condamine's Memoir of 1738 (*Sur l'Arbre du Quinquina*) and corresponded with Bernard de Jussieu, the French naturalist who accepted Sebastiano Bado's uncertain story (*Anastasis Corticis Peruviae, seu China Chinae Defensio, 1663*) about the wife of the Viceroy of Peru. Apparently the Countess Chinchôn had been ill with ague and was cured by the infusion of the bark of a local tree. It is known today from the writings of several historians that the story was not true, but was transmitted to



71. CINCHONA.

QUINQUINA. *Condam. Act. pariſ.* 1738.

Loc: Loxa Peruviæ. *Arbor, peregrina.*

PHARM: CHINÆ Cortex ʒj. *Eſſentia, Extractum.*

QUAL: tenaciſſime amara. *Teſta, Heroica.*

VIS: carnes amaritie inficiens, roborans, ſublaxans

USUS: Febres criticæ, Anorexia, Calculus, Oedema, Sphacelus, Hyſteria.

COMP:

72. PHAM-

FIGURE 3. Reproduction of a page from the annotated copy of Linnaeus' *Materia Medica* (1749). Reproduced with permission from [7].

Bado by Antonius Bollus, a Genoese merchant who lived for many years in Peru. It also appears that in naming the fever bark tree Cinchona after the Countess, Linnaeus misspelled the proper name. This was probably caused by his following the writing of E.T. Geoffroy, a French naturalist, whose *Tractatus de Materia Medica* was published in 1741.

As noted in *Materia Medica* the common name of "quinquina Condam"—given for the genus *Cinchona* by La Condamine—is derived from the Quechua language (Fig 3). However, Jaramillo-Arango[7] found no evidence of such a derivation, although the possibility of confusion with another plant is likely. It must remembered that Linnaeus based his classification solely on the drawings of leaves, flowers, and fruits of the tree made by La Condamine in 1737, and the accompanying description.

The spelling of the genus *Cinchona* given by Linnaeus in 1749 and confirmed in his first and second issues of *Species Plantarum* has not been corrected by modern systematists. However in 1866 Sir Clements Markham requested such a correction, and always used it himself in his writings. The two International Botanical Congresses of 1930 and 1935 settled the matter in favor of the original spelling used by Linnaeus.[9] The International Code of Zoological Nomenclature, approved by the International Commission of 1958, agreed that the tenth edition of Linnaeus's *Systema Naturae* marks the beginning of the consistent application of binominal nomenclature in zoology; the date of January 1st, 1758 is given as the start of the use of the Rules of Priority. Therefore the error made by Linnaeus concerning the genus of the fever bark tree remains enshrined in the scientific nomenclature as a touching memento of the fact that even geniuses may be misled by incorrect references.

As discussed by Huxley in *Evolution—The Modern Synthesis*, living things tend to be divided into discontinuous group-units distinguishable by genetic differences in their character; convenience demands that these units be given specific names. The scale of this process of speciation and systematics may be better appreciated by pointing out that in the 10th edition of his *Systema Naturae* Linnaeus described less than 10,000 species of animals. In 1929, however, the total number of metazoan animal species was estimated as being between 750,000 and slightly over 1 million. A recent work celebrating the centenary of the Natural History Museum in London indicated the magnitude of this problem: "A careful census in 1976 led to an estimate of 22½ million insect specimens as the Museum's holdings, representing about 445,000 named species."[10] However, these are only about one half the known insect species. The number of animal species already identified is over one million, with approximately 10,000 new species added each year. If the 300,000 known plant species are included, then the total number of species, plant and animal combined, is approximately 1.5 million with an annual growth rate of about one percent.

REFERENCES

1. Blunt W: *The Compleat Naturalist.* Collins, London, 1971.
2. Garnham PCC: *Linnaeus' Thesis on Malaria in Sweden,* (Svenska Linnésällskapets Årsskrift). 1978, pp 80-97.
3. Gjertsen D: *The Classics of Science.* New York, Lillian Barber, 1984.
4. Goerke H: *Linnaeus.* New York, Charles Scribner & Sons, 1973.
5. Hagberg K: *Carl Linnaeus.* London, Jonathan Cape, 1952.
6. Jackson BD: *Linnaeus.* London, Witherby, 1923.
7. Jaramillo-Arango J: *The Conquest of Malaria.* London, William Heinemann Medical Books, 1959.
8. Bruce-Chwatt LJ, Zulueta J: *The Rise and Fall of Malaria in Europe.* London, Oxford University Press, 1978.
9. *Proceedings of the 300th Anniversary of the First Recognized Use of Cinchona.* St Louis, Missouri, Botanical Garden, 1931.
10. Stearns WT: *The Natural History Museum.* London, Heinemann Medical Books, 1981.

Clinical significance of serum lactate dehydrogenase in chronic lymphocytic leukemia

TIN HAN, MD, L.J. EMRICH, PHD, H. OZER, MD, P.A. REESE, PHD, R. GAJERA, MD, G.A. GOMEZ, MD, E.S. HENDERSON, MD, M.L. BLOOM, MD, A. BHARGAVA, PHD, J. FITZPATRICK, MD

ABSTRACT. A highly significant relationship (P <0.0001) between clinical stage and lactate dehydrogenase (LDH) level was observed in a study of 101 patients with chronic lymphocytic leukemia (CLL). There was also a significant relationship (P <0.02) noted between LDH level and absolute lymphocyte count, alkaline phosphatase level, and ^{3}H-thymidine uptake of peripheral blood leukocytes. Further, there was a highly significant relationship (P = 0.0001) between LDH activity and survival time. Thirteen of 60 patients with normal LDH levels died; the estimated median survival time for these patients exceeded 156 months. In contrast, 16 of 41 patients with elevated LDH levels died; the median survival time for these patients was only 47 months. When both clinical stage and LDH activity were evaluated for survival using the likelihood ratio test under the proportional hazards model of Cox, a highly significant association between clinical stage and survival time was observed (P <0.00005) while the relationship between LDH activity and survival time was only suggestive (P = 0.08). It can therefore be concluded that the evaluation of LDH activity in patients with CLL may be useful in defining the overall prognostic aspects of the disease.

(NY State J Med 1985; 85:685-690)

Chronic lymphocytic leukemia (CLL) is a disease known to have a variable course; some patients die within a few years after diagnosis while others live for more than a decade. Therefore the prognosis of patients with CLL apparently depends on several factors. Rai and colleagues[1] proposed a clinical staging system defined by anatomic involvement in lymphoid areas and the presence or absence of anemia or thrombocytopenia. This staging system has proven to be a reliable predictor of survival time, whether applied at the time of diagnosis or subsequently during the course of the disease. Other factors not used in the staging system but that have been shown to be significant include lymphocytosis,[2] ^{3}H-thymidine uptake by peripheral blood leukocytes,[3] bone marrow infiltration pattern,[4-6] and abnormal karyotype of leukemic B lymphocytes.[7]

Serum lactate dehydrogenase (LDH) activity was reported to be increased in patients with neoplastic diseases including solid tumors, lymphomas, and leukemia over 25 years ago.[8,9] However, no correlation was established between elevations of LDH and any specific neoplastic disease or any histologic type within a particular disease. During the past decade several investigators have reported the clinical and prognostic value of LDH activity in patients with non-Hodgkin's lymphoma, Burkitt's lymphoma, and acute lymphoblastic leukemia.[10-22] The association of LDH activity with the clinical status of patients with CLL appears not to have been examined.

The present study was conducted for the following reasons: 1) to assess the relationship between LDH activity and clinical stage; 2) to assess the relationship between LDH activity and clinical, laboratory, and investigational data including cell surface markers and cytogenetics; 3) to determine whether or not LDH activity could predict the subsequent course of Stage 0 disease; and 4) to determine whether or not LDH activity correlates with survival time in patients with CLL.

PATIENTS AND METHODS

One hundred and one patients (58 men and 43 women) with CLL seen at Roswell Park Memorial Institute were studied. The patients' ages ranged from 43 years to 86 years and greater than half of the patients were over 60 years of age. Ten patients with Stage 0 disease who were initially seen from 1955 to 1966 and remained without progression of disease were included in this study. All of the patients had active disease and were previously untreated at the time of the LDH determination. The diagnosis of CLL was usually made from a few weeks to a few months prior to referral to Roswell Park Memorial Institute. The diagnosis was then confirmed by surface membrane immunoglobulin analysis in most cases.[23]

From the Departments of Medical Oncology (Drs Han, Ozer, Gajera, Gomez, and Henderson), Biomathematics (Drs Emrich and Reese) and Laboratory Medicine (Drs Bhargava and Fitzpatrick), Roswell Park Memorial Institute, Buffalo, NY and the Department of Medicine (Dr Bloom), State University of New York at Buffalo, NY.

This study was supported in part by US PHS Grant nos. CA-2599, CA-5834, CA-27691, and CA-16056 from the National Cancer Institute, Department of Health and Human Services and was presented at the 35th National Meeting of the American Association for Clinical Chemistry, New York City, July 27, 1983.

Address correspondence to Dr Han, Associate Chief, Department of Medical Oncology, Roswell Park Memorial Institute, 666 Elm Street, Buffalo, NY 14263.

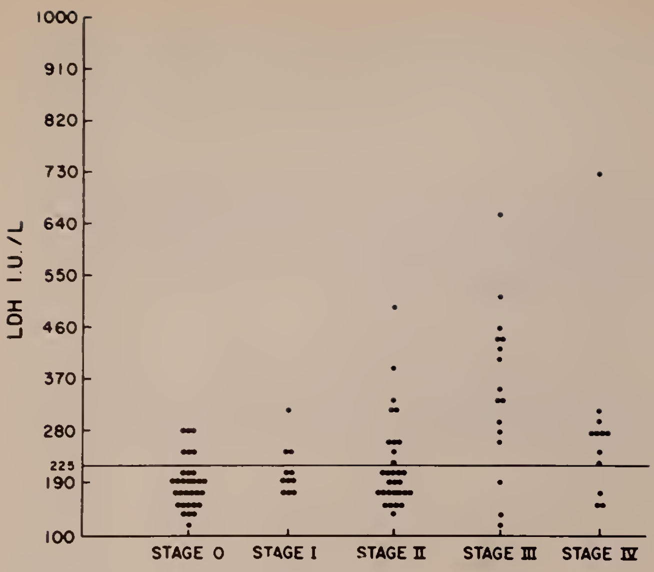


FIGURE 1. LDH levels by clinical stage.

In 89 cases studied, the CLL was of B-cell origin ($\mu\delta$, 54; μ,31; γ,3; κ,49; λ,40). Clinical staging according to Rai's classification[1] indicated that the patients were widely distributed among various stages of disease (Stage 0, 31; Stage I, 11; Stage II, 30; Stage III, 16; Stage IV, 13). Lymphocytosis ranged from 10,000 to greater than 500,000/μL. In 86 patients serum electrophoresis, serum immunoelectrophoresis, and quantitative immunoglobulins (IgM, IgG, and IgA) by radial immunodiffusion methods were performed. Sixty-one of these 86 patients also had urinary light chain determinations by immunoelectrophoresis. In vitro ^{3}H-thymidine uptake of peripheral blood leukocytes was performed in 43 patients, and cytogenetic analyses of polyclonal B-cell mitogen-stimulated leukemic B cells was performed in 48 patients as previously described.[7,24] Patients with Stage 0 or Stage I disease did not receive therapy. Patients with Stage II, III, or IV received combination chemotherapy—either chlorambucil and prednisone (as in most cases); chlorambucil, predisone, and streptonigrin; or cyclophosphamide and prednisone. A few selected patients with Stage II disease were treated with splenic irradiation.

Serum LDH determinations were carried out by a previously published method from 1967 through 1971[25]; since 1972 LDH determinations were carried out on a Technicom SMA IIC at 37°C according to the method of Kessler and colleagues.[26]

All analyses involving LDH activity were performed by comparing patients with normal levels ($\leq$225 IU/L) with those with high levels ($>$225 IU/L), although the results were virtually unchanged when LDH was treated as a continuous variable. Mann-Whitney tests[27] were used to compare the two groups with respect to clinical and immunologic parameters. Chi-square tests were used to compare the two groups with respect to sex, clinical stage, bone marrow infiltration pattern, surface membrane phenotype of leukemic cells, urinary light chain, Coomb's test, ^{3}H-thymidine uptake, and cytogenetic results.

Estimated survival distributions were calculated by the method of Kaplan and Meier.[28] Tests of significance with respect to survival distributions for different stages and for LDH levels were based on the log-rank test.[29] Likelihood ratio tests under the proportional hazards model of Cox[30] were used to determine the relative importance of clinical stage and LDH activity.

RESULTS

The distribution of LDH levels among the five clinical stages is shown in Figure 1. The lowest mean value was seen in patients with Stage 0 disease and the highest mean value was seen in patients with Stage III disease. Of 31 patients with Stage 0, 25 patients (81%), including ten in whom the LDH determinations were carried out one to 12 years after the time of diagnosis, had normal LDH levels and only six (19%) had elevated LDH levels. In contrast, of 29 patients with Stage III or IV disease only seven (24%) had normal LDH levels while 22 (76%) had elevated LDH levels. Of 41 patients with Stage I or II disease 28 (68%) had normal LDH levels and 13 (32%) had elevated LDH levels. The difference in the distributions of normal and elevated LDH levels among patients with all stages of CLL was highly significant ($P = 0.0001$).

Relationships between LDH levels and clinical and immunobiologic data are shown in Tables I and II. Patients with high LDH levels were somewhat older than those with normal levels ($P = 0.05$). Median values of immunoglobulin levels (IgM, IgG, or IgA), E$^+$ lymphocytes, Fc$^+$ lymphocytes, and complement$^+$ lymphocytes were similar between patients with normal LDH levels and those with high levels. Absolute lymphocyte counts were significantly higher in patients with high LDH levels than in those with normal levels ($P = 0.01$). Alkaline phosphatase levels were also significantly higher in patients with high LDH levels than in those with normal values ($P = 0.01$). As expected, the hemoglobin and hematocrit values were significantly higher ($P = 0.0001$) in the normal LDH group than in the high group. Unexpectedly, although the median platelet count was higher in patients with normal LDH levels than in those with high levels, the difference was not statistically significant (Table I).

There was a lack of a statistically significant association between LDH activity and sex, urinary monoclonal free light chain type, leukemic phenotype, karyotypic abnormalities, or Coomb's positivity. Of 29 patients with stage III or IV CLL, six had Coomb's positive autoimmune hemolytic anemia and two had Coomb's negative autoimmune hemolytic anemia. Three of six patients with Coomb's positive autoimmune hemolytic anemia, and one of two patients with Coomb's negative autoimmune hemolytic anemia showed an increased LDH activity. Of 21 stage III or IV patients without autoimmune hemolytic anemia, 18 exhibited an elevated LDH activity. Of 19 patients with diffuse bone marrow infiltration patterns, six (32%) had normal LDH levels and 13 (68%) had elevated LDH levels. In contrast, of 55 patients with nondiffuse infiltration patterns, 32 (58%) had normal LDH levels whereas 23 (42%) had elevated LDH levels. However, this difference was only marginally significant ($P = 0.08$). Of 25 patients with a low ^{3}H-thymidine uptake of peripheral leukocytes 20 (80%) had normal LDH levels and the

TABLE I. Relationship between LDH Levels and Clinical and Immunologic Data

Clinical and immunologic data	Median value for normal LDH level (no. of patients)	Median value for high LDH level (no. of patients)	P value*
Age (years)	57.5 (60)	65.3 (41)	0.05
Absolute lymphocytes × 10^3/μL	27.1 (60)	45.6 (40)	0.01
E$^+$ lymphocytes (%)	8.8 (47)	8.3 (37)	0.71
Fc$^+$ lymphocytes (%)	29.0 (40)	23.5 (28)	0.92
Complement$^+$ lymphocytes (%)	39.0 (46)	43.0 (37)	0.84
Alkaline phosphatase (IU/L)	61.5 (60)	85.8 (41)	0.01
Hemoglobin (g/dL)	14.0 (60)	11.7 (41)	0.0001
Hematocrit (%)	43.0 (56)	37.0 (41)	0.0001
Platelets × 10^3/μL	177.3 (58)	137.0 (41)	0.18
IgM (mg/dL)	63.5 (50)	62.5 (36)	0.55
IgG (mg/dL)	930.5 (50)	769.0 (36)	0.82
IgA (mg/dL)	84.0 (49)	79.5 (36)	0.97

* P values were calculated using a Mann-Whitney test.

TABLE II. Relationships between LDH Levels and Immunobiologic Data

Clinical and investigational data	No. of patients (%) with normal LDH level	No. of patients (%) with high LDH level	P value*
Sex			
Men	33 (57%)	25 (43%)	0.70
Women	27 (63%)	16 (37%)	
Bone marrow infiltration pattern			
Diffuse	6 (32%)	13 (68%)	0.08
Non-diffuse	32 (58%)	23 (42%)	
Leukemic phenotype			
Heavy chain: μ^{δ}	30 (56%)	24 (44%)	0.99
μ	18 (58%)	13 (42%)	
Light chain: κ	24 (49%)	25 (51%)	0.19
λ	26 (65%)	14 (35%)	
Coomb's test			
Positive	3 (50%)	3 (50%)	0.99
Negative	31 (55%)	25 (45%)	
Urinary monoclonal free light chain			
Negative	26 (68%)	12 (32%)	0.16
Positive (κ)	8 (44%)	10 (56%)	
Positive (λ)	2 (40%)	3 (60%)	
Cytogenetic data			
Normal karyotype	16 (57%)	12 (43%)	0.27
Abnormal karyotype	6 (35%)	11 (65%)	
^{3}H-thymidine uptake			
Lower ($\leq$5,000 dpm)	20 (80%)	5 (20%)	0.02
Higher (>5,000 dpm)	7 (39%)	11 (61%)	

* *P* values were calculated using a Chi-square test.

remaining five (20%) had elevated LDH levels. In contrast, of 18 patients with a high ^{3}H-thymidine uptake, 11 (61%) had elevated LDH activity whereas seven (39%) had normal LDH activity. The difference was statistically significant ($P = 0.02$) (Table II).

The relationship between LDH levels and disease progression in Stage 0 patients is shown in Table III. Of the 31 Stage 0 patients studied, nine progressed to advanced stages within five years of diagnosis. Of the remaining 22 patients, 12 were followed for at least five years, giving a total of 21 Stage 0 patients whose five-year progression status could be determined. Seventeen of these patients had normal LDH levels; 11 (65%) remained in Stage 0 and six (35%) progressed. In contrast, of four patients with elevated LDH levels, three (75%) progressed to advanced stages and only one (25%) did not progress. The difference, however, was not statistically significant.

In 11 of 17 Stage 0 patients with initially normal LDH levels, sequential determinations were carried out; initial and sequential LDH values of these 11 patients are shown in Table IV. In each of six nonprogressive Stage 0 cases, the subsequent LDH activity remained within normal limits. However, in four of five Stage 0 cases who subsequently progressed to higher stages (Stage I, one; Stage II and Stage III, two each), the LDH activity rose above normal levels at the time of disease progression.

TABLE III. Relationship between LDH Activity and Five-Year Progression Status in Patients with Stage 0 Disease

LDH activity	No. of patients who progressed (%)	No. of patients remaining in Stage 0 (%)
Normal	6 (35%)	11 (65%)
High	3 (75%)	1 (25%)

P = 0.38 (Chi-square test)

The development of histiocytic or large cell lymphoma (Richter's syndrome) was observed in six patients (two, Stage II; three, Stage III; and one, Stage IV at the time of diagnosis) in the present study. The diagnosis of Richter's syndrome was made in three cases at autopsy and in the remaining three cases Richter's syndrome was noted by lymph node or bone marrow biopsies during the course of the disease. Initial and subsequent LDH values in each of these six cases are shown in Figure 2. In only one patient was the initial LDH level within normal limits; the LDH activity then rose dramatically at the time of diagnosis of Richter's syndrome. In the second patient an initial LDH level was significantly elevated. A subsequent LDH level at the time of clinical remission was within normal limits and the LDH rose once again to a significantly elevated level when Richter's syndrome was identified. In a third patient the subsequent LDH level rose even further when Richter's syndrome was recognized. Of three patients in whom Richter's syndrome was noted at autopsy, two exhibited extremely elevated LDH activity (approximately 1,000 IU/L) and the remaining patient had a moderately elevated LDH value (350 IU/L) just prior to death.

Estimated survival distributions for the five clinical stages are shown in Figure 3. Twenty-nine patients (Stage 0, 1; Stage I, 2; Stage II, 6; Stage III, 11; Stage IV, 9) died of CLL. The estimated median survival time for patients with Stage I was 121 months, whereas the median survival time for patients with Stage III and IV were only 43 and 45 months, respectively. There was a highly significant relationship ($P < 0.00005$) between clinical stage and survival time.

Survival distributions by LDH activity are shown in Figure 4. Thirteen of 60 patients (Stage 0, 1; Stage I, 2; Stage II, 4; Stage III, 2; Stage IV, 4) with normal LDH levels died. In contrast, 16 of 41 patients (Stage II, 2; Stage III, 9; Stage IV, 5) with high LDH levels died; the median survival time of these patients was only 47 months. There was a highly significant relationship ($P = 0.0001$) between LDH activity and survival time.

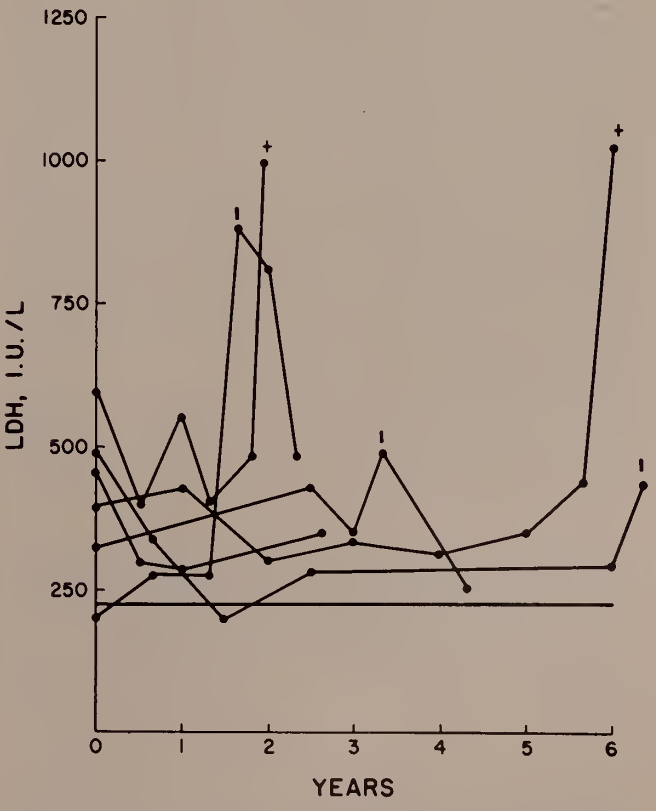


FIGURE 2. Sequential LDH levels in patients with Richter's syndrome. (•) represents the LDH value; (+) signifies that the diagnosis of Richter's syndrome was established at autopsy; and (|) reflects the time of diagnosis of Richter's syndrome during the course of the disease.

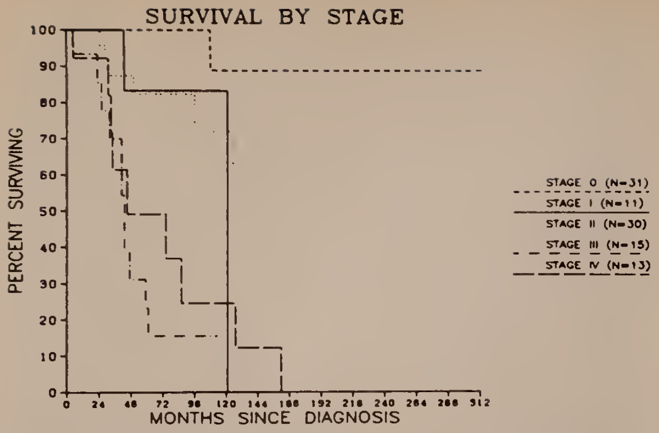


FIGURE 3. Survival distributions by clinical stage ($P < 0.00005$).

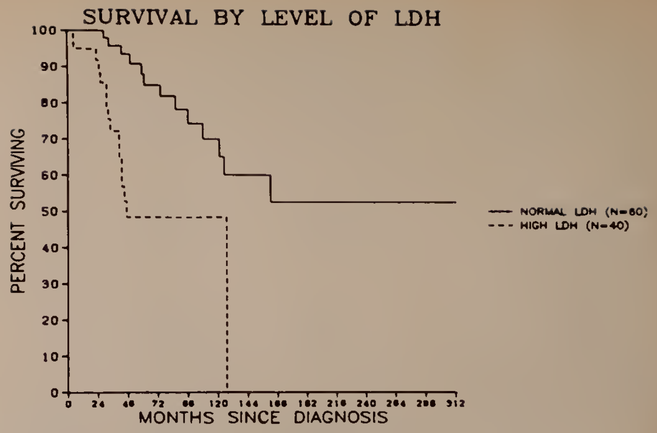


FIGURE 4. Survival distributions by LDH activity ($P = 0.0001$).

Likelihood ratio tests under the proportional hazards model of Cox were used to determine the relative importance of clinical stage and LDH activity with respect to survival time. The results of these regression models for survival by clinical stage and LDH activity are shown in Table V. When clinical stage alone was evaluated for survival, a highly significant relationship was observed ($P < 0.00005$). When LDH activity alone was evaluated for survival, a highly significant relationship ($P = 0.0003$) was also observed. When both clinical stage and LDH activity were evaluated for survival, a highly significant association between clinical stage and survival time was still observed ($P < 0.00005$) whereas the relationship between LDH and survival time was only suggestive ($P = 0.08$).

Discussion

In the present study, serum LDH was found to be elevated in 41 of 101 patients with CLL at various clinical stages. There was a highly significant relationship between LDH activity and clinical stage, alkaline phosphatase, ^{3}H-thymidine uptake of peripheral blood leukocytes, and the degree of lymphocytosis (Fig 1, Table I, II). The fact that LDH was found to be significantly correlated with clinical stage and with the degree of lymphocytosis indicates that increased LDH activity may be a reflection of increased tumor load. However, no relationship was observed between LDH activity and sex, Coomb's test status, serum immunoglobulin levels, bone marrow infiltration pattern, urinary monoclonal free light chain status, leukemic phenotype, or cytogenetics.

An increased LDH level was reported in 47 of 54 patients with lymphocytic leukemia, including ALL and CLL.[9] Although these investigators did not attempt to correlate LDH with other clinical or laboratory data they observed a strong relationship between LDH levels and total white blood cell count in one of two patients with CLL in whom many sequential determinations were performed during monthly follow up periods. No reports could be found in the literature with respect to LDH activity in CLL since the publication of the original study.[9]

In the present study, a highly significant relationship was observed between LDH activity and survival time. However, it should be emphasized that when both clinical stage and LDH activity were evaluated for survival, a highly significant relationship between clinical stage and survival time persisted, whereas the relationship between LDH activity and survival time became only suggestive.

One interesting observation in the present study is that three (75%) of four Stage 0 patients with high LDH activity progressed to advanced stages of disease whereas only six (35%) of 17 Stage 0 patients with normal LDH activity progressed. It should be pointed out, however, that the difference was not statistically significant, possibly because of the small number of patients. The potentially important prognostic value of LDH activity in Stage 0 patients at the time of diagnosis remains to be evaluated in a larger number of patients. It should also be emphasized that the LDH activity remained within normal limits in all six patients with nonprogressive Stage 0 disease whereas the subsequent LDH levels were elevated in four out of five Stage 0 patients at the time of disease progression. These observations suggest that subsequent LDH determinations should be utilized in Stage 0 patients for monitoring the disease progression, and that LDH determinations may be of clinical and prognostic importance for Stage 0 patients.

TABLE IV. Comparisons of Subsequent LDH Activity (IU/L) between Six Patients with Non-progressive Stage 0 Disease and Five Patients with Stage 0 Disease Who Progressed to Higher Stages of Disease

Non-progressive cases		Progressive cases	
Initial LDH	Subsequent LDH	Initial LDH	Subsequent LDH
150	150	225	300
125	171	185	271
125	130	162	261
169	200	200	202
145	177	219	430
200	186	—	—

TABLE V. Regression Models for Survival by Clinical Stage and LDH Activity

Model	Clinical variables	Log likelihood	*P* value
A	none	−109.80	—
B	LDH	−103.30	0.0003
C	Stage*	−91.24	0.00005
D	LDH/Stage*	−89.66	0.08/0.00005

* Stage 0 versus Stage I or II versus Stage III or IV.

Another interesting finding in this study is that all six patients subsequently developed Richter's syndrome, which represents a more malignant transformation in patients with CLL, and exhibited a moderately to markedly elevated LDH level at the time of antemortem diagnosis of Richter's syndrome, or just prior to the postmortem diagnosis of Richter's syndrome. This observation suggests that increased LDH activity may also be a reflection of some intrinsic property of the CLL cell, perhaps transforming it into a more malignant state.

Increased LDH activity is frequently associated with hemolytic anemia or ineffective erythropoiesis. It is possible that increased LDH activity in patients wtih Stage III and Stage IV disease may be related to autoimmune hemolytic anemia. However this is unlikely since there was no relationship between the Coomb's positivity and LDH levels (Table II). Only six cases of Stage III disease associated with Coomb's positive autoimmune hemolytic anemia were observed in the present study, and increased LDH activity was noted in only three (50%) of these six patients. Two cases of stage III or IV disease associated with Coomb's negative autoimmune hemolytic anemia were also observed, and increased LDH activity was found in one of these two patients. It should also be pointed out that in 21 remaining patients with Stage III or IV disease, 18 (86%) exhibited increased LDH activity although none were associated with Coomb's positive or negative autoimmune hemolytic anemia. These observations suggest that increased LDH activity seen in Stage III or IV patients with CLL is not related to autoimmune hemolytic anemia. An ineffective erythropoiesis may occur in patients with advanced-stage CLL and the possibility that ineffective erythropoiesis may be partly responsible for the elevated LDH activity could not be ruled out entirely.

Elevated LDH levels were originally reported in 34 (68%) of 50 patients with malignant lymphomas.[9] However, there was no correlation made between LDH activity and histologic type or clinical stage. More than 20 years later Ferraris and colleagues[14] reported that serum LDH levels were significantly increased in patients with non-Hodgkin's lymphoma including lymphocytic and histiocytic types and that survival time correlated inversely with LDH levels in these patients, irrespective of their histologic classification. These authors, however, failed to investigate the relationship between LDH level and clinical stage in these patients. In the past few years, several investigators have reported the prognostic significance of LDH activity in patients with non-Hodgkin's lymphoma. Elevated LDH activity was recently observed in 46 (41%) of 113 patients and normal LDH values were associated with a better response to therapy and a longer survival time, independent of histiologic type or clinical stage.[13] Associations between LDH activity and clinical stage and tumor burden in patients with Burkitt's lymphoma (including African, American, and Japanese patients) and ALL have also been reported in the literature. [10-12,16,19,20,22] It has also been established that there is a clear relationship between LDH activity and survival time in patients with chronic myelocytic leukemia,[31] carcinoma of the prostate,[3] and malignant melanoma.[33]

The finding that LDH is of clinical and prognostic significance leads to the conclusion that the evaluation of LDH levels in patients with CLL may be of some value for defining overall prognostic aspects of the disease, in addition to a well-established clinical staging system, and the analysis of other immunologic, phenotypic, and cytogenetic factors.

Acknowledgments. The authors wish to thank Dr Jun Minowada for the phenotypic study, Drs Houshang Moayeri and Joseph Sokal for the ^{3}H-thymidine uptake study, Drs Naoki Sadamori and Avery A. Sandberg for the cytogenetic study, and Ms Gail Walsh for manuscript preparation.

REFERENCES

1. Räi KR, Sawitsky A, Cronkite EP, Chanana AD, Levy RN, Pasternack BS: Clinical staging of chronic lymphocytic leukemia. *Blood* 1975; 46:219–234.
2. Baccarani M, Cavo M, Gobbi M, Lauria F, Tura S: Staging of chronic lymphocytic leukemia. *Blood* 1982; 59:1191–1196.
3. Moayeri H, Sokal JE: In vitro leukocyte thymidine uptake and prognosis in chronic lymphocytic leukemia. *Am J Med* 1979; 66:773–778.
4. Bartl R, Frisch B, Burkhardt R, Hoffmann-Fezer G, Demmler K, Sund M: Assessment of marrow trephine in relation to staging in chronic lymphocytic leukaemia. *Br J Haematol* 1982; 51:1–15.
5. Han T, Barcos M, Emrich L, et al: Bone marrow infiltration patterns and their prognostic significance in chronic lymphocytic leukemia: correlations with clinical, immunologic, phenotypic, and cytogenic data. *J Clin Oncol* 1984; 2:562–570.
6. Rozman C, Hernandez-Nieto L, Montserrat E, Brugues R: Prognostic significance of bone-marrow patterns in chronic lymphocytic leukaemia. *Br J Haematol* 1981; 47:529–537.
7. Han T, Sadamori N, Ozer H, et al: Cytogenetic studies in 77 patients with chronic lymphocytic leukemia: correlations with clinical, immunologic and phenotypic data. *J Clin Oncol* 1984; 2:1121–1132.
8. Hill BR, Levi C: Elevation of serum component in neoplastic disease. *Cancer Res* 1954; 14:513–515.
9. Bierman HR, Hill BR, Reinhardt L, Emory E: Correlation of serum lactic dehydrogenase activity with the clinical status of patients with cancer, lymphomas, and the leukemias. *Cancer Res* 1957; 17:660–667.
10. Abe K, Sato T: Burkitt's lymphoma in Japan: clinicopathologic features of twenty-two patients. *Tohoku J Exp Med* 1981; 134:289–293.
11. Arseneau JC, Canellos GP, Banks PM, Berard CW, Gralnick HR, DeVita VT: American Burkitt's lymphoma: a clinicopathologic study of 30 cases. 1. Clinical factors relating to prolonged survival. *Am J Med* 1975; 58:314–321.
12. Csako G, Magrath IT, Elin RJ: Serum total and isoenzyme lactate dehydrogenase activity in American Burkitt's lymphoma patients. *Am J Clin Pathol* 1982; 78:712–717.
13. Endrizzi L, Fiorentino MV, Salvagno L, Segati R, Pappagallo GL, Fosser V: Serum lactate dehydrogenase (LDH) as a prognostic index for non-Hodgkin's lymphoma. *Eur J Cancer Clin Oncol* 1982; 18:945–949.
14. Ferraris AM, Giuntini P, Gaetani GF: Serum lactic dehydrogenase as a prognostic tool for non-Hodgkin lymphomas. *Blood* 1979; 54:928–932.
15. Fisher RI, Hubbard SM, DeVita VT, et al: Factors predicting long-term survival in diffuse mixed, histiocytic, or undifferentiated lymphoma. *Blood* 1981; 58:45–51.
16. Kornberg A, Polliack A: Serum lactic dehydrogenase (LDH) levels in acute leukemia: marked elevations in lymphoblastic leukemia. *Blood* 1980; 56:351–355.
17. Koziner B, Little C, Kurland E, et al: Analysis of prognostic factors in diffuse histiocytic lymphoma (DHL), abstracted. *Proc Am Assoc Cancer Res* 1982; 23:450.
18. Koziner B, Little C, Passe S, et al: Treatment of advanced diffuse histiocytic lymphoma: an analysis of prognostic variables. *Cancer* 1982; 49:1571–1579.
19. Magrath I, Lee YJ, Anderson T, et al: Prognostic factors in Burkitt's lymphoma: importance of total tumor burden. *Cancer* 1980; 45:1507–1515.
20. Ridgway D, Smiley S, Neerhout RC: The prognostic value of presenting serum LDH in non-Hodgkin lymphoma. *J Pediatr* 1981; 99:611–613.
21. Schneider RJ, Seibert K, Passe S, et al: Prognostic significance of serum lactate dehydrogenase in malignant lymphoma. *Cancer* 1980; 46:139–143.
22. Shaw B: Studies on serum lactate dehydrogenase in Burkitt's lymphoma patients during chemotherapy. *East Afr Med J* 1970; 47:327–332.
23. Han T, Ozer H, Bloom M, Sagawa K, Minowada J: The presence of monoclonal cytoplasmic immunoglobulins in leukemic B cells from patients with chronic lymphocytic leukemia. *Blood* 1982; 59:435–438.
24. Sandberg AA: *The chromosomes in human cancer and leukemia.* Elsevier North Holland, New York, 1980, pp 31–425.
25. Wroblewski F, LaDue JS: Lactic dehydrogenase activity in blood. *Proc Soc Exp Biol Med* 1955; 90:210–213.
26. Kessler G, Rush RL, Leon L, Delea A, Cupiola R: Automated 340 measurement of SGOT, SGPT, and LDH, in *Advances in Automatic Analysis.* Technicon International Congress, 1970, vol 1, Miami, Thurmon Assoc, 1971, pp 67–69.
27. Mann HB, Whitney DR: On a test of whether one of two random variables is stochastically larger than the other. *Ann Math Stat* 1947; 18:50–60.
28. Kaplan EL, Meier P: Nonparametric estimation from incomplete observa-

tions. *J Am Stat Assoc* 1958; 53:457–481.

29. Peto R, Peto J: Asymptotically efficient rank invariant test procedures. *J Royal Stat Soc* 1972; 135A:185–206.

30. Cox DR: Regression models and life tables. *J Royal Stat Soc* 1972; 34B:187–220.

31. Gomez GA, Sokal JE, Walsh D: Prognostic features at diagnosis of chronic myelocytic leukemia. *Cancer* 1981; 47:2470–2477.

32. Berry WR, Laszlo J, Cox E, Walker A, Paulson D: Prognostic factors in metastatic and hormonally unresponsive carcinoma of the prostate. *Cancer* 1979; 44:763–775.

33. Finck SJ, Giuliano AE, Morton DL: LDH and melanoma. *Cancer* 1983; 51:840–843.

See also pp 679–680

The impact of technology on laboratory ordering strategies

CRAIG A. LEHMANN, MS, CC (NRCC), ALAN M. LEIKEN, PHD, DAVID FLOERING, MD

ABSTRACT. Changes in the methods of reimbursing hospitals have converted clinical laboratories from revenue producers to major cost centers. This has focused attention on assessing laboratory ordering strategies and on encouraging cost effective utilization of the laboratory. This study evaluates the costs associated with ordering strategies of one group of residents as it rotates through two institutions whose laboratories differ both in philosophy and automated equipment. The data demonstrate that residents utilize the laboratory far more often when there are fewer restrictions as to what tests may be ordered, when they may be performed, and how frequently. The results suggest that providing the option of component testing may create the potential for incurring significant, avoidable costs.

(NY State J Med 1985; 85:690–693)

The appropriateness of using biochemical panels and profiles as an alternative to component testing in health care delivery has been debated by both researchers and clinicians over the past two decades. Although still unresolved, the introduction of federal regulations has created a new and greater concern. The Tax Equity and Fiscal Responsibility Act (TEFRA) passed in 1982, now referred to as Diagnostic Related Groups (DRGs), attempts to reimburse hospitals by case and to set a limit for each case. This changes the hospital's present reimbursement system to one of prospective payment. This prospective payment system later included hospital-based physicians (eg, pathologists) by changing their reimbursement from the portion allocated for professional service rendered to that allocated for total hospital reimbursement.

As this prospective reimbursement system is implemented throughout the United States, hospital administrators and other fiscally concerned officials have focused on the laboratory in an effort to reduce costs. Special concern has been placed on the appropriateness of future investments in automated equipment. While the technologic advances made in laboratory automation have decreased the cost per test, overall laboratory costs have increased.[1–4] The initial cost savings in laboratory automation came about by labor reduction, but was soon offset by physicians increasing the number and variety of tests performed per patient. In fact, it has been demonstrated that as more highly automated tests became available, the demand for such tests became greater. These reports have demonstrated that 50 to 79% of all biochemical testing may be attributed to 20 highly automated tests.[5,6] Because of this, physicians are being persuaded to reduce their use of these tests in the hope that test reduction would lead to cost containment. Although this concept may seem logical, it is neither easily implemented nor necessarily true.

While researchers have debated the relative merits and shortcomings of both types of ordering strategies (component or panels),[7–10] two of the present authors—Lehmann and Leiken—have evaluated laboratory costs associated with physicians' use of component and panel chemistries. These two studies have focused on laboratory utilization at both a health care maintenance organization and an out-patient department of a community hospital.[11,12] In both facilities the physicians were free to order component and panel chemistries. The outcome of the studies at both facilities demonstrated that the utilization of panel chemistries resulted in lower costs and fewer visits to the health care facility.

In today's cost containment climate, many hospitals have attempted to reduce laboratory costs by utilizing discrete analyzers rather than continuous flow profiling analyzers, and by urging physicians to decrease laboratory utilization. However, if physicians continue to order profiles or large quantities of tests at facilities that employ discrete analyzers, costs per test and time for completion of these tests will increase, thereby increasing the financial strain that hospitals have been trying to alleviate.

In order to determine if physicians alter their use of laboratory services to take advantage of the cost saving

---

From the School of Allied Health Professions, Departments of Medical Technology (Prof Lehmann) and Allied Health Resources (Dr Leiken) and the School of Medicine Department of Clinical Pathology (Dr Floering), State University at Stony Brook Health Sciences Center, NY.

Address correspondence to Professor Lehmann, Associate Professor, School of Allied Health Professions, Department of Medical Technology, Health Sciences Center, SUNY at Stony Brook, Stony Brook, NY 11794.

TABLE I. Laboratory Test Groupings

	Facility A	Facility B
Large panel	Albumin, Alkaline phosphatase, Phosphorus, Direct bilirubin, Total bilirubin, Calcium, Total protein, Cholesterol, Triglyceride, Lactate dehydrogenase, Uric acid, Magnesium, Gamma glutamyl transferase, Aspartate aminotransferase, Alanine aminotransferase, Creatine kinase	Uric acid, Sodium, Calcium, Potassium, Chloride, Total protein, Bicarbonate, Albumin, Cholesterol, Alkaline phosphatase, Triglyceride, Glucose, Total bilirubin, Aspartate aminotransferase, Alanine aminotransferase, Lactate dehydrogenase, Creatinine, Urea nitrogen, Creatine kinase, Phosphorus
Small panel	Glucose, Urea nitrogen, Sodium, Potassium, Chloride, Bicarbonate, Creatinine	Glucose, Urea nitrogen, Sodium, Potassium, Chloride, Bicarbonate
Components	Any single or combination of tests from large or small panels	—

potential of discrete analyzers, the costs associated with ordering strategies were examined as a group of residents rotated through two institutions whose laboratories differ both in philosophy and automation requirement.

PATIENTS AND METHODS

The present study was conducted at two teaching hospitals whose automated chemistry equipment and philosophy differed. Hospital "A" is a 250-bed tertiary care facility in suburban New York whose laboratory utilizes a Beckman Astra and a Boehringer Mannheim Hitachi for its biochemical assays. Physicians at this facility may order a 7 (Astra) or 16 (Hitachi) biochemical panel and components without restrictions as to time, day, or volume. Hospital "B" is a 340-bed primary care facility located within 20 miles of facility A whose laboratory utilizes a Technicon SMAC 20 and SMA 6/60 for its biochemical assays. At this institution the medical staff is restricted as to what tests are available and when they may be ordered. The 6 or 20 biochemical panel is available between 8 AM and 5 PM, Monday through Friday. At times other than those and during weekends physicians may only order the 6 chemistry panel. The laboratory offered no unbundling of their 6 or 20 chemistry panel, nor were there any restrictions on the number of panels one could order.

As seen in Table I the 6 and 20 biochemical panel at facility B and the 7 and 16 biochemical panel at facility A are essentially the same. No other tests than those used in the panels were evaluated since the purpose of the study was to evaluate the impact of panels versus component chemistries in health care delivery.

Laboratory requests were monitored for six months, as the same group of internal medicine residents treated myocardial infarction, congestive heart failure, and airway passage blockage at facilities A and B. The number of panels and components of the panels were recorded to evaluate the effect of ordering strategies on laboratory utilization for each of the primary codes. These three primary codes were chosen because they gave the highest number of cases at both facilities.

In addition to the average number of panels and components of the panels associated with each primary code, the average per case cost for each laboratory was calculated.

RESULTS

A comparison of the residents' use of laboratory services was made for three primary diagnostic codes at both facilities. For each diagnostic code the average length of stay, along with the average number of large panels (16 and 20 tests), small panels (6–7 tests), components, and laboratory costs were calculated.

Patient demographics for both facilities were as follows:

- For myocardial infarction the mean age at facility A was 61 years (20% women, 80% men); the mean age at facility B was 60 years (100% men).
- For congestive heart failure the mean age at facility A was 67 years (45% women, 55% men); the mean age at facility B was 67 years (100% men).
- For airway passage blockage the mean age at facility A was 67 years (32% women, 68% men); the mean age at facility B was 68 years (100% men).

At both facilities, the patients' laboratory charges were not billed separately but were part of prospective payment.

The study first evaluated the residents' laboratory utilization in their treatment of myocardial infarction. As demonstrated in Table II, the residents' utilization of laboratory services differed considerably depending on whether patients were treated at facility A or B. While the utilization of large panels was somewhat consistent at both facilities, there was almost a three-fold increase in small panels at facility A as compared to facility B. In addition to the small panel increase noted at facility A, the residents utilized an average of 102 additional components.

Resident laboratory utilization in the care of congestive heart failure is seen in Table III. Unlike with cases of myocardial infarction, the residents decreased their utilization of the large panel at facility A and maintained approximately the same use at facility B. For small panels, residents at facility A demonstrated a slight decrease while those at facility B had a slight increase. The component testing at facility A for congestive heart failure also demonstrated a small decrease (102 versus 86.07) when compared to component testing for myocardial infarction. For the third primary code—chronic airway obstruction—as seen in Table IV, laboratory utilization by the residents was consistent with that of congestive heart failure at both facilities. This is most likely attributed to the chronic implications of these two codes, as compared to the acuteness of myocardial infarction. However, in all three codes, residents at facility A utilized the small panel about twice as much as they did at facility B. As for the two chronic nature codes—heart failure and airway obstruction—residents at facility B utilized the large panel twice as often as they did at facility A.

DISCUSSION

It is difficult to present convincing arguments for either ordering strategy at this time: the greater utilization of small panels at facility A or large panels at facility B. However, a more argumentative point would be the utilization of components at facility A. As demonstrated in the Figure, the utilization of components is practiced for all three primary codes. The availability of this service at facility A did not appear to reduce the utilization of panels

TABLE II. Laboratory Utilization: Myocardial Infarction

	Facility A	Facility B
Number of patients	N = 65	N = 65
Mean number of small panels	10.15 ± 8.89*	3.57 ± 4.74*
Mean number of large panels	7.81 ± 5.33	7.01 ± 6.58
Mean number of component chemistries	102.5 ± 79.81	—

* Statistically significant at $P < 0.05$.

TABLE III. Laboratory Utilization: Congestive Heart Failure

	Facility A	Facility B
Number of patients	N = 43	N = 96
Mean number of small panels	8.11 ± 7.06*	4.51 ± 5.02*
Mean number of large panels	3.46 ± 3.83*	6.72 ± 5.43*
Mean number of component chemistries	86.07 ± 75.13	—

* Statistically significant at $P < 0.05$.

TABLE IV. Laboratory Utilization: Chronic Airway Obstruction

	Facility A	Facility B
Number of patients	N = 44	N = 40
Mean number of small panels	7.6 ± 9.53	4.6 ± 7.18
Mean number of large panels	3.61 ± 5.09*	6.5 ± 5.37*
Mean number of component chemistries	79.68 ± 96.61	—

* Statistically significant at $P < 0.05$.

by residents. In fact, there was a greater use of small panels. In the case of myocardial infarction patients at facility A, the residents not only tripled their use of small panels but also utilized an average of 102 components. In 37% of the cases physicians used 101 to 386 components. When the option of component testing was not available to the residents, as seen with facility B, there was no greater increase in the use of laboratory panels. The differences between the average number of panels at both facilities were tested for statistical significance using student t-tests; those that occurred with a probability of less than 5% were statistically significant.

While the laboratory data was examined by type of case (ie, myocardial infarction, heart failure, chronic airway obstruction), differences in the severity of the cases within each primary code could conceivably affect the results. To determine whether the severity of illness of patients treated at these two facilities differed, each case was examined for the average length of stay and average number of secondary codes. One might conjecture that the greater utilization of the laboratory noted at facility A could be due to the increased severity of cases treated at this facility. This study shows, however, that this is not the case. In fact, patients at facility B had more complications (3.38 versus 2.26) and a greater length of stay (14.08 versus 11.89) than those at facility A.

In order to evaluate the costs associated with the increased utilization of laboratory services, an average cost analysis for each primary code at each facility was performed. Table V demonstrates that laboratory utilization by residents at facility A is far more costly for all three primary codes than at facility B. In fact, even for those cases such as heart failure and airway obstruction at facility B, where there was greater utilization of larger panels, overall laboratory costs were less.

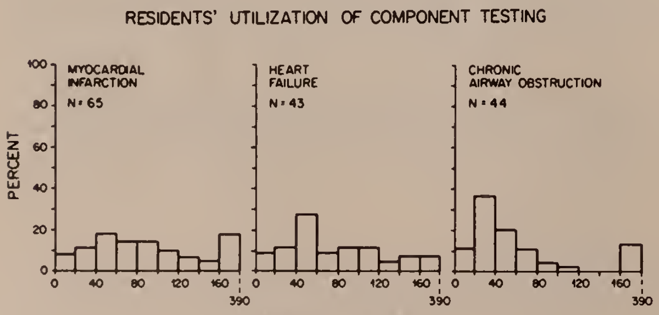


FIGURE. Utilization of component testing for myocardial infarction, heart failure, and chronic airway obstruction.

Previous studies have demonstrated that equipment, sample volume, and number of tests per sample influence overall laboratory costs.[13,14] This is well demonstrated when the costs of panels and components at facility A are compared with those at facility B. The cost for a large panel was $3.68 and a small panel was $2.07 at facility A, while at facility B the cost was $2.35 for a large panel and $0.76 for the small panel. This difference in cost, along with greater utilization of the small panel, placed overall costs for panels at facility A much higher. While costs for the panels are much greater at facility A, it seems that the more costly fraction of the ordering strategy is with the use of components. As stated earlier, when the same group of residents are offered the option of ordering component chemistries, as compared to ordering panels only as seen with facility B, there appears to be no reduction in their use of panels. The high utilization of components by residents at facility A for all three primary codes is extremely costly as demonstrated by the following per case components costs: myocardial infarction, $26.65; airway blockage, $20.72; and heart failure, $22.38.

In addition to costs associated with the actual laboratory test, other factors that result from different ordering strategies must be considered, such as the productive time of technical, clerical, and aid staff. As demonstrated by the Manual for Laboratory Workload Recording Methods,[15] chemical analyzers that perform either one or a number of tests, such as the ASTRA and Hitachi used at facility A, receive a unit number for initial sample set up plus a unit number per test. In contrast to facility A, simultaneous chemical analyzers, such as the SMA 6/60 and SMAC used at facility B together only receive one unit number per specimen. A comparison of workload units for each institution's equipment is as follows: the ASTRA used at facility A requires 2.1 units for each specimen set up, along with an 0.1 additional unit for each test. The Hitachi requires 2.5 units for specimen set up and an additional 0.2 unit for each test. However, the SMA 6/60 used at facility B has an overall value of 4.0, and the SMAC has a value of 2.5 units. Thus at facility A it requires 5.9 workload units for a large panel, as compared to 2.5 workload units at facility B. Small panels at facility A require a total of 2.9 units, as compared to 4.0 at facility B. In addition to panels, every time components were utilized at facility A, a workload unit for specimen prepara-

TABLE V. Laboratory Costs Per Case*

	Facility A	Facility B
Myocardial infarction	$76.40	$19.18
Congestive heart failure	$51.90	$19.21
Chronic airway obstruction	$49.73	$18.78

* Costs include reagents, supplies, service contracts, and capital equipment.

tion of 2.1 or 2.5 was required for each component, regardless of the number of components ordered. Since the bulk of the workload unit is in specimen set-up, especially in the small panel, the advantages of its components are questionable, especially since the small panel was utilized much more often at facility A.

The data demonstrate that residents utilize laboratory services far more often when at facility A as compared to when they are at facility B. This pattern generated greater workload units and higher costs. If the choice of automation for facility A was to offer their physicians an alternative to panels and reduce the number of tests per case, this goal has not been accomplished. In fact, just the opposite has taken place as each primary code demonstrated increased testing. While each primary code had greater utilization of the small panel at facility A as compared to facility B, the predominate factor was in the utilization by the residents of component testing. Since the same residents managed all three primary codes at facility B without component testing and demonstrated no greater utilization of the panel, the appropriateness of offering components is questionable. This is especially true for highly automated tests, since studies have demonstrated that component testing is only cost effective if kept at a minimum.

References

1. Morrison JI, Tydeman J, Cassidy PA, Hardwick DF, Davies CT: Costs of clinical chemistry laboratory tests. *Lab Med* 1983; 14:567–570.
2. Tydeman J, Morrison JI, Cassidy PA, Hardwick DF: Analyzing the factors contributing to rising laboratory costs. *Arch Pathol Lab Med* 1983; 107:7–12.
3. Hardwick DF, Morrison JI, Tydeman J, Cassidy PA, Chase WH: Laboratory costs and utilization: a framework for analysis and policy design. *J Med Educ* 1981; 56:307–315.
4. Tydeman J, Morrison JI, Kasap D, Poulin M: The cost of laboratory technology: a framework for cost management. *Med Instrum* 1983; 17:79–83.
5. Morrison JI, Tydeman J, Cassidy PA, Hardwick DF: Automation and distribution of testing in the clinical laboratory. *J Med Technol* 1984; 1:132–137.
6. Stilwell JA: Costs of a clinical chemistry laboratory. *J Clin Pathol* 1981; 34:589–594.
7. Werner M, Altshuler CH: Utility of multiphasic biochemical screening and systematic laboratory investigations. *Clin Chem* 1979; 25:509–511.
8. Caceres C: By limiting the number of tests ordered and using automated panels, physicians can help the lab reduce unit costs from $250.00 to $7.00. *Lab World* 1980; 31:45–47.
9. Friedman G, Marshall G, Ahuja J, Siegelaub AB: Biochemical screening tests. *Arch Intern Med* 1972; 129:91–97.
10. Durbridge TC, Edwards F, Edwards RG, Atkinson M: Evaluation of benefits of screening tests done immediately on admission to hospital. *Clin Chem* 1976; 22:968–971.
11. Lehmann CA, Leiken AM: Influence on selective vs. panel chemistry tests on cost and diagnostic time. *Am J Med Technol* 1982; 48:833–836.
12. Lehmann CA, Leiken AM: Costs of ordering strategies. *J Lab Med* 1984; 15:759–760.
13. Weinstein MC, Pearlman LA: The implications of cost effectiveness analysis of medical technology. Office of Technology Assessment, Washington, DC, 1981.
14. Berkly Scientific Laboratories, *A Study of Automated Clinical Laboratory Systems.* DHEW Publ. no. (HSM)72-3004, US Dept of Health, Education and Welfare, Rockville, MD, 1971.
15. *Manual for Laboratory Workload Recording Method.* College of American Pathologists, Skokie, IL, 1985.

See also pp 680–681.

COMMENTARY

The difficulties of doing research on cost effectiveness in health care

The research paper by Professor Lehmann and Drs Leiken and Floering attempts to evaluate the cost effectiveness of providing individual chemical tests in addition to the more common panels of tests. The study compares test ordering practices of the same group of residents treating patients with the same diagnoses at two hospitals, one at which panel and individual tests were available and the other at which only panel tests were available. The commentary by Dr Conn (p 680) points out some of the shortcomings of this study, such as the differences in the institutions regarding patient populations, in biochemical panels, and in availability of test results. These differences illustrate the difficulty of doing research on cost effectiveness in situations where the randomized trial—the gold standard—is not readily applicable.

In spite of the differences between hospitals it is tempting to assume that the residents ordered only those tests that they believed necessary and appropriate at each institution. Given that assumption, the question is whether the availability of component chemical tests reduced the use of panel tests. On this issue the data indicate that it did not. For myocardial infarction, there were almost twice as many panel tests (both small and large) at the institution that provided component tests. For the other two diagnoses, there was little difference in the average number of panel tests (both small and large) between the two hospitals. Although the facility that offered component tests offered three additional procedures, it seems unlikely that this would totally negate a reduction in the frequency of panel tests as when compared to the other institution.

Accordingly, the authors of the study question the wisdom of providing component tests. Noting the frequent use of panel testing at both institutions, the commentator makes a plea that physicians select tests more rationally and avoid relying on arbitrary panels.

The article and commentary both highlight an important problem in medical care, one that transcends economics and may only be addressed at the level of the individual physician.

JOSEPH G. FELDMAN, DrPH
Consultant in Biostatistics
New York State Journal of Medicine

A 40-year-old man with chills, fever, weight loss, and a productive cough

CASE PRESENTATION

Susan Cantor, MD (Medical Resident): A 40-year-old veteran of the US Armed Forces was admitted to the Northport Veterans Administration Medical Center complaining of a high fever during the week prior to admission and severe frontal headaches for three days. Because of low grade fever for seven weeks before admission he had been treated with doxycycline for two weeks, but without improvement. The patient also complained that for several weeks he had had chills, night sweats, malaise, generalized weakness, and a cough productive of small amounts of clear sputum. Over one month's time he had lost 17 lbs.

The patient had served in the military in Southeast Asia from 1968 to 1972. He had experienced five febrile illnesses that he referred to as "fevers of unknown origin" in the period from 1965 to 1972, the first at Fort Dix in New Jersey, and the remainder in Southeast Asia. One such episode in Vietnam lasted three weeks with very high fever, generalized joint swelling, and arthralgias; treatment with penicillin was associated with an anaphylactic reaction. At no time was an infectious cause identified.

In 1981 the patient had a two month period of low-grade fever, chills, weight loss, and anorexia; he was treated with tetracycline with some improvement. In 1983, he developed three skin conditions that have persisted to the present admission: rosacea on the nose and face; psoriasis on the knees, elbows, and hands; and molluscum contagiosum on the lower abdomen.

His medical history was otherwise notable only for childhood measles and mumps. He denied exposure to toxic chemicals except for Agent Orange and marine solvents. His tine test for tuberculin sensitivity was negative in 1979. The patient smoked cigars and drank alcohol only on social occasions; he denied intravenous drug abuse, homosexuality, and the receiving of blood transfusions. He admitted being allergic to penicillin and erythromycin. At the time of admission, the patient was not taking any medications.

Physical examination on admission revealed a well developed thin white man in no distress. His blood pressure was 120/80 mm Hg; pulse, 86/min with a regular rhythm; respiratory rate, 22/min; and temperature, 102° F orally. Examination of the skin revealed several facial telangiectases, psoriatic plaques on the knees and elbows, and a few superficial ulcers consistent with molluscum contagiosum on the lower abdomen. Slight reddening was noted in the left otic canal. The fundi showed no abnormalities; the oropharynx was clear. The neck was supple and without masses. The chest was clear to auscultation and percussion and the cardiac examination was within normal limits. Abdominal examination revealed on percussion a liver span of 8 cm; the liver edge was smooth and the spleen was not felt. There were no abdominal masses. Rectal examination revealed a small, reddened area without discharge around the anus; the stool was negative on chemical test for hemoglobin. There was no clubbing, cyanosis, or edema of the extremities. Shotty lymphadenopathy was noted in the left anterior cervical, left supraclavicular, right axillary, and both inguinal areas. A 1 cm nontender firm movable node was felt in the left supraclavicular area, and a slightly smaller but otherwise similar node was present in the right axilla. The neurologic examination was entirely normal.

Laboratory evaluation revealed a normal urine analysis. Hemoglobin was 12.3 g/dL; hematocrit, 35.5%; white blood cell count, 4,700/μL, with 72% polymorphonuclear cells, 4% band forms, 19% lymphocytes, and 4% monocytes. The mean corpuscular volume (MCV) was 87 cu μ (normal, 80–94 cu μ); platelet count, 283,000/μL; prothrombin time (PT) and partial thromboplastin time (PTT) were within the normal range. The blood chemistry profile was within normal limits. The erythrocyte sedimentation rate (ESR) was 62 mm/h. Arterial blood gas analysis, drawn with the patient breathing room air, revealed a pH of 7.49, a $P\text{CO}_2$ of 34 mm Hg, and a $P\text{O}_2$ of 80 mm Hg.

An electrocardiogram demonstrated normal sinus rhythm, with a heart rate of 80/min, normal axes and intervals, and poor R-wave progression in leads V1 and V2. The chest radiograph showed a diffuse reticulonodular pattern with nodules up to 5 mm in diameter. There was relative sparing of the upper lung fields and the heart size was normal. Bilateral hilar adenopathy was noted (Fig 1). An abdominal flat plate was normal.

The Venereal Disease Research Laboratory test (VDRL), monospot test, antinuclear antibody test

---

Based on the clinicopathologic conference (CPC) held at the Northport Veterans Administration Medical Center, Northport, NY, on April 10, 1985.

Case Presentation: Susan Cantor, MD, Medical Resident; *Radiology Presentation*: Michael Clair, MD, Chief, Radiology Service; *Clinical Discussions*: Stuart Nelson, MD, Assistant Professor of Clinical Medicine, State University of New York at Stony Brook; Benjamin Luft, MD, Assistant Professor of Medicine, State University of New York at Stony Brook; *Pathology Presentation*: Sergey Lyubsky, MD, Pathologist; *Conference Moderator*: Edward L. Gogel, MD, Assistant Professor of Clinical Medicine, State University of New York at Stony Brook.

Editor: JOHN T. FLYNN, MD

Address correspondence to Dr Flynn, Consulting Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeview Road, Lake Success, NY 11402.

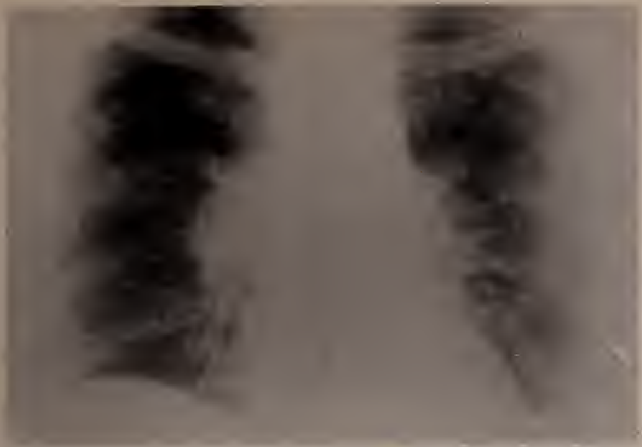


FIGURE 1. Posteroanterior chest radiograph at time of initial admission, demonstrating hilar and paratracheal adenopathy, and multiple 3 to 4 mm pulmonary nodules.

(ANA), serum cold agglutinins, and hepatitis profile were all negative. The serum levels of vitamin B_{12}, folate, thyroid hormones, and complement were all normal; serum protein electrophoresis was also normal. The serum rheumatoid factor was positive. Four sets of blood cultures—aerobic, anaerobic, and fungal—were negative. Urine culture was negative. No sputum was produced for evaluation. Viral and fungal titers were obtained.

Lumbar puncture performed on the first hospital day produced clear colorless fluid, with an opening pressure of 18.5 cm of water (normal range, 7 to 8 cm); no cells were seen. The spinal fluid protein was 25 mg/dL; the glucose level was 52 mg/dL, with a simultaneous serum glucose level of 92 mg/dL.

Purified Protein Derivative (PPD) skin test for tuberculin sensitivity was placed with controls; all were negative. Gallium scan of the lungs showed uptake in the right hilar area. Computerized tomographic (CT) scan of the chest revealed a 2 cm paratracheal node, small nodular densities throughout the lung fields, and two nodules in the liver. Bone marrow biopsy revealed a normal active marrow; cultures were obtained.

Pulmonary function tests revealed all volumes to be decreased—the vital capacity was 78% of predicted value; inspiratory capacity (IC), 88% of predicted; expiratory reserve volume (ERV), 59%; total lung capacity (TLC), 76%; forced vital capacity (FVC), 79%; forced expiratory volume in one second (FEV 1), 74%; and diffusion capacity for carbon monoxide (DLCO), 87%. There was no response to the administration of bronchodilators. This profile was interpreted as being consistent with restrictive lung disease.

CLINICAL DISCUSSION

Stuart Nelson, MD (Assistant Professor of Clinical Medicine, State University of New York at Stony Brook): In analyzing this case, I have chosen to concentrate on the main reason for the patient's admission, and to make the assumption that one diagnosis will account for the findings in this case.

Of the features of this man's illness, those most helpful to me in analyzing the case were the abnormal chest radiograph, the lymphadenopathy, and the presence of fever. Because the chest radiograph was of such importance, I would like Dr Clair to review the films.

RADIOLOGY PRESENTATION

Michael Clair, MD (Chief, Radiology Service): As Dr Nelson stated, it would be helpful if we could tie all the findings together, particularly with regard to those we can see on the chest radiographs.

On admission the initial chest film presented a diffuse reticulonodular pattern with relative sparing of the pulmonary apices (Fig 1). We must keep in mind, however, when evaluating the chest radiograph, that there is relatively less lung parenchyma in the apices than at the bases. Therefore, a slight difference in visibility of lesions between the apices and the bases may be a reflection more of the difference in parenchymal volume than of pattern density; it may not necessarily indicate apical sparing. I believe that this is the situation in this case, and that the reticulonodular pattern is uniformly diffuse. The borders of the pulmonary nodules in general are relatively shaggy. Also, there is no evidence of any pleural changes, or of pleural fluid. There is evidence of hilar prominence, especially on the right side; and there is a suggestion of paratracheal widening.

Beginning at the apical regions of the chest and visualizing a series of cross sections caudally to the region just above the carina, the CT scan demonstrates the normal appearance of the vascular structures in this region. Immediately behind the superior vena cava there is an enlarged 22-mm pretracheal node. The maximum diameter of a normal lymph node in this region ranges from 17 to 20 mm; if larger, such a node is presumed to be either neoplastic or enlarged due to active inflammation. The pathologic change within this enlarged node in relation to the underlying disease process is uncertain, but in trying to identify such a relationship one can presume that whatever agent is causing the changes in the lung parenchyma is also responsible for the paratracheal adenopathy. Additionally, shotty nodes were described in the axilla on physical examination although not well demonstrated on the CT scan. Their small size renders their significance uncertain.

On several of the CT images both hilar regions appear prominent, particularly so on the right side. There is a fairly large nonenhanced area with the administration of contrast medium, most probably representing lymphadenopathy. CT is not the imaging procedure of choice for adenopathy in general; indeed, it can be confusing in instances of borderline hilar adenopathy. Fifty-five-degree oblique tomograms may usually suffice.

In evaluating pulmonary parenchyma from the apices to the bases, at different window and level settings of the CT a diffuse nodular or reticulonodular pattern is seen throughout both lung fields. Most of these nodular masses are not well circumscribed and none show cavitation; the largest is about 5 to 7 mm in diameter. Most of these nodules were somewhat peripheral in location.

Dr Nelson: Several years ago Dr Jerome Brody classified the causes of reticulonodular patterns on chest radiographs by cause[1]: malignancy; cardiovascular causes; infectious diseases; inhalational diseases including those related to systemic exposure to drugs; immunologic diseases; and idiopathic causes.

May any of these categories be eliminated at this point? Without a history of exposure to agents known to cause interstitial pulmonary disease, I think we can eliminate that category. Cardiovascular causes should not lead to a syndrome with both fever and lymphadenopathy.

Of the idiopathic causes, amyloidosis and the interstitial pneumonitides may be eliminated because of their rare association with fever. In that category, there remains for serious consideration only sarcoidosis and eosinophilic granulomata. I am always wary of making a diagnosis of sarcoidosis in the presence of recurrent fever. Fever is not usually a prominent symptom in sarcoidosis, except when granulomatous inflammation occurs in areas outside of the lungs or lymph nodes. Heerfordt's syndrome, or uveoparotid fever, is a manifestation of sarcoidosis in which fever is not usual; another is Lofgren's syndrome, which presents with erythema nodosum and arthralgias. We have little in the protocol to support either of these syndromes.

Could eosinophilic granuloma—histiocytosis X—involve only the lungs and lymph nodes? The present of eosinophilic granuloma without involvement of the bones is a rare occurrence; similarly, involvement of the mediastinal lymph nodes in this disorder appears to be rare.

Having considered these potential causes, they should now be disregarded as significant possibilities, and thus leave behind us the idiopathic causes of this chest radiograph pattern.

The next category of disease to be considered is that of immunologic diseases. Although the patient had a history of arthritis, on admission at this time he did not have any joint complaints nor objective joint findings. His urine analysis was reported as normal. There seemed to be little on his physical examination that was suggestive of vasculitis. Thus there was little to suggest the possibility of scleroderma, Wegener's granulomatosis, polyarteritis nodosa, or Sjögren's syndrome, all of which may present with similar chest radiographs.

The positive rheumatoid factor is of concern since it is associated with the possibility of rheumatoid lung disease. While this degree of rheumatoid lung involvement could occur without any joint manifestations, it is relatively unlikely. Splenomegaly and lymphopenia may occur, but I would not expect to find this much lymphadenopathy. Therefore I would tend to discount the possibility of rheumatoid disease.

Another major cause of the signs and symptoms that could possibly explain this patient's illness is that of neoplasia. Of the solid neoplasms, there are only a few noted to cause fever, or to spread in the lung in this manner. However, lymphomas are noted for presenting with fever and lymphadenopathy; it is not unusual for lymphomas to have pulmonary involvement presenting as diffuse reticulonodular infiltrate on chest radiograph. Almost any of the lymphomas could present in this manner, but I particularly wish to call attention to mycosis fungoides since the skin lesions caused by mycosis fungoides frequently appear like those of psoriasis. Diffuse pulmonary involvement is usually associated more with T-cell lymphomas, such as mycosis fungoides, than with B-cell varieties. However, mycosis fungoides is a disease that usually afflicts elderly men, unlike this patient of forty years.

Another possibility is that this patient may have Hodgkin's disease. However, this would have to be a presentation of Hodgkin's disease in the lung. The most common forms are mediastinal node involvement or a combination of both pleural and parenchymal disease.

The last group of illnesses that I wish to consider is that of infectious diseases. In view of this patient's clinical course, which is more subacute or chronic, together with the chest radiograph pattern, a mycobacterial or fungal infection should be suspected as a cause of this patient's illness. There is nothing in the protocol that would lead me to believe that the patient could not have disseminated tuberculosis, although the pulmonary nodules on the chest film appear somewhat larger than one might expect in a case of miliary tuberculosis.

There are a variety of fungal infections, particularly coccidioidomycosis, paracoccidioidomycosis, and histoplasmosis, that could present similar appearances on the chest radiograph with coexistent lymphadenopathy. Blastomycosis would be unlikely without additional changes in the bones. We can also eliminate paracoccidioidomycosis on epidemiologic grounds; however, we had been told that the patient visited areas where coccidioidomycosis and histoplasmosis are endemic. The calcifications seen in the liver are somewhat suggestive of histoplasmosis, although I would also expect to see calcifications in the spleen, suggestive of previous involvement with that disease; or splenomegaly, if the spleen were currently involved in an active infection of that type. Furthermore, the presence of mucosal ulcerations is frequently reported in individuals with disseminated histoplasmosis. I found no mention of that type of lesion in the protocol.

Other than tuberculosis, there is one bacterial disease, melioidosis, that should also be considered. Melioidosis has a number of forms, one of which is a chronic granulomatous disease involving the lymph nodes and the lungs. It is caused by the free living organism *Pseudomonas pseudomallei*. Originally described in derelicts in Rangoon, this disease occurs in Southeast Asia, and should be considered in any veteran of Vietnam who presents with a history such as this.[2] Although at one time speculation on how the organism was acquired centered on the inhalation of spores from helicopter dust, I believe current thinking tends to implicate the contamination of skin wounds as entry into the body. It should be also noted that this disease, like reactivation tuberculosis, tends to present when there are other physiologic stresses present such as surgery or intercurrent illness.

Having mentioned a number of potential causes of this man's illness, we should now turn to a consideration of the diagnostic procedure of choice. There are a number of possible procedures to be considered. I believe that the most judicious choice depends on the assessment of the relative likelihood of the diagnostic possibilities.

First, a biopsy of a peripheral lymph node would have the advantage of minimal risk. It should have a reasonable chance of attaining a diagnosis successfully, if one is convinced prior to surgery that the node is diseased. It certainly would be the best choice if the most likely diagnosis was thought to be either lymphoma or Hodgkin's disease.

One of the chronic granulomatous infections might be demonstrated as well.

A second choice for a diagnostic procedure would be a transbronchial lung biopsy. This procedure has the advantage of inspection of tissue other than lymph nodes, and it is the least invasive means of obtaining lung tissue by biopsy. However, its usefulness might be limited for the following reasons: the tissue obtained might exhibit only old scarring or noncaseating granulomas; or since such a small amount of tissue is usually obtained, the finding of a lymphoma might not be sufficient to type the neoplasm for therapy.

A third choice for a diagnostic procedure is an open lung biopsy. This would be the procedure of choice if some type of vasculitis were high on the list of considerations.

At this point, my differential diagnosis is largely between lymphoma or Hodgkin's disease, and one of the infectious causes. Although I suspect that the procedure performed in this case was a biopsy of one of the large subcutaneous nodes, I would be inclined toward transbronchial lung biopsy. However, there is a distinct possibility that either procedure will bring about a definitive diagnosis.

At this point, I do not feel that if I were taking care of this patient I could make a diagnosis with the present amount of information given. I therefore can only speculate as to the possible cause. Although I cannot rule out other infectious causes such as lymphoma or Hodgkin's disease, I favor the diagnosis of melioidosis. If in fact the patient proves to have a chronic granulomatous infection, I would further suspect the presence of another disorder providing an additional physiologic stress on the patient.

CLINICAL DIAGNOSIS

Chronic granulomatous infection, with possible melioidosis.

PATHOLOGY PRESENTATION

Sergey Lyubsky, MD (Pathologist): A biopsy of a supraclavicular lymph node was performed during the hospital admission. As is evident from microscopic sections, the lymph node structure is almost completely effaced and for the most part replaced by caseating epithelioid granulomas. In the areas of caseation there are abundant cryptococci, and on mucicarmine stain the capsules of the cryptococci are delineated. Under higher power budding cryptococci are noted.

Edward Gogel, MD (Assistant Professor of Clinical Medicine, State University of New York at Stony Brook): After the diagnosis of cryptococcosis was made, this patient had a prolonged clinical course that Dr Cantor will present. We will then review further the radiographs with Dr Clair, and additional pathologic findings with Dr Lyubsky.

Dr Cantor: Certain details from the original protocol have been omitted so as not to mislead Dr Nelson. The initial crytococcal antigen test of the spinal fluid was negative. However, the diagnosis of cryptococcosis was made by lymph node biopsy and was further confirmed by transbronchial biopsy; following these procedures, a second lumbar puncture was done and the cryptococcal antigen test was positive. The cryptococcal antigen test was also positive on subsequent follow up lumbar punctures during treatment.

The patient was treated with a long course of amphotericin that was complicated by renal insufficiency. This resolved after the treatment was completed. The total period of hospitalizaton was nine weeks. He was then seen as an outpatient every three weeks to observe his clinical course and to assess the efficacy of the treatment with the aid of repeated lumbar punctures. On successive spinal fluid specimens, the titers of cryptococcal antigen progressively decreased and eventually became negative. Unfortunately, within two months he presented again with weakness, fever, and weight loss; after further tests and procedures the patient was found to have another illness.

Dr Clair: To reaffirm some of Dr Nelson's comments, several conditions may be excluded due to radiographic findings, such as a Loeffler's pneumonia and eosinophilic granuloma. Loeffler's pneumonia is usually a peripheral air space disease; eosinophilic granuloma is usually somewhat more of an interstitial infiltrate without lymph node involvement. Rheumatoid lung involvement and Wegener's granulomatosis were dismissed as possibilities on clinical grounds. These two conditions may simulate one another, although Wegener's granulomatosis with radiologic findings in the lung usually produces symptoms, whereas rheumatoid involvement of the lungs is less likely to do so unless a fibrosing alveolitis is present.

Other less likely possibilities were metastatic disease, particularly epidermoid metastases, that might be considered because of the subsequent suspicion of cavitations. However, metastatic disease of the lung usually presents with well-defined lesions of various sizes; pulmonary metastases of generally uniform size are common only with thyroid and pancreatic neoplasms. Hodgkin's disease may present with hilar adenopathy and multiple pulmonary nodules. However, infectious disease is still most common; fungal disease may present in this manner, with irregular borders of the lesions and adenopathy. Post-primary or reinfection tuberculosis is unlikely in the presence of adenopathy, unless two separate disease processes are present. Immunocompromised hosts may be at risk for such unusual pathogens as atypical mycobacterium, cytomegalovirus (CMV) and other herpes viruses, cryptococcus, candida and protozoans, and Kaposi's sarcoma; all of these conditions may have radiographic findings similar to those seen in this patient.

Another interesting aspect of the infectious disease possibilities in this patient is the particular distribution of lesions on the chest radiograph. Inhalation of an infectious disease agent will produce lesions more often at the apices, whereas an agent distributed by the hematogenous route will be more likely to present in basal locations. The pattern on this patient's films is more diffuse; consequently it offers no help in determining the nature of the lesions, and raises the question of two or more coincident pathologic processes.

The liver deformities described as nodules were in fact two small punctate areas of calcification that are often seen in our patient population. Radiographic evidence of fewer than three calcifications in the liver suggests tuberculosis as the cause; with greater than three, the cause is more likely to be histoplasmosis. Such a dictum does not

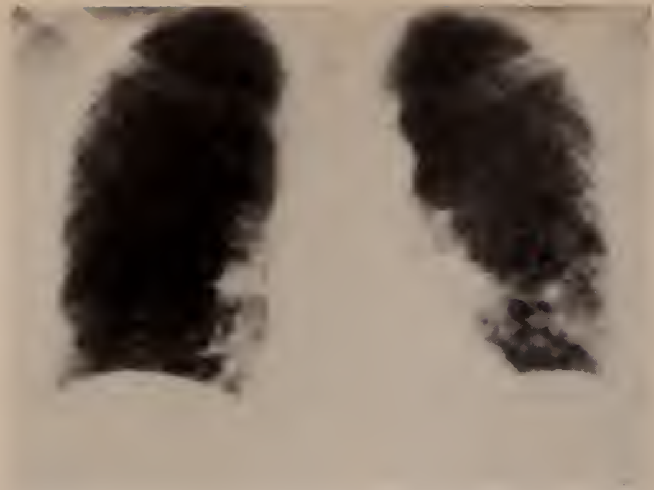


FIGURE 2. Chest radiograph during second hospital admission, showing decreased hilar fullness but increasing size of pulmonary nodules. Ill-defined left lower lobe infiltrate may represent pneumonia or atelectasis.

always hold true, but it is reasonable to remember.

Follow up CT scan on this patient two months after admission revealed that the paratracheal adenopathy had become less obvious; comparison with the earlier CT scan suggests also that the region of the right hilum is smaller in size. Thus there had been partial resolution of the adenopathy. However, the chest radiograph at this time (Fig 2) as well as several images of the lung fields on CT scan (Fig 3) show that in spite of the regression of the adenopathy, the nodular pattern throughout the lung fields had in fact progressed. Several pulmonary areas suggest coalescence; and indeed, in several regions the appearance is either that of coalescence of nodules or of cavitation. The cavitations do not show air-fluid levels. This is an observation that might have significance. The problem is now a more difficult one from a radiologic standpoint; the adenopathy is regressing, but the nodularity in the lungs is worsening. Certainly, these observations have excluded lymphoma and Hodgkin's disease, since they do not demonstrate such changes unless the patient has either received radiation therapy or has developed a superimposed infection.

Thus infectious disease is a major concern, but I am unable to detect why the parenchymal disease is worsening while the adenopathy is improving unless two distinct processes are present.

PATHOLOGY PRESENTATION

Dr Lyubsky: A transbronchial biopsy was performed on the second hospital admission; however, pathologic study of the biopsy material was negative.

Following thoracotomy, the specimen from the wedge resection of the lung measured 7 × 4 × 2 cm. On external examination there were five sacs filled with necrotic material bulging through the pleural surface. Otherwise the lung parenchyma appeared grossly unremarkable.

On microscopic examination focal subpleural collections of necrotic material without inflammatory reaction in the surrounding lung parenchyma are seen (Fig 4). The remaining lung parenchyma appear unremarkable, except for bubbly exudate in some of the alveolar spaces. With the use of methenamine-silver staining, abundant *Pneumocystic carinii* organisms were identified both within and outside the areas of necrosis (Fig 5). Special stains for other organisms were negative. On electron microscopic examination of this necrotic material, there were occasional type II pneumocytes with surfactant granules and *Pneumocystis* organisms (Fig 6).

It is unusual for pneumocystis pneumonia to present with necrosis of the lung parenchyma. To the best of our knowledge it was not described in the literature. Usually the morphologic changes consist of a bubbly exudate in the alvelolar spaces.[3,4]

Benjamin Luft, MD (Assistant Professor of Medicine, State University of New York at Stony Brook): After the diagnosis of cryptococcosis was made, the possibility that this patient may have had the acquired immune deficiency syndrome (AIDS) was considered based on the presence of a disseminated cryptococcal infection at a time when he had no known immunocompromising illness. Therefore, his illness could be included under the case definition of AIDS.

T-cell subset analysis at that time revealed a remarkable abnormality: most of the T-lymphoctes in the patient's peripheral blood were immature and carried both helper- and suppressor-cell markers on the same lymphocytes. This has not been described before in patients with AIDS, and we were perplexed by the possibility that perhaps this patient had an underlying lymphoma that had entered a leukemic phase. On follow up he developed abnormalities described on his subsequent chest films and required readmission. Tissue specimens were obtained from both lung and lymph nodes and forwarded to the National Cancer Institute for isolation of the HTLV-III virus. The virus could not be isolated from these tissues or from the patient's blood. However, serologic studies indicated HTLV-III infection in this patient. Therefore it was concluded that the patient did have an HTLV-III infection and that he most likely had AIDS.

A major point that was not made in taking his medical history was a more complete account of his sexual activities, that were ascertained only after the diagnosis of

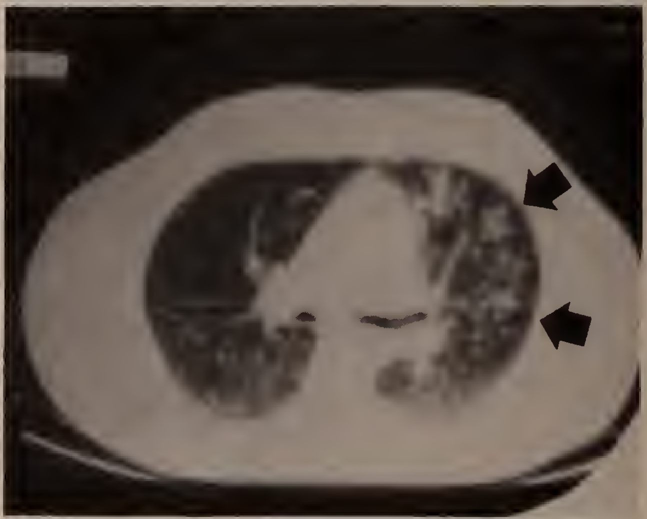


FIGURE 3. Chest CT scan during second hospital admission; lung window setting demonstrates diffusely scattered, ill-defined nodules (arrows) now 5 to 10 mm in size with possible cavitation.

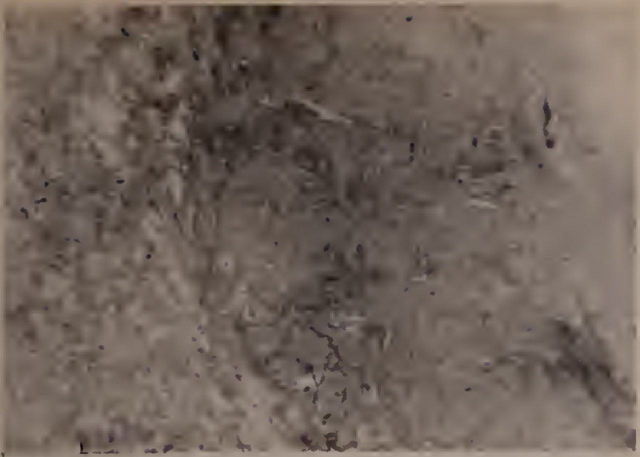


FIGURE 4. Specimen from wedge resection of the lung showing subpleural area of necrosis. (hematoxylin and eosin stain; original magnification × 25)

AIDS was made. He is neither a homosexual, nor a drug abuser, nor a hemophiliac, and he cannot be included in the usual risk groups that are associated with AIDS. A careful sexual history from the patient revealed that following his divorce seven years earlier, he began to be more promiscuous heterosexually—having sexual contact with approximately 50 to 60 partners per year over four years. Over this period, sexual relations with prostitutes was confined to fellatio. That is a very important point since it probably categorizes the patient within a risk group more akin to the type of AIDS that has been observed in central Africa.

Therefore, I wish to review recent data in relation to the expanding knowledge of this increasingly widespread disease. In particular, I would like to review some facets relating to HTLV-III infection, to the different populations that are at risk, and to the need to assess the importance of sexual behavior that may confront patients who have been identified as serologically positive for previous HTLV-III infection.

The original definition of AIDS that was used for epidemiologic purposes in order to identify most of the cases[5] included the following:

- The presence of a reliably diagnosed disease at least moderately indicative of cellular immune deficiency (eg, Kaposi's sarcoma in a patient less than 60 years of age, or pneumocystis pneumonia).
- The absence of known causes of underlying immune deficiency and of any other reduced resistance reported to be associated with the disease (eg, in patients not receiving immunosuppressive therapy, or in patients not having lymphoreticular malignancy).
- Life threatening illnesses that would be considered abnormal in a normal patient, including progressive multifocal leukoencephalopathy; cryptococcal meningitis or disseminated cryptococcosis; aspergillus pneumonia, and infections due to disseminated cytomegalovirus; progressive mucocutaneous herpes virus; and fungi, including esophageal candidiasis, disseminated *mycobacterium avium-intracellulare*, protozoa, *Pneumocystis*, toxoplasma, and cryptosporidium with enteritis.

However, most investigators realized that these manifestations could be just the tip of the iceberg, and that this case definition was insufficient to characterize all of the cases of AIDS or all of the people who were infected with the causative agent of AIDS. By this definition we were identifying only those patients who had the most severe manifestations of this infection. Other possible victims of AIDS might include patients with either other malignancies, with the generalized lymphadenopathy syndrome, with idiopathic thrombocytopenia purpura (ITP), or with transient immunologic defects. In addition, there was the likelihood that there were patients who were asymptomatic carriers of the AIDS virus. Asymptomatic carriers present a growing problem with regard to counselling since the causative agent of AIDS has now been identified.

The various risk groups that are predisposed toward either developing AIDS or becoming infected with the HTLV-III virus have been fairly well characterized to include homosexual and bisexual men, Haitians, transfusion recipients, heterosexual partners of patients with AIDS, hemophiliacs, intravenous drug abusers, and recipients of blood products. The syndrome is also endemic in central Africa. Other as yet unidentified groups would probably include such patients as the one which we are discussing today. He might be placed in the group of heterosexual partners of persons who have AIDS, since his frequent contact with prostitutes might have been the

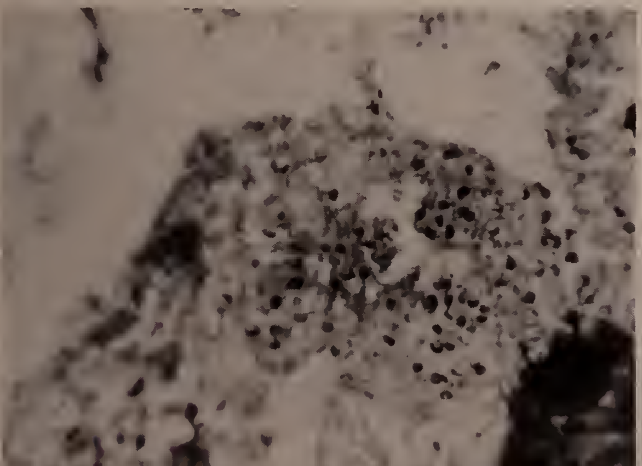


FIGURE 5. Resected lung tissue showing *Pneumocystis* organisms. (methenamine-silver stain; original magnification × 160)

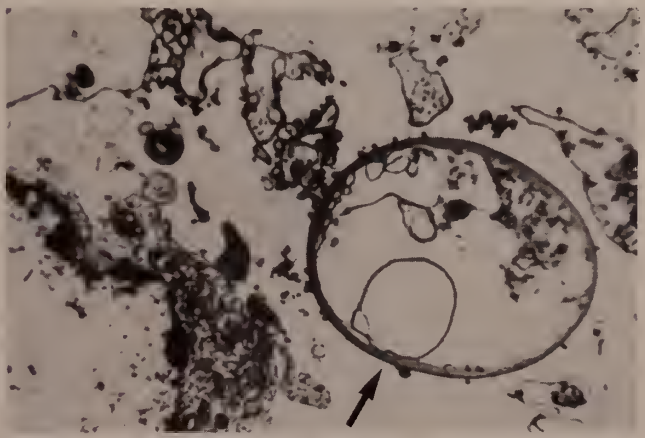


FIGURE 6. Electron micrograph of resected lung tissue showing *Pneumocystis carinii* organism **(arrow)**. (original magnification × 20,000)

source of this virus.

A study by Robert Gallo at the National Cancer Institute[6] firmly correlated the association of HTLV-III infection with AIDS, as well as with the AIDS-type of prodrome, including lymphadenopathy and fevers of unknown origin among patients who may be in a high-risk group. Of the patients who had a diagnosis of the lymphadenopathy type of AIDS prodrome, 86% had positive serologic tests for antibody against HTLV-III. Seventy-five percent of clinically normal mothers of juvenile AIDS patients had positive tests for HTLV-III infection. Patients with AIDS and Kaposi's sarcoma were positive in 30% of cases and adult AIDS patients with opportunistic infections were positive in 47.6% of cases. This was an early study but as the sensitivity of the serologic antibody tests was subsequently improved, the percentage of patients with AIDS who had Kaposi's sarcoma or opportunistic infections and who were antibody-positive increased to 100%, depending on the antigen preparation used and the method of antibody detection.[7-9]

Another study using a different radioimmunoassay technique to detect HTLV-III antibody indicated seropositivity in 97% of patients with AIDS, in 89% of patients with the progressive lymphadenopathy syndrome, in 59% of symptomatic homosexuals, and in 42% of persons who had contact with patients with AIDS or the lymphadenopathy syndrome. Thus there is positive serologic evidence of prior HTLV-III infection in a large percentage of people who are merely contacts with others who have had HTLV-III infection in the past. Antibody positive to HTLV-III infection was found in 25% or higher of healthy homosexuals, hemophiliacs, and intravenous drug abusers although studies in New York City indicate approximately a 50% positivity in this last group.[8,9]

The number of individuals who have positive serologic antibody responses to the HTLV-III virus is going to pose a special problem as to what information should be given to these people concerning their sexual practices and their social relationships. In addition, there seems to be a small number of patients who have the HTLV-III virus but who are negative on serologic testing for the infection.

The patient under discussion today appears to have the type of AIDS found in central Africa that was first reported by Clumeck and colleagues.[10] In central Africa 18 patients were afflicted with various illnesses including Kaposi's sarcoma, infection due to *Candida albicans*, cryptococcal meningitis, mucocutaneous genital herpes, toxoplasmosis, and tuberculosis. The latter infection is not one that would ordinarily be included in the case definition of AIDS. However, in patients from Haiti and central Africa tuberculosis has been found to be one of the earliest infections in patients who eventually develop AIDS. The patients in Dr Clumeck's study presented the classic prodrome with weight loss, diarrhea, fever, anorexia, skin rashes, and lymphadenopathy.

The epidemiology of AIDS in African populations show that many of the men who are infected do not engage in homosexual activity. Approximately 90% are married, and there is an equal proportion of female-to-male patients with AIDS in this population, though most of the women are unmarried. The men who tend to get AIDS are fairly promiscuous—frequenting prostitutes and having multiple sexual partners during the course of a year. A number of the female patients gave evidence that they are or have been prostitutes.

A study performed by Saxinger and co-workers[11] looked for HTLV-III antibodies in African patients by reexamining stored serum samples that had been collected prior to 1973 during a screening study for the prevalence of Epstein-Barr virus. Antibodies to HTLV-III were noted in 66% of these serum samples, indicating that this or a similar virus was present in Uganda prior to 1973. However AIDS either did not become manifest or perhaps no one noticed its presence in Africa until 1982. None of the individuals whose serum was found to be positive for HTLV-III went on to develop, or were known to have developed AIDS or a disease that was similar to AIDS. It is noteworthy that the group of individuals whose sera were studied were children averaging 6 years of age, and possibly that the behavior of the virus in children is different from that in adults, as is true for the Epstein-Barr virus and other infectious agents.

Richard Balter, MD (Clinical Assistant Professor of Medicine, State University of New York at Stony Brook): Is there any connection between the previous febrile illnesses in this patient during the 1960s and 1970s and his more recent clinical course?

Dr Luft: No, I do not think there is any connection, as far as the development of AIDS is concerned. To date, the HTLV-III virus has not been reported to be endemic to southeast Asia, where this patient lived for so long. The only available data concerning the presence of HTLV-III virus in 1973 was that which I detailed concerning its presence in Uganda.

Dr Gogel: When this patient was first found to have cryptococcal infection, the question was raised as to whether the HTLV-III serologic tests would be useful in determining whether this patient did in fact have AIDS. Are any of these tests useful in this connection, or do we still distinguish AIDS on the basis of the clinical syndrome and findings?

Dr Luft: Serologic tests for HTLV-III antibody are now available at blood banks and in out-of-state laboratories. New York State is developing an alternate-site testing program and county health departments have the test available. At the time of this patient's initial hospital admission, the test was not readily available. The test that we requested was done by a group of investigators at the National Cancer Institute. Now that the test is more readily available, it may cause difficulties for spouses of individual's with AIDS, other family members, contacts, and asymptomatic carriers. If these groups show positive serologic evidence of HTLV-III infection, what will you advise them concerning its significance? There is no good information concerning the risks of virus transmission when the serum is antibody-positive. The positive test tells you that the subject is at risk of transmitting the virus. However, it does not indicate the degree of certainty that such a transmission will take place as a result of normal sexual activity, or of the sharing of personal items such as toothbrushes and shaving equipment or of other close-contact activities. At the present time, it has been difficult to advise such individuals concerning precautionary changes in their way of life, based on the findings of a positive serologic test. The patients must be made aware of such a finding, and must make their own decisions as to what changes they are willing to make.

MaryAnn Lowen, MD (Medical Service): What do you recommend to people who are worried about being exposed to patients known to have AIDS?

Dr Luft: I tell such persons that they may have to change their way of life until more information is available as to the nature of transmission and the actual risk that seropositive individuals pose to their family and friends. Some changes might be quite drastic, such as abstinence from sexual activity or at the least the use of a condom. However, it is important to realize that there is no evidence to date indicating that the AIDS virus may be transmitted by casual contact.

FINAL DIAGNOSES

Acquired immunodeficiency syndrome, associated with human T-lymphotropic virus type III (HTLV-III) infection.

Caseating granulomas with abundant cryptococci in the supraclavicular lymph node.

Pneumocystis carinii pneumonia with focal necrosis.

REFERENCES

1. Case Records of the Massachusetts General Hospital. *N Engl J Med* 1977; 296:215–222.
2. Everett ED, Nelson RA: Pulmonary melioidosis. Observations in thirty-nine cases. *Am Rev Respir Dis* 1975; 112:331–340.
3. Hasleton PS, Curry A, Rankin EM: Pneumocystis carinii pneumonia: a light microscopical and ultrastructural study. *J Clin Pathol* 1981; 34:1138–1146.
4. Reichert CM, O'Leary TJ, Levens DL, Simrell CR, Macher AM: Autopsy pathology in the acquired immune deficiency syndrome. *Am J Pathol* 1983; 112:357–382.
5. Fauci AS, Masur H, Gelmann EP, Markham PD, Hahn BH, Lane HC: The acquired immunodeficiency syndrome: an update. *Ann Intern Med* 1985; 102:800–813.
6. Gallo RC, Salahuddin SZ, Popovic M, et al: Frequent detection and isolation of cytopathic retroviruses (HTLV-III) from patients with AIDS and at risk for AIDS. *Science* 1984; 224:500–503.
7. Sarngadharan MG, Popovic M, Bruch L, Schüpbach J, Gallo RC: Antibodies reactive with human T-lymphotropic retroviruses (HTLV-III) in the serum of patients with AIDS. *Science* 1984; 224:506–508.
8. Safai B, Sarngadharan MG, Groopman JE, et al: Seroepidemiological studies of human T-lymphotropic retrovirus type III in acquired immunodeficiency syndrome. *Lancet* 1984; 1:1438–1440.
9. Levy JA, Hoffman AD, Kramer SM, et al: Isolation of lymphocytopathic retroviruses from San Francisco patients with AIDS. *Science* 1984; 225:840–842.
10. Clumeck N, Sonnet J, Taelman H, et al: Acquired immunodeficiency syndrome in African patients. *N Engl J Med* 1984; 310:492–497.
11. Saxinger WC, Levine PH, Dean AG, et al: Evidence for exposure to HTLV-III in Uganda before 1973. *Science* 1985; 227:1036–1038.

INDEX

AUTHORS OF BOOK REVIEWS

Atkins, Harold L., 614
Bartholomew, Lee, 230
Bellin, Talah, 80
Berkman, Irwin J., 612
Bihari, Bernard, 272
Birrer, Richard, 49, 711
Bluestone, Naomi, 673
Bohorquez, Joseph L., 49
Brewer, John R., 271
Clarke, J.T.R., 167
Derby, Bennett M., 228
Dinardo, James A., 674
Doyle, Thomas L., 272
Edson, John N., 271, 713
Fein, Steven A., 519
Fontana, Vincent J., 168
Foster, Zelda, 613, 710
Gerson, Charles D., 518
Handelsman, Milton B., 49, 673
Hirschman, Shalom, 82
Imperato, Pascal James, 81, 167
Kagey, Betsy T., 712
Kahaner, Kenneth P., 273
Kay, Martin M., 612
Kuhn, Leslie A, 80
Landesman, Robert, 47
Lockshin, Michael Dan, 119
Matjasko, M. Jane, 228
Mebel, Frederick, 613
Minkin, Wilfred, 48
Moodley, Jules, 168
Moudy, Philip C., 118
Mustalish, Anthony C., 518
Nordlicht, Stephen, 118
Novick, Lloyd, 169
O'Connor, Robert J., 517
Ostrovsky, Paul, 674
Perry, Henry B. III, 120
Rodstein, Manuel, 169
Rosen, Gerald, 118
Rudolph, Nathan, 672
Salvoni, Daniele, 713
Sanko, Stephan R., 168
Scarpa, William J., 82
Schluger, Joseph, 170
Sheehy, John P., 47
Strosberg, James M., 712
Silver, Archie A., 273
Slade, John, 474
Spiegel, Allen, 119, 229
Spiera, Harry, 169
Stolfi, Julius E., 229, 671
Strosberg, James M., 712
Tenner, Michael S., 519
Thorbjarnarson, Bjorn, 517
Wolfe, Samuel, 671
Wolintz, Arthur H, 672

Intestinal fistula formation in a man with mycobacterial disease and AIDS

F. DUNCANSON, MD, J. PRABAKARAN, MD, S. SELVARAJ, MD, C. S. PITCHUMONI, MD

Homosexual men may be afflicted by a wide variety of protozoal and bacillary pathogens of the gastrointestinal tract. Aside from giardiasis, these disorders primarily involve the distal colon and rectum and rarely affect the upper gastrointestinal tract. The following is a report of a bisexual man who developed mycobacterial disease of the third portion of the duodenum with subsequent fistula formation. To our knowledge, this latter complication has not been previously reported in homosexual men nor in patients with the acquired immune deficiency syndrome (AIDS).

CASE REPORT

A 38-year-old man was admitted to the hospital complaining of fever, a 12 lb weight loss, and a constant, diffuse, moderately severe abdominal pain of three weeks duration. The abdominal pain became worse when he ate greasy and spicy foods but was relieved with antacids. The patient denied nausea, vomiting, or diarrhea. Although bisexual, he had only two male sexual partners for many years. His health had been excellent without other gastrointestinal illnesses. The patient had, however, noted nontender cervical lymphadenopathy for the past eight years. Purified protein derivative of tuberculin (5 TU PPD) was positive four months prior to admission, for which he received no treatment. The patient denied travel outside of New York, use of intravenous drugs, or blood transfusions. He was not of Haitian extraction.

In-hospital evaluation included blood and urine cultures that were negative and an upper gastrointestinal series that showed a bilobed defect in the proximal portion of the duodenum. A right supraclavicular lymph node biopsy revealed a large abscess with multiple granulomas showing caseation, occasional giant cells, and many acid-fast bacilli that despite direct culture inoculation, subsequently failed to grow. The patient was transferred to Misericordia Hospital Medical Center after the 17th hospital day for evaluation of persistent fever, chills, night sweats, diffuse abdominal pain, and for further investigation of the duodenal lesions.

Physical examination revealed an ill-appearing man in no acute distress. His temperature was 102°F; pulse rate, 130/min; and blood pressure, 130/90 mm Hg. Funduscopic examination revealed a cotton wool exudate in the left eye. Oral candidiasis was present. The patient had firm, nontender lymph nodes 0.5 to 2 cm in diameter in the anterior and posterior cervical regions and in the supraclavicular fossae bilaterally. Abdominal examination revealed a scaphoid abdomen that was tender on palpation in the periumbilical area with normal bowel sounds. There were no masses, ascites, or enlargement of the viscera noted. Rectal examination was unremarkable and no occult blood was noted in stool.

Laboratory examination included serial normal chest radiographs with no calcifications, pleural thickening, or apical infiltrates. Hemoglobin was 6.5 g/dL; hematocrit, 19.2%; mean corpuscular volume (MCV), 78 μm^3; mean corpuscular hemoglobin (MCH), 26.8 pg; and mean corpuscular hemoglobin concentration (MCHC), 33.3%. The white blood cell count was 4,000/μL with 64% segmented forms, 23% band forms, 2% myelocytes, 6% lymphocytes, 2% atypical lymphocytes, and 3% monocytes.

A 5 TU PPD and a Multitest CMI® (Merieux Institute, Miami, Florida) were both nonreactive. Upper gastrointestinal endoscopy showed a normal esophagus, stomach, and duodenal cap. A fistulous tract was seen emanating from the third portion of the duodenum. A 1 cm diameter submucosal elevation of the third portion of the duodenum was visualized and a biopsy was taken. The specimen consisted of white, purulent material; a Kinyoun stain of the pus revealed many acid-fast bacilli. Cultures of this material were directly inoculated onto Lowenstein-Jensen medium. These cultures were checked weekly for eight weeks but yielded no growth. Upper gastrointestinal series also showed a long fistulous tract extending from the third portion of the duodenum into the distal ileum (Fig). Computerized tomography (CT) of the abdomen showed no lymphadenopathy.

The patient was treated daily with oral doses of isoniazid 300 mg, rifampin 600 mg, and ethambutol 1,500 mg. Adequate absorption was assessed by confirming therapeutic serum levels of isoniazid and ethambutol. The patient was placed on parenteral hyperalimentation and received only medications by mouth. Over the next six weeks his fever abated, he became asymptomatic, and he gained 30 lbs. A repeat upper gastrointestinal endoscopy showed narrowing of the fistulous opening and a subsequent upper gastrointestinal series on hospital day 60 failed to show any fistula. The patient's weight gain and clinical improvement appeared to parallel the resolution of the fistula.

Lymphocyte studies utilizing OKT monoclonal antibodies (Roche Laboratories, Raritan, NJ) on day 6 showed 450 lymphocytes/μL, 77 B-cells/μL, 317 T-cells/μL,

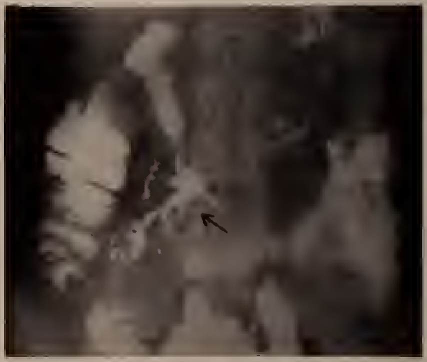


FIGURE. Upper gastrointestinal series showing a fistula (arrow) from the second part of the duodenum to the right lower quadrant.

From the Divisions of Infectious Diseases and Gastroenterology, Misericordia Hospital Medical Center, Bronx, NY and New York Medical College, Valhalla, NY.

Address correspondence to Dr Pitchumoni, Chief, Division of Gastroenterology, Misericordia Hospital Medical Center, 600 East 233rd St, Bronx, NY 10466.

35 total helper cells (OKT4)/μL (7.8%), and 220 total suppressor cells (OKT8)/μL (48.9%). The ratio of OKT4 to OKT8 was 0.16.

The patient subsequently developed disseminated cryptococcosis and *Pneumocystis carinii* pneumonia approximately 69 days after admission. He died on day 74 from progressive respiratory insufficiency; no autopsy was permitted.

DISCUSSION

Disseminated atypical mycobacterial infections have been frequently associated with AIDS and serve as a diagnostic criterion for AIDS.[1] Little attention, however, has been given to *Mycobacterium tuberculosis* infection in AIDS although this infection may be similar. Vieria and colleagues[2] and Pitchenik and associates[3] noted that 6/10 and 28/82 of their Haitian patients with AIDS had tuberculosis. The diagnosis of tuberculosis in these studies preceded the diagnosis of AIDS by one to 17 months.[2-4] The high frequency of tuberculosis in their study populations, combined with the observation that *Mycobacterium tuberculosis* is a more virulent pathogen than the opportunistic pathogens that have been commonly associated with AIDS may explain why this infection is more likely to cause disease at an earlier stage of immunosuppression.[3-4] Another explanation might be that such cases represent reactivation disease. A latent focus of *Mycobacterium tuberculosis* from a prior infection might become active as the patient's cellular immunity decreases. Several patients with AIDS who developed oral candidiasis, cutaneous anergy, lymphopenia, and reversed helper to suppressor T-cell ratios, and then developed disseminated tuberculosis were later subject to opportunistic infection (F. Duncanson, unpublished observation).

This patient had evidence of preexisting mycobacterial disease since he had a positive PPD test and cervical lymphadenopathy that showed numerous caseating granulomas and multiple acid-fast bacilli on lymph node biopsy. The positive PPD and the presence of granulomas suggest that his cellular immune system was at least partially intact at this time. Over an uncertain period of time, the patient developed a negative PPD, oral candidiasis, cutaneous anergy, lymphopenia, helper T-cell depletion, and reversal of his helper/suppressor T-cell ratio, indicative of marked cellular immune deficiency. It was at this time that the patient was found to have mycobacterial disease of the intestine as seen on endoscopic biopsy of pus from a swelling in the duodenal wall. The cause of the immunosuppression was not determined but it appeared to be related to AIDS. It is tempting to speculate that the patient had been incubating HTLV-III for an undetermined length of time. The virus would have led to a gradual waning of his cellular immune function permitting the mycobacteria to again disseminate and lead to the development of intestinal mycobacterial disease. However, this could not be proven. His cellular immunity continued to wane even more until he developed disseminated cryptococcosis and *Pneumocystis carinii* pneumonia.

Due to the absence of cultural confirmation, it is unclear whether this patient's infection was caused by *Mycobacterium tuberculosis* or by an atypical mycobacterium. Infection caused by *Mycobacterium tuberculosis* is suggested but not proven by the history of a positive tuberculin skin test four months prior to admission at the first hospital. It is also suggested by the finding of caseating granulomas with multiple acid-fast organisms both from the lymph node biopsy and from the duodenal lesion together with fistulous tract formation and the clinical response to three-drug antituberculous therapy. Further, the above findings are consistent with previously reported cases of tuberculosis of the duodenum. However, the absence of positive cultures despite the presence of numerous organisms on smears and biopsies is a finding more often seen with atypical mycobacterial infections than with tuberculosis of the duodenum.

In reviewing tuberculosis of the duodenum, Gleason and co-workers[5] emphasized that it is a rare disorder and in the period before chemotherapy was available, patients dying from pulmonary tuberculosis had intestinal involvement in 39 to 80% of cases. Eighty-five percent of intestinal tuberculous involvement occurred in the ileocecal region. In necropsy studies of 4,360 patients that died from tuberculosis, only 22 patients (0.5%) had duodenal tuberculosis.[5] Duodenal tuberculosis is also difficult to diagnose clinically. In their review of the literature, these authors were only able to find 49 patients who had localizing signs and symptoms—36% had vague epigastric or right upper quadrant pain. In most instances, the pain occurred after a meal but this was not always consistent. Twenty-seven patients had nausea and vomiting, 17 demonstrated weight loss, but only six had fever. Fistula formation was found to be a fairly uncommon complication of duodenal tuberculosis; an exact percentage is not specified.[5]

Definitive diagnosis of duodenal tuberculosis requires confirmation on tissue specimens, but indirect evidence may permit presumptive diagnosis. Upper gastrointestinal series may show extrinsic compression defects, stricture, obstructions, ulcerations, and polypoid changes of the duodenum. Chest radiographs and tuberculin skin tests are usually positive. Fiberoptic endoscopic examination may visualize tuberculous gastrointestinal involvement but biopsies obtained in this manner rarely lead to a diagnosis of duodenal tuberculosis since they often are not deep enough. Excisional biopsies have been the best methods for confirming the diagnosis.

Patients with AIDS who have disseminated infections due to *Mycobacterium avium-intracellulare* complex have been found to have gastrointestinal tract involvement. Histologic evidence of gastrointestinal involvement was found in three of ten patients studied at autopsy; the small intestine was involved in two of the patients and the rectum in one.[6] The authors noted macrophage collections containing acid-fast organisms. In another study, nonsuppurative granulomas with macrophages that contained numerous intracytoplasmic acid-fast bacilli were found in the small intestine of one of five patients that died of disseminated *Mycobacterium avium-intracellulare* infection.[7] Of eight patients with disseminated *Mycobacterium avium-intracellulare* infection, three showed massive small intestinal infiltration with histiocytes filled with acid-fast bacilli and who clinically had fever and malabsorption; these patients responded poorly to therapy.[8] From these studies, it would appear that gastrointestinal involvement of disseminated *Mycobacterium avium-intracellulare* infection does occur. However, the incidence and spectrum of gastrointestinal illness due to atypical mycobacteria has not been fully explained and must be studied further before conclusions and definitive statements can be made.

In the absence of confirmation of disease by culture, it is impossible to determine whether this patient had either tuberculosis or an atypical mycobacterial infection of the duodenum. The unusual presentation of this patient—intestinal mycobacterial disease complicated by an ileal-duodenal fistula that responded to antituberculous therapy—should be added to the growing clinical spectrum of mycobacterial diseases of the gastrointestinal tract observed in patients with AIDS.

REFERENCES

1. Greene JB, Sidhu GS, Lewin S, et al: Mycobacterium avium-intracellulare: a cause of disseminated life-threatening infection in homosexuals and drug abusers. *Ann Intern Med* 1982; 97:539–546.
2. Vieira J, Frank E, Spira TJ, Landesman SH: Acquired immune deficiency in Haitians: opportunistic infections in previous healthy Haitian immigrants. *N Engl J Med* 1983; 308:125–129.
3. Pitchenik AE, Cole C, Russell BW, Fischl MA, Spira TJ, Snider DE Jr: Tuberculosis, atypical mycobacteriosis, and the acquired immunodeficiency syndrome among Haitian and non-Hàitian patients in south Florida. *Ann Intern Med* 1984; 101:641–645.
4. Pitchenik AE, Fischl MA, Dickinson GM, et al: Opportunistic infections and Kaposi's sarcoma among Haitians: evidence of a new acquired immunodeficiency state. *Ann Intern Med* 1983; 98:277–284.
5. Gleason T, Prinz RA, Kirsch EP, Jablokow V, Greenlee HB: Tuberculosis of the duodenum. *Am J Gastroenterol* 1979; 72:36–40.
6. Gottlieb MS, Groopman JE, Weinstein WM, Fahey JL, Detels R: The acquired immunodeficiency syndrome. *Ann Intern Med* 1983; 99:208–220.
7. Sakowski P, Fligieb S, Berlin GW, Johnson BL Jr: Disseminated *Mycobacterium avium-intracellulare* infection in homosexual men dying of acquired immuno-deficiency. *JAMA* 1982; 248:2980–2982.
8. Gold J, Armstrong D: Diagnosis of opportunistic infections. The acquired immune deficiency syndrome and infections of homosexual men, in Ma P, Armstrong D (eds): New York, Yorke Medical Books, 1984.

Disseminated cryptococcosis presenting as the adult respiratory distress syndrome

ELLIOTT N. PERLA, MD, SHLOMO MAAYAN, MD, STEVEN N. MILLER, MD,
GITA RAMASWAMY, MD, HARVEY EISENBERG, MD

The adult respiratory distress syndrome caused by opportunistic pulmonary infectious agents is a well known complication for immunocompromised patients. Fungal infection due to *Cryptococcus neoformans* is a common cause of meningitis in such patients, and it may also be associated with a wide range of pulmonary disorders.[1,2] However, the adult respiratory distress syndrome due to cryptococcosis in immunocompromised patients has rarely been reported.[3-5] In this report a patient is described who appears to have the acquired immunodeficiency syndrome (AIDS) and who presented with the adult respiratory distress syndrome caused by disseminated cryptococcosis.

CASE REPORT

A 32-year-old man was admitted to Metropolitan Hospital Center on July 9, 1981 after complaining of weight loss and weakness of a few weeks' duration. He had apparently been well until three to four months prior to admission. At that time he began to develop intermittent diarrhea. For approximately one month prior to admission he had fever and a cough productive of frothy, mucoid sputum. The patient had been hospitalized in the recent past for a hemorrhoidectomy and for the removal of penile warts. The patient emigrated to the United States from Haiti three years prior to admission, and since that time had not traveled outside of the New York City area. He was unemployed at the time of admission but had previously worked as a musician and as a doorman. He lived with his wife and four children; they had no pets. The patient denied any homosexual activity, cigarette smoking, excessive ethanol consumption, or other drug abuse. He had received no medications prior to admission.

Physical examination revealed a lethargic, ill-appearing man in moderate respiratory distress. Vital signs included blood pressure, 100/60 mm Hg; pulse, 120/min; respiratory rate, 32/min; and temperature, 100.2°F. The patient had poor dentition and oral thrush. The neck was supple and small diffuse cervical lymphadenopathy was noted. Auscultation of the lungs revealed a few scattered rales posteriorly. Examinations of the heart, abdomen, and rectum were within normal limits. Clubbing of the digits was noted. No skin rashes were seen. Neurologic examination revealed an abnormal mental status characterized by lethargy and confusion.

Laboratory examination revealed a hemoglobin of 11.3 gm/dL; hematocrit, 33.1%; white blood cells, 7,200/μL; polymorphonuclear cells, 50%; band cells, 22%; lymphocytes, 25%; and metamyelocytes, 3%. The serum electrolytes, glucose, BUN, creatinine, and urine analysis were within normal limits. Initial arterial blood gas data revealed a Po_2 of 47 mm Hg, a Pco_2 of 27.4 mm Hg, and a pH of 7.50. A chest radiograph revealed extensive diffuse bilateral infiltrations (Fig 1). A sputum gram stain revealed leukocytes but no organisms and a sputum acid-fast bacillus smear was negative. A lumbar puncture was performed and the results included an opening pressure of 120 mm H_2O; 10 red blood cells/μL; 7 white blood cells/μL (all lymphocytes); glucose, 56 mg/dL (simultaneous serum glucose, 130 mg/dL); and protein, 56 mg/dL. Gram stain, acid-fast bacillus stain, and India ink preparation of the cerebrospinal fluid were all negative.

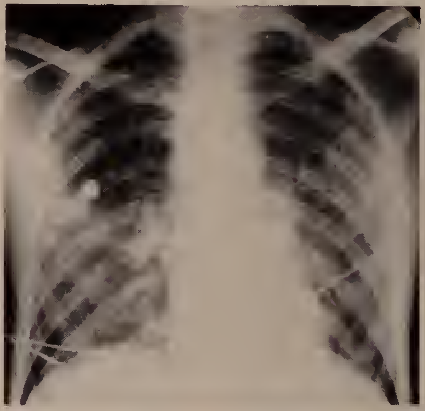


FIGURE 1. Chest radiograph revealing diffuse, relatively symmetrical bilateral infiltrates.

From the Departments of Medicine (Drs Perla, Maayan, Miller, and Eisenberg) and Pathology (Dr Ramaswamy), Metropolitan Hospital Center, New York Medical College, NY.

Address correspondence to Dr Perla, Assistant Professor of Medicine, Pulmonary Disease Section, Metropolitan Hospital Center, 1901 First Ave, NY 10029.

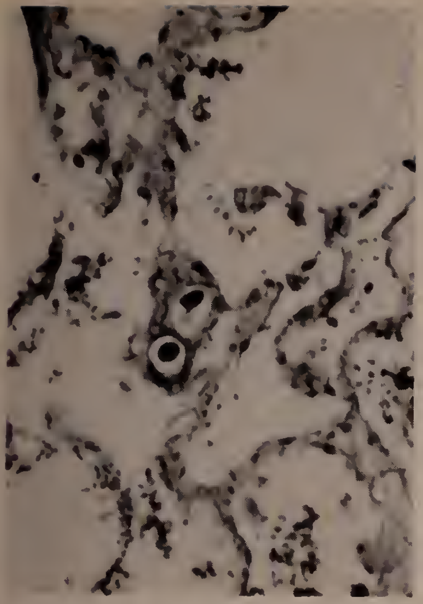


FIGURE 2. An open lung biopsy specimen showing cryptococcal organisms within alveolar septae. (Grocott's silver stain × 40)

The patient's initial therapy included supplemental inspired oxygen, isoniazid, rifampin, streptomycin, and intravenous cephalosporin. On the evening after admission there was a further deterioration of the patient's respiratory status. Endotracheal intubation and assisted mechanical ventilation were required. Over the next several days, continuous oxygen therapy with an F_IO_2 of .50 was required to maintain an arterial Po_2 of greater than 60 mm Hg. The patient remained on the ventilator throughout his hospital course.

On the sixth hospital day an open lung biopsy was performed. The specimen revealed minimal inflammatory response, but yeast like organisms were seen on Grocott's silver stain (Fig 2). A mucicarmine stain was also positive. Cultures of blood, urine, spinal fluid, pleural fluid, and lung tissue were all subsequently positive for *Cryptococcus neoformans*. The patient was treated with amphotericin-B and subsequently 5-flucytosine was also added. Serum cryptococcal antigen obtained on the eighth hospital day was positive in a titre of 1:16,000. Cryptococcal antigen of the cerebrospinal fluid obtained on the same day was positive in a titre of 1:256. On the eleventh hospital day, an India ink preparation of cerebrospinal fluid was positive for cryptococci.

During the third week of hospitalization the patient developed grand mal seizures. A computed tomography scan revealed a nonenhancing pontine mass with midline shift. The patient was given an Ommaya reservoir for the intrathecal instillation of amphotericin-B. Despite antifungal therapy his clinical status progressively deteriorated and on August 18, 1981, during the sixth week of hospitalization, the patient died.

At postmortem examination there was evidence of disseminated cryptococcosis involving the lungs, liver, kidneys, spleen, brain, lymph nodes, and bone marrow (Fig 3). There were no other infectious agents noted, and there was no evidence of lymphoma or other malignancies.

DISCUSSION

This patient came to the hospital with an illness due to disseminated infection by *Cryptococcus neoformans*. This infection produced a pattern of acute injury in the lungs that resulted in the adult respiratory distress syndrome.

Although acute respiratory failure precipitated by cryptococcosis or any other systemic fungal infection is unusual, fungal pneumonias should be included in the differential diagnosis of disorders that may be associated with the adult respiratory distress syndrome. Progressive, disseminated histoplasmosis generally does not result in respiratory failure,[6] but acute pulmonary histoplasmosis caused by massive inhalation has been associated with the adult respiratory distress syndrome.[7,8] Coccidioidomycosis may disseminate and cause respiratory failure, particularly in patients with predisposing risk factors.[9,10] Disseminated blastomycosis also has occasionally resulted in a lethal adult respiratory distress syndrome.[11-15]

It has long been known that disseminated cryptococcosis in certain immunocompromised individuals may result in widespread lesions of the lungs that are indistinguishable from those of miliary tuberculosis on radiographic and pathologic examination.[16-18] Nevertheless, the development of the adult respiratory distress syndrome, such as in the patient described here, has only rarely been described in the literature. Kent and Layton[3] describe two immunocompromised patients, an 18-year-old woman receiving steroids for hepatic disease, and a 21-year-old man receiving steroids for acute lymphatic leukemia, who appear most likely to have died from fulminant respiratory failure due to cryptococcosis. Sakowitz and Sakowitz[4] describe a 67-year-old man who had acute respiratory failure due to cryptococcosis while receiving systemic steroids for the treatment of keratitis. Ferguson and Smith[5] report on a case of fulminant cryptococcal lobar pneumonia associated with dissemination and acute respiratory failure in a 59-year-old man with Cushing's syndrome.

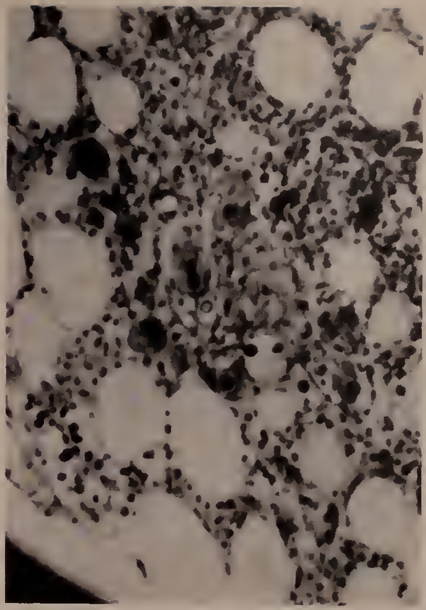


FIGURE 3. Budding cryptococcal organisms seen within a bone marrow specimen. (Grocott's silver stain × 40)

Another important aspect of the present case concerns the patient's risk factors for developing disseminated cryptococcosis. In retrospect, there is reason to believe that he had AIDS. The patient had never received systemic steroid therapy and he had no evidence of a malignancy or other obvious immunologic disorder at postmortem examination. However, the patient was an immigrant to this country from Haiti, and AIDS has been well described among Haitian immigrants.[19,20] Disseminated cryptococcosis is recognized as one of the opportunistic infections that may occur among patients with AIDS.[20,21]

In summary, the case presented here is one of disseminated cryptococcosis with the adult respiratory distress syndrome in a patient who probably had AIDS. The adult respiratory distress syndrome due to fungal disease is unusual, but disseminated crypotococcosis must be considered along with the more commonly encountered clinical entities such as *Pneumocystis carinii* infection, disseminated mycobacterial disease, and viral disease in the differential diagnosis for any immunocompromised patient with bilateral pulmonary infiltrates and respiratory distress. It is essential in such cases that vigorous efforts be made, including the use of biopsy procedures whenever necessary, to determine the precise cause of the disease.

Acknowledgment. The authors thank Dr Daniel J. Stone, Professor of Medicine, New York Medical College, for his review of this article.

REFERENCES

1. Kerkering TM, Duma RJ, Shadomy S: The evolution of pulmonary cryptococcosis. *Ann Intern Med* 1981; 94:611-616.
2. Gordonson J, Birnbaum W, Jacobson G, Sargent EN: Pulmonary cryptococcosis. *Radiology* 1974; 112:557-561.
3. Kent TH, Layton JM: Massive pulmonary cryptococcosis. *Am J Clin Path* 1962; 38:596-604.
4. Sakowitz AJ, Sakowitz BH: Disseminated cryptococcosis. Diagnosis by fiberoptic bronchoscopy and biopsy. *JAMA* 1976; 236:2429-2430.
5. Ferguson RP: Cryptococcosis and Cushing's syndrome. *Ann Intern Med* 1977; 87:65-66.
6. Smith JW, Utz JP: Progressive disseminated histoplasmosis. A prospective study of 26 patients. *Ann Intern Med* 1972; 76:557-565.
7. Kataria YP, Campbell PB, Burlingham BT: Acute pulmonary histoplasmosis presenting as adult respiratory distress syndrome: effect of therapy on clinical and laboratory features. *South Med J* 1981; 74:534-537, 542.
8. Wynne JW, Olsen GN: Acute histoplasmosis presenting as the adult respiratory distress syndrome. *Chest* 1974; 66:158-161.
9. Drutz DJ, Catanzaro A: Coccidioidomycosis, Part II. *Am Rev Respir Dis* 1978; 117:727-771.
10. Rowland VS, Westfall RE, Hinchcliffe WA, Jarrett PB: Acute respiratory failure in miliary coccidioidomycosis, in Ajello L (ed): Coccidioidomycosis: Current clinical and diagnostic status. Miami, Florida, Symposia Specialists, 1977, pp 139-155.
11. Evans ME, Haynes JB, Atkinson JB, Delvaux TC Jr, Kaiser AB: Blastomyces dermatitides and the adult respiratory distress syndrome. Case reports and review of the literature. *Am Rev Respir Dis* 1982; 126:1099-1102.
12. Lockridge RS Jr, Glauser FL: Adult respiratory distress syndrome secondary to diffuse pulmonary blastomycosis. *South Med J* 1979; 72:235-236.
13. Palmer PE, McFadden SW: Blastomycosis. Report of an unusual case. *N Engl J Med* 1968; 279:979-983.
14. Onal E, Lopata M, Lourenço RV: Disseminated pulmonary blastomycosis in an immunosuppressed patient. Diagnosis by fiberoptic bronchoscopy. *Am Rev Respir Dis* 1976; 113:83-86.
15. Sarosi GA, Davies SF: Blastomycosis. *Am Rev Respir Dis* 1979; 120:911-938.
16. Littman ML: Cryptococcosis (torulosis): current concepts and therapy. *Am J Med* 1959; 27:976-998.
17. McCullough NB, Louria DB, Hilbish TF, Thomas LB, Emmons C: Cryptococcosis: clinical staff conference of the National Institutes of Health. *Ann Intern Med* 1958; 49:642-661.
18. Haugen RK, Baker RD: The pulmonary lesions in cryptococcosis with special reference to subpleural nodules. *Am J Clin Path* 1954; 24:1384-1390.
19. Pitchenik AE, Fischl MA, Dickinson GM, et al: Opportunistic infections and Kaposi's sarcoma among Haitians: evidence of a new acquired immunodeficiency state. *Ann Intern Med* 1983; 98:277-284.
20. Vieira J, Frank E, Spira TJ, Landesman SH: Acquired immune deficiency in Haitians: opportunistic infections in previously healthy Haitian immigrants. *N Engl J Med* 1983; 308:125-129.
21. Gottlieb MS, Groopman JE, Weinstein WE, Fahey JL, Detels R: The acquired immunodeficiency syndrome. *Ann Intern Med* 1983; 99:208-220.

Pleural effusion due to communicating gastric ulcer

ROBERT D. BRANDSTETTER, MD, STEPHEN C. KLASS, MD, PHILIP GUTHERZ, MD,
WALTER NEGLIA, MD, DONALD J. PINALS, MD

Pleural effusion may be caused by diseases of the gastrointestinal tract.[1] However, it is unusual for gastrointestinal organs to rupture spontaneously into, or directly communicate with the pleural space. Esophageal rupture[2] and pancreaticopleural fistulas[3] have been reported to cause such complications, although rarely in the latter. The following report is of a patient who was found at autopsy to have had a fatal rupture of a gastric ulcer through the diaphragm into the left pleural cavity. Gastric rupture should be considered in the differential diagnosis of a pleural effusion, particularly in the presence of supradiaphragmatic air; if not promptly recognized and corrected surgically the outcome may be fatal.

CASE REPORT

A 63-year-old woman was admitted to the hospital because of uremic encephalopathy associated with intermittent nausea and vomiting for two months. During this period, the patient had ill-defined left shoulder pain of unknown cause and had received ibuprofen and hydromorphone hydrochloride. The patient had been recently treated for bladder carcinoma complicated by bilateral hydronephrosis by the implantation of percutaneous nephrostomy tubes. There was no known gastrointestinal disease.

On physical examination the patient responded to verbal stimuli. Her temperature was 97 °F; pulse, 108/min; blood pressure, 140/79 mm Hg; and respiration, 20/min. Crackles were noted throughout both lower lung fields. The abdomen was without tenderness or masses and the nephrostomy tubes were patent. A chest roentgenogram was normal without evidence of infiltrates or effusions.

Laboratory examination demonstrated a hematocrit of 37.6%; white blood cell count, 22,200/μL; and 293,000 platelets/μL. Electrolytes were within normal limits except for an elevated lactic dehydrogenase (LDH) of 1,000 IU/L and a serum blood urea nitrogen of 54 mg/dL, with creatinine, 3.6 mg/dL. The bilirubin was .6 mg/dL. Room air arterial blood gas revealed the following: pH, 7.23; Pco_2, 26 mm Hg; Po_2, 100 mm Hg; and Hco_3, 11 mEq/L. Microscopic urine analysis showed 10 white blood cells, 35 red blood cells, and gram-negative bacteria.

Shortly after admission the patient became tachypneic and hypotensive (70 mm Hg systolic). Urosepsis was suspected and broad spectrum antibiotics were instituted—penicillin, oxacillin, and gentamicin sulfate—as well as intravenous volume repletion. However, the patient sustained a cardiopulmonary arrest requiring ventilatory and hemodynamic support.

A central venous pressure (CVP) catheter was inserted into the left subclavian vein. A follow up supine roentgenogram revealed a left pleural effusion and irregular collections of air at the left lung base that were felt to be within the pulmonary parenchyma. A Swan-Ganz catheter was then inserted. The pulmonary artery and pulmonary capillary

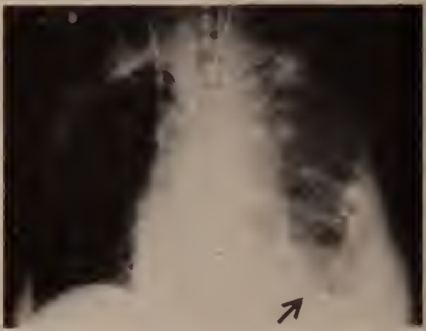


FIGURE 1. Supine anteroposterior chest roentgenogram demonstrating a streak of air (arrow) paralleling and probably lying just above the left hemidiaphragm in association with some fluid in the left pleural space.

From the Departments of Medicine, Radiology, and Pathology, New Rochelle Hospital Medical Center, New Rochelle, NY.

Address correspondence to Dr Brandstetter, Assistant Director, Department of Medicine, New Rochelle Hospital Medical Center, 16 Guion Pl, New Rochelle, NY 10802.

NEW YORK STATE
JOURNAL OF MEDICINE

INDEX
Volume 85
January–December
1985

Editor:
ALAN BLUM, MD

Deputy Editor:
PASCAL JAMES IMPERATO, MD

Published by the
MEDICAL SOCIETY OF THE STATE OF NEW YORK
PO Box 5404
420 Lakeville Road
Lake Success, NY 11042

ISSUE	NUMBER	PAGES
January	1	1-54
February	2	55-89
March	3	90-124
April	4	125-190
May	5	191-234
June	6	235-277
July	7	278-478
August	8	479-522
September	9	523-570
October	10	571-618
November	11	619-678
December	12	679-715

CLASSIFICATION CODES

CPC = Clinicopathologic Conference
CR = Case Report
ED = Editorial Commentary
HIST = History of Medicine
LE = Letter to the Editor
NF = News Feature
RPC = Correlation Conference in Radiology and Pathology

SUBJECT INDEX

- **Abortion**
  - Need for routine sonography prior to late abortion (ED), 4
- **Accidents**
  - Accidents in the skilled nursing facility, 202
- **Acquired Immunodeficiency Syndrome (see also AIDS)**
  - A 39-year-old man with thrombocytopenia, cough, fever, and shaking chills (CPC), 27
  - Disseminated toxoplasmosis with signs and symptoms of a cerebral lesion (CR), 40
- **Adenocarcinoma**
  - A 74-year-old man with a lung mass, arthritis, and difficulty walking (CPC), 647
- **Adolescents**
  - *Helping Youth Decide:* "When the fox preaches, beware the geese," 357
  - Normal adolescent development. Helping teenagers cope with change, 214
  - Obstacles to the prevention of teenage smoking, 360
- **Adult-onset Still's disease**
  - Adult-onset Still's disease (ED), 92
  - Adult-onset Still's disease and inflammatory orbital pseudotumor (CR), 110
- **AIDS (acquired immune deficiency syndrome)**
  - AIDS: a disease of ancient Egypt? (LE), 200
  - AIDS and the alleged Haitian connection (LE), 664
  - A 40-year-old man with chills, fever, weight loss, and a productive cough (CPC), 694
  - A 45-year-old man with weight loss, fever, chills, and diarrhea (CPC), 539
  - Disseminated cryptococcosis presenting as the adult respiratory distress syndrome (CR), 704
  - Disseminated histoplasmosis in a man with AIDS (LE), 665
  - Intestinal fistula formation in a man with mycobacterial disease and AIDS (CR), 702
- **Alcohol-related problems**
  - Preventing alcohol-related violence: the physician's role (ED), 56
- **Amaurosis fugax**
  - Neuro-ophthalmologic aspects of occlusive cerebrovascular disease, 18
- **Amylase**
  - Amylase in serous ovarian neoplasms (CR), 113
- **Aneurysm**
  - A 77-year-old woman with giant-cell arteritis, back pain, and shock (CPC), 502
- **Angioplasty**
  - Gruntzig balloon dilation for fibromuscular dysplasia of the internal carotid arteries (CR), 115
- **Appreciation**
  - Luther Terry, MD, 1911–1985, 293
  - Milton Rosenberg, MD, 1923–1985, 121
  - Our referees, 42
- **Arteriovenous malformation**
  - Arteriovenous malformation of the stomach (ED), 93
  - Massive gastric hemorrhage from the rupture of an arterial malformation (CR), 112
- **Arteritis**
  - A 77-year-old woman with giant-cell arteritis, back pain, and shock (CPC), 502
- **Atelectasis**
  - Rounded atelectasis (ED), 527, (CR), 563
- **Bacterial endocarditis**
  - A 45-year-old man with weight loss, fever, chills, and diarrhea (CPC), 539
- **Black Americans (see also Minorities)**
  - Black doctors on television, 151
  - Black-Jewish relationships. Access to health services and health careers, 137
  - Black woman physician, 145
  - Comparison of selected health indicators for black and white children in New York State, 131
  - Coronary heart disease in black Americans, 164
  - Eugene Percy Roberts: black physician and leader, 143
  - Gastrointestinal malignancies in black Americans, 157
  - Hypertension in blacks. Is it a different disease?, 160
  - Inequality in the availability of black physicians, 139
  - Minorities and New York medical schools. Past failures and the prospect for future successes, 148
  - Racial equality in American health care system: an unfulfilled ideal, 125
  - Restraint of progress. Declining participation of blacks and other minorities in medical schools, 135
  - Sarcoidosis: focus on the black patient, 162
- **Board of Trustees**
  - Board of Trustees 1984–1985, 174
- **Books received, annotations of**
  - 83, 231, 567
- **Books reviewed**
  - *A Calculus of Suffering. Pain, Professionalism, and Anesthesia in Nineteenth-Century America* (Pernick), 674
  - *A Child Dies. A Portrait of Family Grief* (Arnold, and Gemma), 613
  - *African Fever: A Study of British Science, Technology, and Politics in West Africa, 1781–1864* (Carlson), 167
  - *Alexander Fleming. The Man and the Myth* (Macfarlane), 229
  - *Alternatives in Health Care Delivery: Emerging Roles for Physician Assistants* (Carter, and Perry), 229
  - *Alzheimer's Disease: The Standard Reference* (Reisberg, ed.), 273
  - *An Atlas of Critical Care Roentgenography* (Victor, ed.), 674
  - *Antinuclear Antibodies. Contemporary Techniques and Clinical Application to Connective Tissue Diseases* (McCarty, Valencia, and Fritzler), 119
  - *A Practical Handbook of Joint Fluid Analysis* (Gatter), 230
  - *A Private Practice*, (Reilly), 48
  - *A Smoking Gun: How The Tobacco Industry Gets Away with Murder* (Whelan), 474
  - *Atlas of Osteoarthritis* (Meisel, and Bullough), 712
  - *Canadian Medicine: A Study in Restricted Entry* (Hamowy), 671
  - *Cancer Therapy in Children* (Pochedly, ed.), 118
  - *Cardiology 1984* (Roberts, ed.), 170
  - *Chronic Exposure of Mice to Cigarette Smoke* (Henry, and Kouri), 474
  - *Chronic Ulcers of the Skin* (Lee), 613
  - *Clinical Anesthesia in Neurosurgery (Frost), 228*
  - *Color Atlas of Geriatric Medicine* (Kamal, and Brocklehurst), 169
  - *Color Atlas of the Newborn* (Milner, and Herber), 672
  - *Computed Tomography of Congenital Brain Malformations* (Sarwar), 519

Conversations in Medicine. The Story of Twentieth-Century American Medicine in the Words of Those Who Created It (Weisse), 671
Diabetes: The Glucograf Method of Normalizing Blood Sugar (Bernstein), 49
Diagnostic Procedures in Cardiology: A Clinician's Guide (Warren, and Lewis, eds.), 713
Differential Therapeutics in Psychiatry: The Art and Science of Treatment Selection (Frances, Clarkin, and Perry), 118
Doppler Echocardiography (Goldberg, Allen, Marx, and Flinn), 612
Doppler Ultrasound in Cardiology. Physical Principles and Clinical Applications (Hatle, and Angelsen), 519
Double Exposure. Women's Health Hazards on the Job and at Home (Chavkin, ed.), 712
Drug Treatment of Heart Failure (Cohn, ed.), 82
Health and Healing. Understanding Conventional and Alternative Medicine (Weil), 120
Hepatic Radiography (Bernardino, and Sones), 712
"I Love Not Smoking!" A Coloring Book for Non-Smoking Children (Dehr, and Bazar), 476
Infections: Recognition, Understanding, Treatment (Pechere and Cherubin, eds.), 82
Inflammatory Bowel Disease (Shiratori, and Nakano, eds.), 518
Invasive Uroradiology. A Manual of Diagnostic and Therapeutic Techniques (Elyaderani, Kandzari, Castaneda, and Lange), 118
Life Stress and Coronary Heart Disease (De Faire, and Theorell), 271
Maingot's Abdominal Operations (Schwartz, and Ellis, eds.), 517
Major Depression: Prevention and Treatment (Lowry), 273
Making an Issue of Child Abuse. Political Agenda Setting for Social Problems (Nelson), 168
Management of Chronic Schizophrenia (Caton, ed.), 272
Medicine and the Management of Living. Taming the Last Great Beast. (Arney, and Bergen), 710
Merritt's Textbook of Neurology (Rowland, ed.), 228
Operative Perinatology: Invasive Obstetric Techniques (Iffy, and Charles, eds.), 47
Origins and Evolution of Behavior Disorders. From Infancy to Early Adult Life (Chase, and Thomas), 612
Pediatric Risk Factors for Major Chronic Disease (Williams), 271
Practical Ophthalmic Problems (March), 672
Preterm Birth: Causes, Prevention and Management (Fuchs, and Stubblefield, eds.), 168
Principles of Sports Medicine (Scott, Nisonson, and Nicholas, eds.), 47
Prison Health Care (Smith), 169
Progress in Cardiology (Yu and Goodwin, eds.), 80
Prospective Payment. What It Is/How to Cope. Your Questions Answered, 119
Reforming Medicine: Lessons of the Last Quarter Century (Sidel and Sidel, eds.), 45
Schizophrenia: Straight Talk for Families and Friends (Walsh), 673
Technological Basis of Radiation Therapy: Practical Clinical Applications (Levitt, and Tapley, eds.), 49
Textbook of Nuclear Medicine, Volume I: Basic Science (Harbert, and Goncalves da Rocha, eds.), 614
Textbook of Nuclear Medicine, Volume II: Clinical Applications (Harbert, and Goncalves da Rocha, eds.), 614
The Addictive Behaviors (Shaffer, and Stimmel), 272
The Aging Musculoskeletal System: Physiological and Pathological Problems (Nelson, and Dwyer, eds.), 169
The AMA and US Health Policy Since 1940 (Campion), 517
The Diabetic's Brand-Name Food Exchange Handbook. Food Exchanges for Over 3,000 Supermarket, Grocery Store, and Fast-Food Products (Barrett), 673
The Gene Doctors: Medical Genetics at the Frontier (Baskin), 167
The Health of China (Sidel and Sidel), 81
The Horror of Life (Williams), 80
The Lives of Lesions Chronology in Dermatopathology (Ackerman, and Ragaz), 48
The Practice of Emergency Care (Cosgriff, Jr., and Anderson), 518
The Psychopharmacology of Smoking (Mangan, and Golding), 474
The Smoke Ring (Taylor), 474
To Provide Safe Passage: The Humanistic Aspects of Medicine (Rabin, and Rabin, eds.), 711

Breast
Erythroleukemia in the adjuvant treatment of breast cancer (CR), 601
Silicone granulomas of the breast (CR), 599

Breast cancer
Breast self-examination (LE), 664
Breast self-examination—a practice whose time has come? (ED), 482
Current status of research in breast self-examination (ED), 480
Has breast self-examination had a fair trial? (ED), 479

Bronchial artery embolization
Treatment of hemoptysis via bronchial artery embolization (CR), 38

BUGA-UP
BUGA-UPdate, 434

Burns
Scalds of the lap (LE), 485

Cancer
A 28-year-old man with a left-sided palatal mass (CPC), 585
A 74-year-old man with a lung mass, arthritis, and difficulty walking (CPC), 647
Breast self-examination (LE), 664
Breast self-examination—a practice whose time has come? (ED), 482
Cigarette smoking and ill health among black Americans, 344
Clinical and pathologic features of endolymphatic stromal myosis (ED), 237
Clinical significance of serum lactic dehydrogenase in chronic lymphocytic leukemia, 685
Current status of research in breast self-examination (ED), 480
Disease model of cigarette use, 294
Erythroleukemia in the adjuvant treatment of breast cancer (CR), 601
Gastrointestinal malignancies in black Americans, 157
Has breast self-examination had a fair trial? (ED), 479
Humoral hypercalcemia of malignancy (pseudohyperparathyroidism) (LE), 242
Humoral hypercalcemia of malignancy. The role of parathyroid hormone (ED), 235
Indoor air quality standard for ambient tobacco smoke based on carcinogenic risk, 381
Japan: land of the rise in lung cancer. US government aids cigarette sales, 425
Lactic dehydrogenase and malignancy (ED), 679
Leiomyosarcoma of the ovary (CR), 218
"Less hazardous" cigarette: a deadly delusion, 313
Long-term survival with metastatic uterine sarcoma (ED), 195
Metastatic leiomyosarcoma of iliac bone (CR), 220
Primary osteogenic sarcoma of the skull. Five-year disease-free survival following surgery and high dose methotrexate therapy (CR), 598
Smoke-free society by the year 2000 (ED), 290
Stage II benign cystic teratoma with malignant squamous degeneration (CR), 224
The negligible role of ethylene dibromide in overall human cancer risk, 103

Candida tropicalis
Candida tropicalis papillary necrosis treated with ketoconazole (LE), 97

Carcinogens
(see also Smoking)
The negligible role of ethylene dibromide in overall human cancer risk, 103

Cardiac siderosis
Cardiac siderosis in thalassemia major (CR), 513

Carotid artery stenosis
Quantitative Doppler spectrum analysis of extracranial carotid artery stenosis, 577

Cataphoresis
Medical cataphoresis. Electrical experimentation and 19th century therapeutics (HIST), 257

Cephalosporins
Impact of third-generation cephalosporins on clinical outcome of gram-negative meningitis in a municipal hospital (LE), 243

Chest pain
Thrombophlebitis: an unusual cause of chest pain (LE), 609

Chlorpromazine
Chlorpromazine in experimental gastric ulcers induced by restraint and cold stress, 101

Cholecystitis
Acute cholecystitis due to *Aeromonas hydrophila* (CR), 564

Chronic brain syndrome
Fiscal and moral considerations of chronic brain syndrome (LE), 9

Clinicopathologic Conferences (series)
A 28-year-old man with a left-sided palatal mass, 585
A 74-year-old man with a lung mass, arthritis, and difficulty walking, 647
A 40-year-old man with chills, fever, weight loss, and a productive cough, 694
A 39-year-old man with thrombocytopenia, cough, fever, and shaking chills, 27

A 45-year-old man with weight loss, fever, chills, and diarrhea, 539
A 77-year-old woman with giant-cell arteritis, back pain, and shock, 502
Clitoris
Glomus tumor of the clitoris (LE), 611
Cocaine
Cocaine-induced myocardial infarction (CR), 660
Colonic varices
Massive lower gastrointestinal hemorrhage secondary to colonic varices (CR), 604
Commentaries
(see also **Editorial Commentaries**)
Coronary artery
Spontaneous dissection of the right coronary artery (LE), 485
Correlation Conferences in Radiology and Pathology (series)
A pelvic mass with periosteal reaction, 106
Councilors
Councilors 1984–1985, 174
Cyclosporine
Cyclosporine: preliminary experience in 79 patients with renal transplants, 531
Immunosuppression for renal allograft recipients: The role of cyclosporine (ED), 523
Cyst
Solitary nonparasitic liver cysts (LE), 95
Cystic lymphangioma
Retroperitoneal cystic lymphangioma (CR), 36
Cytomegalovirus
A 39-year-old man with thrombocytopenia, cough, fever, and shaking chills (CPC), 27
Diabetes mellitus
Advances in the treatment of diabetes mellitus (LE), 200
Management of diabetes mellitus in children, 251
Diverticulitis
Recurrent chronic epididymitis as initial symptom of colovesical fistula (CR), 222
Doppler recordings
Quantitative Doppler spectrum analysis of extracranial carotid artery stenosis, 577
Drug abuse
Psychiatric approach to drug abuse (LE), 96
Editorial Commentaries
Adult-onset Still's disease, 92
Application of non-invasive diagnostic techniques to the pediatric patient, 528
Arteriovenous malformation of the stomach, 93
Breast self-examination—a practice whose time has come?, 482
Charitable obligations of the physician, 574
Clinical and pathologic features of endolymphatic stromal myosis, 237
Competing agenda in smoking control agencies. "*Those who pay the piper...*", 287
Current status of research in breast self-examination, 480
Emergency medical services system in New York State: time for a change, 1
Has breast self-examination had a fair trial?, 479
Hemodynamic monitoring of the patient in shock, 238
Hodgkin's disease in siblings, 91
Humoral hypercalcemia of malignancy. The role of parathyroid hormone, 235
Identifying cost effective diagnostic strategies, 680
If smoking killed baby seals..., 282
Immunosuppression for renal allograft recipients: The role of cyclosporine, 523
Laboratory ordering: Bridging the gap, 693
Lactic dehydrogenase and malignancy, 679
Legal and administrative barriers to health care, 126
Linnaeus: right and wrong. On the 250th anniversary of his MD thesis and of his first edition of *Systema Naturae*, 682
Long-term survival with metastatic uterine sarcoma, 195
Massive hemoptysis, 573
Maternal and child health care: a deepening crisis, 128
Membranoproliferative glomerulonephritis, 571
Multiple pregnancies. Spontaneous and induced, 525
Need for routine sonography prior to late abortion, 4
New "regulators" of health care, 6
Parathyroid adenoma in children, 237
Practical aspects of immunohistochemistry, 191
Preventing alcohol-related violence: the physician's role, 56
Psychiatry and the prevention of violence, 57
Reflecting on the lectures of Professor Budd, 529
Rounded atelectasis, 527
Sarcoidosis revisited, 240
Searching for alternatives to war, 55
Smoke-free society by the year 2000, 290
Status of Asian Americans in the health care delivery system in New York, 153
Strengths of organized medicine, 576
Transurethral endoscopy of upper urinary tract, 194
Ureteropyeloscopy, 193
Education
(see also **Medical Education**)
Cigarette companies go to college, 349
The Comprehensive Smoking Education Act, 363
Electron microscopy
Practical aspects of immunohistochemistry (ED), 191
Embolism
Amniotic fluid embolism (CR), 265, (CR), 267
Emergency medical care
Emergency medical services system in New York State: time for a change (ED), 1
Emergency medical services system
Emergency medical services system in New York State: time for a change (ED), 1
Employment
Loss of jobs due to *not* smoking, 323
Endolymphatic stromal myosis
Clinical and pathologic features of endolymphatic stromal myosis (ED), 237
Endolymphatic stromal myosis—a misconception (LE), 242
Endoscopy
Transurethral endoscopy of upper urinary tract (ED), 194
Eosinophilia
Factitious hypoglycemia associated with eosinophilic leukemoid reaction (CR), 34
Epidermoid cysts
Epidermoid cysts of the cranium (CR), 596
Erythroleukemia
Erythroleukemia in the adjuvant treatment of breast cancer (CR), 601
Esophageal obstruction
Nd:YAG laser photocoagulation (LE), 59
Esophageal ulceration
Esophageal ulceration associated with oral penicillin in Marfan's syndrome (LE), 199
Esophagitis
Type I herpes simplex esophagitis with candidal esophagitis in an immunocompetent host (CR), 655
Esophagus
A large swallowed foreign body in the esophagus (LE), 610
Ethics
Cigarette marketing: ethical conservatism or corporate violence?, 324
Ethical double standard (LE), 244
Institutional ethics and graduate medical education, 590
When researchers accept funding from the tobacco industry, do ethics go up in smoke?, 451
Ethylene dibromide
The negligible role of ethylene dibromide in overall human cancer risk, 103
Falope ring
Tubal sterilization with the Falope ring in an ambulatory-care surgical facility, 98
Farmer's lung
Farmer's lung in a 10-year-old boy (CR), 658
Fetal viability
Need for routine sonography prior to late abortion (ED), 4
Fibroma
Fibroma of the lung (CR), 37
Fibromuscular dysplasia
Gruntzig balloon dilation for fibromuscular dysplasia of the internal carotid arteries (CR), 115
Fistula
Intestinal fistula formation in a man with mycobacterial disease and AIDS (CR), 702
Foreign body
A large swallowed foreign body in the esophagus (LE), 610
Gallbladder disease
Application of non-invasive diagnostic techniques to the pediatric patient (ED), 528
Neonatal hydrops of the gallbladder. Evaluation by cholescintigraphy and ultrasonography (CR), 565
Gastric ulcers
Chlorpromazine in experimental gastric ulcers induced by restraint and cold stress, 101
Gastrointestinal bleeding
Arteriovenous malformation of the stomach (ED), 93
Massive gastric hemorrhage from the rupture of an arterial malformation (CR), 112
Gastrointestinal disease
Gastrointestinal malignancies in black Americans, 157
Massive lower gastrointestinal hemorrhage secondary to colonic varices (CR), 604
Pleural effusion due to communicating gastric ulcer (CR), 706
Recurrent chronic epididymitis as initial symptom of colovesical fistula (CR), 222

Glomus tumor
Glomus tumor of the clitoris (LE), 611
Glossary
Glossary of tobacco terms, 297
Gonadotropins
Use of gonadotropins in ovulation induction. Analysis of treatment protocols and pregnancy outcome, 580
Granuloma
A 40-year-old man with chills, fever, weight loss, and a productive cough (CPC), 694
Silicone granulomas of the breast (CR), 599
Gruntzig balloon dilation
Gruntzig balloon dilation for fibromuscular dysplasia of the internal carotid arteries (CR), 115
Health benefits
New "regulators" of health care (ED), 6
Health care
Comparison of selected health indicators for black and white children in New York State, 131
Future patterns of chronic disease incidence, disability, and mortality among the elderly. Implications for the demand for acute and long-term health care, 623
Health care payment reform. Projections for next 15 years, 644
Implications of changes in payment practices for health care. The insurer's perspective, 641
Implications of new medical technology, 634
Legal and administrative barriers to health care, 126
Maternal and child health care: a deepening crisis, 128
Physicians, smoking, and health in the Netherlands, 394
Racial equality in American health care system: an unfulfilled ideal, 125
Reflections on health care at the coming turn of the century, 619
Restraint of progress. Declining participation of blacks and other minorities in medical schools, 135
Status of Asian Americans in the health care delivery system in New York, 153
The roles of new medical technology and health care delivery systems in shaping medicine into the 21st century, 637
Health care costs
Maternal and child health care: a deepening crisis, 128
New "regulators" of health care (ED), 6
Heart disease
Cigarette smoking and ill health among black Americans, 344
Coronary heart disease in black Americans, 164
Pseudoinfarction pattern associated with myocardial bridging of the left anterior descending artery (CR), 510
Hemodynamic monitoring
Hemodynamic monitoring of the patient in shock (ED), 238
Hemoptysis
Massive hemoptysis (ED), 573
Massive hemoptysis due to retained rib fragment 40 years after thoracotomy (CR), 605
Treatment of hemoptysis via bronchial artery embolization (CR), 38
Hemorrhage
Massive lower gastrointestinal hemorrhage secondary to colonic varices (CR), 604
Hernia
Repair of inguinal hernia with Marlex mesh (LE), 486
Herpes
Type I herpes simplex esophagitis with candidal esophagitis in an immunocompetent host (CR), 655
Highway fatalities
Violence on the highway. A national public health problem, 69
Histoplasmosis
Disseminated histoplasmosis in a man with AIDS (LE), 665
History of Medicine
Medical cataphoresis. Electrical experimentation and 19th century therapeutics, 257
Medical education in New York City in 1866–1867. A student's notebook of Professor Charles A. Budd's lectures on obstetrics at New York University—Part I, 488
Medical education in New York City in 1866–1867. A student's notebook of Professor Charles A. Budd's lectures on obstetrics at New York University—Part II, 548
The physician and war. A layman's view, 77
The poultice, 207
Hodgkin's disease
Hodgkin's disease in half-siblings (CR), 109
Hodgkin's disease in siblings (ED), 91
House Committee on Bylaws
House Committee on Bylaws, 183
House of Delegates
House of Delegates, 171
1985 House of Delegates Members
1985 House of Delegates Members, 176
Hypercalcemia
Humoral hypercalcemia of malignancy (pseudohyperparathyroidism) (LE), 242
Humoral hypercalcemia of malignancy. The role of parathyroid hormone (ED), 235
Hypertension
Hypertension in blacks. Is it a different disease?, 160
The relationship of thrombocytopenia to the onset of persistent pulmonary hypertension of the newborn in the meconium aspiration syndrome, 245
Hypoglycemia
Factitious hypoglycemia associated with eosinophilic leukemoid reaction (CR), 34
Immunohistochemistry
Practical aspects of immunohistochemistry (ED), 191
Immunosuppression
Immunosuppression for renal allograft recipients: The role of cyclosporine (ED), 523
Infant health
Abdominal distention associated with lactose malabsorption in a premature infant (CR), 227
Application of non-invasive diagnostic techniques to the pediatric patient (ED), 528
Comparison of selected health indicators for black and white children in New York State, 131
Maternal and child health care: a deepening crisis, 128
Inflammatory orbital pseudotumor
Adult-onset Still's disease and inflammatory orbital pseudotumor (CR), 110
Informed consent
Informed consent. The New York approach, 210
Insurance
Implications of changes in payment practices for health care. The insurer's perspective, 641
The impact of technology on laboratory ordering strategies, 690
Interstitial pregnancy
Ultrasound diagnosis of interstitial pregnancy (CR), 655
Intrauterine device
Asymptomatic perforation of the small intestine by a Copper-7 intrauterine device (CR), 515
Ischemia
Neuro-ophthalmologic aspects of occlusive cerebrovascular disease, 18
Ketoconazole
Candida tropicalis papillary necrosis treated with ketoconazole (LE), 97
Kidney disease
Cyclosporine: preliminary experience in 79 patients with renal transplants, 531
Hypertension in blacks. Is it a different disease?, 160
Immunosuppression for renal allograft recipients: The role of cyclosporine (ED), 523
Increased survival time of renal allograft recipients, 534
Membranoproliferative glomerulonephritis (ED), 571
Laboratory
The impact of technology on laboratory ordering strategies, 690
Laboratory ordering: Bridging the gap (ED), 693
Lactic dehydrogenase
Clinical significance of serum lactic dehydrogenase in chronic lymphocytic leukemia, 685
Lactic dehydrogenase and malignancy (ED), 679
Lactose malabsorption
Abdominal distention associated with lactose malabsorption in a premature infant (CR), 227
Laparoscopy
Tubal sterilization with the Falope ring in an ambulatory-care surgical facility, 98
Laser therapy
Nd:YAG laser photocoagulation (LE), 59
Lead poisoning
Effects of present and past occupational exposures (LE), 8
Legionella pneumophila
Seroepidemiology of *Legionella pneumophila* serogroup 1 in healthy residents of New York City, 10
Legionellosis
Seroepidemiology of *Legionella pneumophila* serogroup 1 in healthy residents of New York City, 10
Letters to the Editor
Ad agency conflict: selling cigarettes and dentistry, 469
Advances in the treatment of diabetes mellitus, 200
AIDS: a disease of ancient Egypt?, 200
AIDS and the alleged Haitian connection, 664
A large swallowed foreign body in the esophagus, 610
A program for rubella surveillance and control, 59
Attorney General Abrams speaks out against smokeless tobacco, 471

- Berwid case, 198
- Berwid case: author's reply, 198
- Breast self-examination, 664
- *Candida tropicalis* papillary necrosis treated with ketoconazole, 97
- Chronic tobacco use confounds the diagnosis of thyroid illness, 469
- Cigarette advertising in the Third World, 467
- Correction: Procainamide versus Procan SR®, 611
- *Death in the West* curriculum project, 470
- Disseminated histoplasmosis in a man with AIDS, 665
- Dr Stiles D. Ezell, 201
- Effects of present and past occupational exposures, 8
- Endolymphatic stromal myosis—a misconception, 242
- Esophageal ulceration associated with oral penicillin in Marfan's syndrome, 199
- Ethical double standard, 244
- Extra-adrenal myelolipoma, 60
- Fiscal and moral considerations of chronic brain syndrome, 9
- Glomus tumor of the clitoris, 611
- Hospital smoking policy, 464
- Humoral hypercalcemia of malignancy (pseudohyperparathyroidism), 242
- Impact of third-generation cephalosporins on clinical outcome of gram-negative meningitis in a municipal hospital, 243
- Inappropriate magazines for the waiting room, 465
- Medical approval for a driver's license, 486
- Nd:YAG laser photocoagulation, 59
- Neutropenia associated with procainamide, 197
- Offshore medical schools, 7
- Physician/participant in war, 663
- Placental pseudotumor of the uterus: an iatrogenic disease?, 663
- Porcelain gallbladder in a 24-year-old woman, 609
- Progress against smoking in Kuwait, 466
- Psychiatric approach to drug abuse, 96
- Recurrent malignant melanoma following a 46-year disease-free interval, 95
- Registry of health facilities that fail to provide clean indoor air, 465
- Repair of inguinal hernia with Marlex mesh, 486
- Roswell Park, 484
- Scalds of the lap, 485
- Searching for alternatives to war, 484
- Smoking prevalence rates for Western Australian adolescents, 470
- Solitary nonparasitic liver cysts, 95
- Spontaneous complete remission in acute promyelocytic leukemia, 662
- Spontaneous dissection of the right coronary artery, 485
- Sudan: physicians act against cigarette advertising, 467
- Symposium on violence, 197
- Thrombophlebitis: an unusual cause of chest pain, 609
- Tobacco industry and legislative redress, 468
- Tobacco related diseases among native Americans, 464
- Towards smoke-free comic strips, 472
- Use of *Journal* in schools, 465
- Wounds of war, 484

Leukemia
- Clinical significance of serum lactic dehydrogenase in chronic lymphocytic leukemia, 685
- Lactic dehydrogenase and malignancy (ED), 679
- Spontaneous complete remission in acute promyelocytic leukemia (LE), 662
- Spontaneous remission in acute nonlymphocytic leukemia (CR), 269

Leukemoid reaction
- Factitious hypoglycemia associated with eosinophilic leukemoid reaction (CR), 34

Linnaeus, Carl
- Linnaeus: right and wrong. On the 250th anniversary of his MD thesis and of his first edition of *Systema Naturae* (ED), 682

Lung disease
- A 40-year-old man with chills, fever, weight loss, and a productive cough (CPC), 694
- Disseminated cryptococcosis presenting as the adult respiratory distress syndrome (CR), 704
- Endobronchial foreign body (cigarette filter tip) with inflammatory pseudotumor of the lung. A "True" xanthogranuloma (CR), 389
- Farmer's lung in a 10-year-old boy (CR), 658
- Japan: land of the rise in lung cancer. US government aids cigarette sales, 425

Lymphangioma
- Retroperitoneal cystic lymphangioma (CR), 36

Marfan's syndrome
- Esophageal ulceration associated with oral penicillin in Marfan's syndrome (LE), 199

Mass Media
- Ad agency conflict: selling cigarettes and dentistry (LE), 469
- Attempt to circumvent the ban on cigarette advertising in Norway, 403
- Black doctors on television, 151
- Canada's oldest daily newspaper is country's first to drop cigarette advertising, 438
- Ending the social acceptability of smoking and tobacco advertising in New Zealand, 422
- Getting women hooked: defending the indefensible, 341
- *Helping Youth Decide:* "When the fox preaches, beware the geese," 357
- Inappropriate magazines for the waiting room (LE), 465
- Israel: gaining momentum against smoking and cigarette advertising, 416
- Mixed messages for women. A social history of cigarette smoking and advertising, 335
- Sham news and sports events exposed, 377
- Sudan: physicians act against cigarette advertising (LE), 467
- Superman and the Marlboro woman. The lungs of Lois Lane, 342
- Towards smoke-free comic strips (LE), 472

Maternal health
- Amniotic fluid embolism (CR), 265, (CR), 267
- Maternal and child health care: a deepening crisis, 128

Meconium aspiration
- The relationship of thrombocytopenia to the onset of persistent pulmonary hypertension of the newborn in the meconium aspiration syndrome, 245

Medical Directory Errata
- Medical Directory Errata, 50

Medical education
- Black-Jewish relationships. Access to health services and health careers, 137
- Inequality in the availability of black physicians, 139
- Institutional ethics and graduate medical education, 590
- Medical education in New York City in 1866–1867. A student's notebook of Professor Charles A. Budd's lectures on obstetrics at New York University—Part I (HIST), 488
- Medical education in New York City in 1866–1867. A student's notebook of Professor Charles A. Budd's lectures on obstetrics at New York University—Part II (HIST), 548
- Minorities and New York medical schools. Past failures and the prospect for future successes, 148
- Reflecting on the lectures of Professor Budd (ED), 529
- Restraint of progress. Declining participation of blacks and other minorities in medical schools, 135
- Underemphasis on smoking in medical education, 299

Medical Society of the State of New York
- The role of the Medical Society of the State of New York in workers' compensation, 24

Medical Society of the State of New York, House of Delegates
- Board of Trustees 1984–1985, 174
- Councilors 1984–1985, 174
- House Committee on Bylaws, 183
- House of Delegates, 171
- 1985 House of Delegates Members, 176
- Nominating Committee, 182
- Officers 1984–1985, 173
- Schedule of Sessions, 171

Melanoma
- Recurrent malignant melanoma following a 46-year disease-free interval (LE), 95

Membranoproliferative glomerulonephritis
- Membranoproliferative glomerulonephritis (ED), 571

Meningitis
- Impact of third-generation cephalosporins on clinical outcome of gram-negative meningitis in a municipal hospital (LE), 243

Merchant seamen
- Use of health services by merchant seamen, 14

Methotrexate
- Primary osteogenic sarcoma of the skull. Five-year disease-free survival following surgery and high dose methotrexate therapy (CR), 598

Minorities
(see also Black Americans)
- Black doctors on television, 151
- Black woman physician, 145
- Cigarette companies go to college, 349
- Cigarette smoking and ill health among black Americans, 344
- Comparison of selected health indicators for black and white children in New York State, 131
- Coronary heart disease in black Americans, 164
- Gastrointestinal malignancies in black Americans, 157
- Hypertension in blacks. Is it a different disease?, 160
- Legal and administrative barriers to health care, 126

Minorities and New York medical schools. Past failures and the prospect for future successes, 148
Racial equality in American health care system: an unfulfilled ideal, 125
Restraint of progress. Declining participation of blacks and other minorities in medical schools, 135
Sarcoidosis: focus on the black patient, 162
Status of Asian Americans in the health care delivery system in New York, 153

MOP-UP
MOP-UP battles tobacco sponsorship—legally, 436

MSSNY
MSSNY Liability Reform Fund Honor Roll, 708
MSSNY Malpractice Fund Honor Roll, 1b, 666

Myelolipoma
Extra-adrenal myelolipoma (LE), 60

Myocardial bridging
Pseudoinfarction pattern associated with myocardial bridging of the left anterior descending artery (CR), 510

Myocardial infarction
Cocaine-induced myocardial infarction (CR), 660

Nd:YAG laser photocoagulation
Nd:YAG laser photocoagulation (LE), 59

Neuropathy
Humoral abnormalities in three patients with idiopathic chronic polyneuropathy, 248

Neutropenia
Neutropenia associated with procainamide (LE), 197

Nominating Committee
Nominating Committee, 182

Nursing homes
Accidents in the skilled nursing facility, 202
Fiscal and moral considerations of chronic brain syndrome (LE), 9

Obituaries
Abbene, Michael M., 568
Abramson, Jacob, 232
Adler, Ernest Ludwig, 714
Adlerman, Edwin Jerome, 51
Alderman, Reuben Peter, 520
Alderson, Stanley Earl, 568
Alexander, John T., 51
Allan, William Brewster, 122
Allen, Farrow Robert, 274
Allen, Lloyd Fred, 520
Allerhand, Jona, 615
Alperin, Benjamin J., 122
Alsever, William Dewey, 232
Alterman, Robert H., 520
Amyot, Rudolph F., 714
Arnold, William D., 51
Badia, Mario Della, 520
Baker, G. Stanley, 568
Bakwin, Ruth M., 675
Bang, Edgar Philip, 615
Bannerman, Robin Mowat, 520
Barbosa-Saldivar, Jose Luis, 520
Bardavid, Jacques Jacob, 675
Barnat, Ivan, 520
Barsa, Joseph Albert, 232
Basirico, Cosimo, 274
Bates, Alfred Kelley, 568
Baumann, F. Albert, 675
Bellaire, Richard P., 615
Bergmann, Hans, 615
Berk, Alexei Nicolai, 568
Berlinghof, Clifton H., 51
Bernheim, Alice R., 568
Binder, Morris, 714
Birnkrant, William Benjamin, 122
Blinder, Samuel, 714
Bloch, Fritz Jakob, 274
Bloom, David, 615
Bloom, Jacob Maurice, 51
Blumenfeld, David B., 51, 84
Bodon, George R., 714
Booher, Robert Joseph, 520
Bornstein, Maurice, 568
Borota, Alexander, 615
Bott, Kenneth Francis, 274
Bradley, Bruce E., 274
Brandon, William D., 274
Brinsley, Bertram, 520
Brown, Daniel Noyes, 675
Bucci, James William, 714
Caggiano, Giovanni N., 274
Campbell, Carleton, 232
Carpati, Mark, 615
Cennerazzo, Ubaldo M., 568
Cerulli, Remo Romolo, 122
Chait, Robert Abbe, 51
Chang, Shin Ju, 520
Charters, Andrew James, 615
Chiappisi, Gus, 520
Chlenoff, Simeon O., 51
Chou, Tzu Pei, 122
Chusid, Erwin Leslie, 615
Cohen, Jacob Yonny, 568
Cohn, Harold, 675
Cole, John Gordon, 51
Comeau, Berthold R., 274
Comstock, Edwin Fisk, 274
Cooper, Henry S. F., 51
Counselman, Richard E., 84
Cracovaner, Arthur Joseph, 520
Crosby, John Philip, 568
Curth, R. William, 122
Dacks, George, 51
D'Arecca, John, 675
Davenport, Harold S., 232
DeBellis, Louis James, 675
DeCanio, Patrick Henry, 675
De Cardenas, Armando, 232
De Grandpre, Gerard C., 274
DeLuca, Louis Richard, 714
Dolan, William Russell, 714
Doucett, James A., 232
Eagan, Joseph Theodore, 51
Ellenberg, Max, 51
Epstein, Joseph, 274
Ertel, Harry, 122
Euphrat, Edwin Joaquin, 85
Evans, Jay I., 615
Falk, Emil Albert, 122
Farber, David, 615
Feder, Aaron, 520
Feinstein, Harold R., 122
Feld, Enid Lynn, 274
Feldman, Egon, 714
Ferrante, Frank Michael, 232
Fersko, Perry Michael, 274
Feuerstein, Benjamin Louis, 676
Fiderer, Solomon Salmen, 51
Fink, Edward Leonard, 675
Finklestein, Benjamin H., 122
Fischler, Julia, 714
Fishman, Morris, 122
Flack, J. Vincent, 232
Francis, Charles Collins, 714
Franklin, John Edward, 274
Freid, Jacob Robert, 520
Freireich, Abraham W., 232
Frischberg, Samuel Barnett, 675
Frohman, Albert, 615
Gaines, Thomas Patrick, 568
Gais, Elmer Stewart, 232
Ganong, Gilbert Edward, 568
Garson, Percy, 615
Genauer, Mortimer Benjamin, 714
Gershman, Maurice, 232
Giliberty, Richard P., 122
Ginsberg, Harry, 274
Gipner, John Frederick, 675
Glaser, Jerome, 568
Goldenberg, Morris H., 52
Goldsmith, Edward I., 714
Goodrich, Arthur L., 568
Gorsch, Rudolph Victor, 52
Grainer, Max, 232
Gray, James Henry, Jr., 52
Greeley, Arthur Vose, 275
Griffin, William Robert, 85
Grover, Herbert Warner, 85
Grunwald, Fred Seymour, 615
Guccione, Joseph, 52
Gunaratnam, Subramaniam, 521
Guzzetta, Anthony James, 615
Haas, Joseph, 232
Hall, Stanley Cheney, 675
Halpern, Iser Abraham, 675
Harman, Donald Davis, 615
Hart, Joseph Charles, 616
Hartmann, Jeno, 675
Hartnett, Bernard John, 616
Heilbrun, Norman, 275
Hemmett, Gordon M., 52
Henschel, Mervin Albert, 714
Hess, Alfred J., 616
Hillel, Henry, 52
Hite, K. Eileen, 122
Hollin, Sidney A., 85
Holt, Mary Brickett, 233
Houri, Rene, 122
James, Donald S., 122
Jenkins, Leo, 85
Jones, Kenneth, 233
Jurkowitz, Lawrence, 521
Kaiser, Murray L., 85
Kalenscher, Harry, 616
Karwowski, Walter J., 233
Katowicz, Leon, 233
Katz, Abraham, 85
Kauffman, Samuel H., 521
Keefe, William J., 52
Kenny, Francis E., 275
Kersten, Gerhard, 521
Kimberg, Morris, 52
Kim, In Hwan, 569
Kleban, Leo, 123
Klein, George, 676
Krauss, George G., 569
Kremer, Malvina Weinstock, 123
Krohn, Stuart E., 123
Kropf, William, 123
Kuehn, Hans, 52
Kuhlke, John E., 616
Kulka, Hans, 52
Kullman, Anne M., 275
Lacombe, Rene Pierre, 52
Lamberti, John J., 569
Lamstein, Jacob I., 715
Langsam, Martin, 569
Larkin, Vincent de Paul, 616
Leneman, Eli-Herz, 715
Lesnick, Harry J., 676
Levin, Thomas, 233
Lindauer, Russell George, 676
Little, Ethel E., 275
Liverman, Phillip C.L.Y., 233
Locke, John Joseph, 521
Lott, Bruce Davenport, 123
Lunsford, Charles Terrel, 521
Lynch, George Emanuel, 123
Maggiore, Michael J., 275

Mandel, Charles, 676
Marcus, Ernest, 521
Marmon, Martin Gary, 123
Mascola, Richard F., 52
Mathewson, Joseph Bryant, 52
Mazza, Luigi, 52
Mazzeo, Albert Anthony, 676
McDonald, Edward P., 52
McIlmoyl, Sherman William, 233
McLean, Preston Gamaliel, 521
McMahon, Robert Francis, 521
Meiers, Joseph, 521
Mellen, Harry John, 233
Mendelson, Milton, 52
Merlino, Anthony S., 521
Migliaccio, Arnold A., 569
Milazzo, Pasquale S., 676
Millen, Robert Stephen, 233
Moore, Jack, 275
Morris-Russell, Ethel C., 123
Needles, William, 86
Neumann, Ernst, 569
Newlands, William Alexander, 569
Nixdorf, Wallace Bortle, 676
Opin, Edward, 715
Ozlu, Cavit, 521
Palais, Isidor, 233
Panebianco, Richard R., 521
Panvini, Vincent J., 616
Panzarella, Joseph J., 123
Papanek, Helene, 616
Parise, Nicholas F., 275
Parker, Jesse Silsby, 275
Parlow, Allen Laurence, 569
Patella, Armand Major, 52
Patterson, Howard A., 569
Peimer, Ralph, 715
Peller, Sigismund, 676
Pelzman, Oscar, 123
Perrotta, Louis, 569
Peterfy, Albert B., 521
Petronio, Arthur A., 521
Pincus, Joseph Aaron, 123
Poetsch, Heinz J., 521
Polisar, Ira Allan, 275
Post, Bernard Saul, 275
Powalski, Robert J., 616
Price, George Francis, 123
Priest, Robert S., 676
Pschierer, Francis M. J., 569
Pulsifer, Libby, 233
Purdy, Anna Earl, 616
Rakieten, Morris Louis, 86
Ranallo, John J., Jr., 676
Rapoport, Paul, 616
Ratner, Irvine M., 616
Ravich, Abraham, 569
Reason, Clifford H. J., 123
Reichert, Philip, 521
Reich, George Aloysius, 123
Reid, Edward Kessinger, 715
Richman, Alfred Abraham, 123
Richman, George, 569
Rivkin, Hyman, 569
Rizzuti, Geremia, 52
Robbins, Abner R., 233
Romano, Domenico L., 52
Romm, Barnet Irving, 275
Rosenberg, Daniel W., 715
Rosenberg, Milton, 521
Rosenfeld, Livia Halasz, 53
Rosenhain, Felix R., 86
Rosenow, George, 616
Rothballer, Alan Burns, 124
Rubinow, Mitchell Jay, 53
Rubinstein, Joseph E., 715
Rubler, Herman B., 124
Rudnick, Julius, 233
Rust, Nelson Hopson, 616
Rutenberg, Joseph, 124
Rutzler, Henry Lupton, 616
Sabatini, William Arthur, 715
Sack, S., Sr., 569
Safirstein, Samuel Leib, 522
Safran, Morton Morris, 522
Saitta, Peter Michael, 676
Salomon, Mardoqueo I., 124
Sanders, Martin, 522
Santora, Edward, 569
Santy, Albert Christy, 676
Schein, Hans, 616
Schenk, Max, 522
Scherma, Angelo S., 715
Schiano, Alfredo, 616
Schiff, Sydney Paul, 676
Schittone, Marilyn T., 124
Schwalben, Seymour, 275
Schwartz, A. Albert, 124
Schwartzfarb, Morris B., 676
Schwartz, Milton J., 569
Schweitzer, Harold T., 124
Sebesta, Joseph F., Jr., 616
Seides, Jerome M., 53
Selinger, Avram N., 53
Shaffer, Charles Alfred, 275
Shapiro, David Raymond, 53
Shea, Robert E., 616
Sherlock, Paul, 617
Shnayerson, Ned, 275
Shornstein, Charles, 676
Sicat, Jose C., 275
Silberman, Isidor, 676
Sills, Joseph Robert, 617
Silver, Aaron, 677
Silverberg, Sigmund B., 522
Simon, Norman, 522
Sinberg, Samuel Edward, 233
Singson, Calixto C., 124
Skok, Paul, 569
Slater, Ralph, 570
Slo-Bodkin, Samuel Garson, 276
Smilay, Malcolm Goff, 677
Smith, Bernard Hooper, 276
Smith, Dean Davidge, 53
Smith, Herbert John, 715
Snyder, Raymond Dimmick, 570
Sohval, Arthur Robert, 233
Solis, Mario H., 276
Soloway, David, 233
Spivak, Jack, 87
Squillace, Patricia Gill, 570
Stebbins, Edward, 124
Steckel, Sidney Solomon, 677
Steele, Paul Dewey, 276
Stein, Leonard, 522
Steinthal, Erich F. A., 522
Stephenson, James, 617
Sternberg, Otto, 617
Stern, Frederick, 715
Stohr, Grete, 124
Stoloff, Emile G., 677
Stoner, Lee Rowland, 677
Stovin, Joseph Samuel, 124
Strauss, Lotte, 677
Stroll, George Morris, 677
Sullivan, Richard Edward, 276
Sullivan, Thomas F., 677
Swick, Moses, 677
Szabo, Gustav, 276
Szabo, Nicholas Aladar B., 570
Szuldberg, Bronislawa, 124
Tabacco, Robert James, 522
Tamis, Abraham Bernard, 715
Tatelbaum, Abraham Joseph, 677
Taylor, Howard C., 522
Thompson, Howard Thomas, 677
Thompson, Samuel Alcott, 522
Tiangco, Herminio Cruze, 124
Turel, Solomon J., 124
Vaisberg, Maurice, 617
Van Doren, James D., 53
Van Orden, Philip S., 124
Veer, Albert Vander, 570
Velardi, Francis G., 87
Verme, John Lo, 123
Vigdor, Arthur, 276
Vinci, Guido, 715
Violyn, Roman R., 522
Vitanza, Peter P., 276
Vogel, Samuel A., 570
Vrooman, David H., 87
Wahl, Orin A., 124
Walker, Herbert Earl, 570
Watton, Walter F., 276
Weile, Fritz, 522
Weinstein, Theodore R., 87
Weishaar, Leo G., 53
Welsh, Catherine A., 87
Werdein, Edward J., 124
Weston, Lanpher Chas, 53
West, Robert, 570
Wetchler, Bernard Bruce, 677
Whitney, Chester H., 276
Widder, Felix, 570
Wolfe, Louis A., 677
Woodford, Norman L., 570
Worcester, Blandina V., 276
Yolles, Tamarath Knigin, 677
Young, Dennison, 522
Zaia, Catherine M., 276
Zak, George Albert, 53
Zaldin, Samuel, 276
Zeifman, Israel, 677
Zimet, Ralph Reuben, 617
Zohman, Burton L., 522

Occlusive cerebrovascular disease
  Neuro-ophthalmologic aspects of occlusive cerebrovascular disease, 18
Occupational exposures
  Effects of present and past occupational exposures (LE), 8
Officers
  Officers 1984–1985, 173
Offshore medical schools
  Institutional ethics and graduate medical education, 590
  Offshore medical schools (LE), 7
Organized medicine
  Strengths of organized medicine (ED), 576
Osteogenic sarcoma
  A 28-year-old man with a left-sided palatal mass (CPC), 585
  Primary osteogenic sarcoma of the skull. Five-year disease-free survival following surgery and high dose methotrexate therapy (CR), 598
Ovulation
  Use of gonadotropins in ovulation induction. Analysis of treatment protocols and pregnancy outcome, 580
Parathyroid
  Humoral hypercalcemia of malignancy (pseudohyperparathyroidism) (LE), 242
  Humoral hypercalcemia of malignancy. The role of parathyroid hormone (ED), 235
  Intrathymic parathyroid adenoma in a child (CR), 263
  Parathyroid adenoma in children (ED), 237
Periosteal reaction
  A pelvic mass with periosteal reaction (RPC), 106
Placenta
  Placental pseudotumor of the uterus: an iatrogenic disease? (LE), 663

Uterine tumor due to placenta increta (CR), 512
Placenta increta
Uterine tumor due to placenta increta (CR), 512
Pleural effusion
Pleural effusion due to communicating gastric ulcer (CR), 706
Pleural thickening
Effects of present and past occupational exposures (LE), 8
Pneumococcal inguinal adenitis
Suppurative pneumococcal inguinal adenitis (CR), 509
Pneumocystis carinii
A 39-year-old man with thrombocytopenia, cough, fever, and shaking chills (CPC), 27
Pneumonia
A 74-year-old man with a lung mass, arthritis, and difficulty walking (CPC), 647
A 40-year-old man with chills, fever, weight loss, and a productive cough (CPC), 694
Disseminated cryptococcosis presenting as the adult respiratory distress syndrome (CR), 704
Pneumonitis
Farmer's lung in a 10-year-old boy (CR), 658
Porcelain gallbladder
Porcelain gallbladder in a 24-year-old woman (LE), 609
Poultice
The poultice (HIST), 207
Poverty
Comparison of selected health indicators for black and white children in New York State, 131
Maternal and child health care: a deepening crisis, 128
Pregnancy
Multiple pregnancies. Spontaneous and induced (ED), 525
Perinatal aspects of high multiple gestation (CR), 560
Pregnancy obtained by in vitro fertilization in an ambulatory-care surgical facility (CR), 654
Ultrasound diagnosis of interstitial pregnancy (CR), 655
Prehospital care
Emergency medical services system in New York State: time for a change (ED), 1
Procainamide
Correction: Procainamide versus Procan SR® (LE), 611
Neutropenia associated with procainamide (LE), 197
Product liability
Cigarette marketing: ethical conservatism or corporate violence?, 324
Cipollone v Liggett Group, Inc. Application of theories of liability in current cigarette litigation, 318
Product liability in cigarette-caused fires, 322
Tobacco products liability project, 390
Psychiatric institutions
Violence in psychiatric institutions, 64
Psychiatry
Psychiatric approach to drug abuse (LE), 96
Psychiatry and the prevention of violence (ED), 57
Public Health Service
Use of health services by merchant seamen, 14
Racial equality
Black-Jewish relationships. Access to health services and health careers, 137
Eugene Percy Roberts: black physician and leader, 143
Inequality in the availability of black physicians, 139
Racial equality in American health care system: an unfulfilled ideal, 125
Referees
Our referees, 42
Renal transplants
Cyclosporine: preliminary experience in 79 patients with renal transplants, 531
Immunosuppression for renal allograft recipients: The role of cyclosporine (ED), 523
Increased survival time of renal allograft recipients, 534
Respiratory disease
(see also Lung diseases)
Rhabdomyosarcoma
A pelvic mass with periosteal reaction (RPC), 106
Rosenberg, Milton, MD
Milton Rosenberg, MD, 1923–1985, 121
Rubella
A program for rubella surveillance and control (LE), 59
Sarcoidosis
Sarcoidosis: focus on the black patient, 162
Sarcoidosis of the breast (CR), 262
Sarcoidosis revisited (ED), 240
Schedule of Sessions
Schedule of Sessions, 171
Serous ovarian neoplasms
Amylase in serous ovarian neoplasms (CR), 113
Shock
A 77-year-old woman with giant-cell arteritis, back pain, and shock (CPC), 502
Hemodynamic monitoring of the patient in shock (ED), 238
Small intestine perforation
Asymptomatic perforation of the small intestine by a Copper-7 intrauterine device (CR), 515
Smoking
Ad agency conflict: selling cigarettes and dentistry (LE), 469
American Nurses' Association passes resolutions for action on smoking issues, 444
An insurance company perspective on smoking, 307
Assessment of the effectiveness of nicotine chewing gum: unfulfilled expectations, 378
Attempt to circumvent the ban on cigarette advertising in Norway, 403
Attorney General Abrams speaks out against smokeless tobacco (LE), 471
Automobile racing: slaughter on tobacco road, 458
Basics of beating the tobacco industry, 372
BUGA-UPdate, 434
Canada's oldest daily newspaper is country's first to drop cigarette advertising, 438
Canadian Non-Smokers' Rights Association: shaming the establishment, 439
Changes in overwrap and butt length of American filter cigarettes. An influence on reported tar yields, 310
Chronic tobacco use confounds the diagnosis of thyroid illness (LE), 469
Cigarette advertising in the Third World (LE), 467
Cigarette companies go to college, 349
Cigarette marketing: ethical conservatism or corporate violence?, 324
Cigarette smoking and ill health among black Americans, 344
Cigarette smoking in Papua New Guinea. A model for keeping a people down, 420
Cipollone v Liggett Group, Inc. Application of theories of liability in current cigarette litigation, 318
Clinician and smoking in Great Britain, 391
Competing agenda in smoking control agencies. "*Those who pay the piper...*" (ED), 287
Comprehensive Smoking Education Act, 363
Conferences on smoking, 412
Death in the West curriculum project (LE), 470
Disease model of cigarette use, 294
Diversification in the tobacco industry, 328
Economic dependence on tobacco manufacturing in Northern Ireland. An obstacle to prevention, 396
Elected officials must also bear responsibility, 393
Ending the social acceptability of smoking and tobacco advertising in New Zealand, 422
Endobronchial foreign body (cigarette filter tip) with inflammatory pseudotumor of the lung. A "True" xanthogranuloma (CR), 389
Follow-up of a cover-up, 285
Getting women hooked: defending the indefensible, 341
Glossary of tobacco terms, 297
Health claims made for filter cigarettes, 317
Helping Youth Decide: "When the fox preaches, beware the geese", 357
Hospital smoking policy (LE), 464
How smoking promotes hunger, 442
How tobacco companies have found religion, 445
If smoking killed baby seals... (ED), 282
Inappropriate magazines for the waiting room (LE), 465
In China, cigarettes are big business, 424
Indoor air quality standard for ambient tobacco smoke based on carcinogenic risk, 381
Innovation in Iceland: graphic health warnings on tobacco products, 405
Insurance incentives for not smoking, 309
Israel: gaining momentum against smoking and cigarette advertising, 416
Japan: land of the rise in lung cancer. US government aids cigarette sales, 425
Last cigarette advertisement, 359
"Less hazardous" cigarette: a deadly delusion, 313
Local values versus tobacco industry power. The experience of GASP of Massachusetts, 375
Loss of jobs due to *not* smoking, 323
Maverick doctor-politician puts pillory on tobacco and alcohol companies, 428
Medical journal editor stuns and angers health campaigners, 432
Medical students take action outside children's hospital, 431
Mixed messages for women. A social history of cigarette smoking and advertising, 335
MOP-UP battles tobacco

sponsorship—legally, 436
New Jersey's hard-fought battle for clean indoor air legislation. An insider's view, 366
Obstacles to the prevention of teenage smoking, 360
Perspectives on the economic magnitude of cigarette smoking, 302
Pharmacists who dispense cigarettes. With reference to drug store chains and pharmaceutical companies, 350
Physicians, smoking, and health in the Netherlands, 394
Pilot's guide to cabin air quality and fire safety, 384
Product liability in cigarette-caused fires, 322
Progress against smoking in Kuwait (LE), 466
Proposition P: anatomy of a nonsmokers' rights ordinance, 369
Registry of health facilities that fail to provide clean indoor air (LE), 465
Saving or smoking, 306
Scottish epidemic, 399
Sham news and sports events exposed, 377
Slow but steady progress against smoking in Ireland, 400
Smoke-free society by the year 2000 (ED), 290
Smoking and health in the Soviet Union and Eastern Europe, 413
Smoking and its promotion in Greece. A 20th century Trojan horse, 409
Smoking and the military, 354
Smoking control in Sweden, 404
Smoking in France: a dismal picture, 407
Smoking prevalence rates for Western Australian adolescents (LE), 470
Sudan: physicians act against cigarette advertising (LE), 467
Suffolk's successful clean indoor air act, 368
Superman and the Marlboro woman. The lungs of Lois Lane, 342
Tobacco dilemma intensifying in North Carolina, 455
Tobacco industry and legislative redress (LE), 468
Tobacco products liability project, 390
Tobacco related diseases among native Americans (LE), 464
Towards smoke-free comic strips (LE), 472
Underemphasis on smoking in medical education, 299
Use of *Journal* in schools (LE), 465
Western Australia gives health promotion the nod, 429
When researchers accept funding from the tobacco industry, do ethics go up in smoke?, 451

Sonography
(see also Ultrasonography)
Need for routine sonography prior to late abortion (ED), 4

Sterilization
Tubal sterilization with the Falope ring in an ambulatory-care surgical facility, 98

Sternoclavicular joint
Posterior dislocation of the sternoclavicular joint (CR), 225

Technology
Future patterns of chronic disease incidence, disability, and mortality among the elderly. Implications for the demand for acute and long-term health care, 623
Health care payment reform. Projections for next 15 years, 644
Identifying cost effective diagnostic strategies (ED), 680
Implications of changes in payment practices for health care. The insurer's perspective, 641
Implications of new medical technology, 634
Laboratory ordering: Bridging the gap (ED), 693
Reflections on health care at the coming turn of the century, 619
The impact of technology on laboratory ordering strategies, 690
The roles of new medical technology and health care delivery systems in shaping medicine into the 21st century, 637

Thalassemia
Cardiac siderosis in thalassemia major (CR), 513

Thoracotomy
Massive hemoptysis due to retained rib fragment 40 years after thoracotomy (CR), 605

Thrombocytopenia
A 39-year-old man with thrombocytopenia, cough, fever, and shaking chills (CPC), 27
Management of idiopathic thrombocytopenic purpura in childhood, 499
The relationship of thrombocytopenia to the onset of persistent pulmonary hypertension of the newborn in the meconium aspiration syndrome, 245

Thrombophlebitis
Thrombophlebitis: an unusual cause of chest pain (LE), 609

Thyroid disease
Chronic tobacco use confounds the diagnosis of thyroid illness (LE), 469

Toxoplasmosis
A 39-year-old man with thrombocytopenia, cough, fever, and shaking chills (CPC), 27
Disseminated toxoplasmosis with signs and symptoms of a cerebral lesion (CR), 40

Tubal sterilization
Tubal sterilization with the Falope ring in an ambulatory-care surgical facility, 98

Tuberculosis
Intestinal fistula formation in a man with mycobacterial disease and AIDS (CR), 702

Tumors
A 28-year-old man with a left-sided palatal mass (CPC), 585
Clinical and pathologic features of endolymphatic stromal myosis (ED), 237
Disseminated toxoplasmosis with signs and symptoms of a cerebral lesion (CR), 40
Epidermoid cysts of the cranium (CR), 596
Glomus tumor of the clitoris (LE), 611
Leiomyosarcoma of the ovary (CR), 218
Long-term survival with metastatic uterine sarcoma (ED), 195
Metastatic leiomyosarcoma of iliac bone (CR), 220
Placental pseudotumor of the uterus: an iatrogenic disease? (LE), 663
Primary osteogenic sarcoma of the skull. Five-year disease-free survival following surgery and high dose methotrexate therapy (CR), 598
Stage II benign cystic teratoma with malignant squamous degeneration (CR), 224
Uterine tumor due to placenta increta (CR), 512

Ulcer
Pleural effusion due to communicating gastric ulcer (CR), 706

Ultrasonography
Application of non-invasive diagnostic techniques to the pediatric patient (ED), 528
Need for routine sonography prior to late abortion (ED), 4
Neonatal hydrops of the gallbladder. Evaluation by cholescintigraphy and ultrasonography (CR), 565
Ultrasound diagnosis of interstitial pregnancy (CR), 655

Ureteropyeloscopy
Rigid ureteropyeloscopy, 205
Ureteropyeloscopy (ED), 193

Urinary tract
Transurethral endoscopy of upper urinary tract (ED), 194
Ureteropyeloscopy (ED), 193

Violence
Berwid case (LE), 198
Berwid case: author's reply (LE), 198
How the police view violence, 72
Obligation to assess dangerousness, 67
Physician/participant in war (LE), 663
Preventing alcohol-related violence: the physician's role (ED), 56
Psychiatry and the prevention of violence (ED), 57
Searching for alternatives to war (ED), 55, (LE), 484
Symposium on violence (LE), 197
The physician and war. A layman's view (HIST), 77
Violence in psychiatric institutions, 64
Violence in the Third World, 74
Violence on the highway. A national public health problem, 69
Wounds of war (LE), 484
Wounds of war. With particular reference to 1861–1865, 61

Visual defects
Neuro-ophthalmologic aspects of occlusive cerebrovascular disease, 18

In vitro fertilization
Pregnancy obtained by in vitro fertilization in an ambulatory-care surgical facility (CR), 654

War
Physician/participant in war (LE), 663
Searching for alternatives to war (ED), 55, (LE), 484
Symposium on violence (LE), 197
The physician and war. A layman's view (HIST), 77
Wounds of war (LE), 484
Wounds of war. With particular reference to 1861–1865, 61

Workers' compensation
The role of the Medical Society of the State of New York in workers' compensation, 24

Xanthogranuloma
Endobronchial foreign body (cigarette filter tip) with inflammatory pseudotumor of the lung. A "True" xanthogranuloma (CR), 389

AUTHOR INDEX

Ablin, Richard J., 200
Abrams, Robert, 471
Adler, Leon, 38
Adriaanse, Hans, 394
Aftalion, Belinde, 539
Ahmed, Pervez, 267
Al-Awadi, Abdul Rahman, 466
Al-Mumen, Hussain A., 466
Alpern, Harlan D., 502, 647
Alpert, Morris, 7
Amodio, John B., 565
Anderson, Gerard F., 644
Antignano, Louis V., 197
Arden, Sheldon V., 665

Back, Florence, 654
Badawy, Shawky, 663
Badawy, Shawky Z. A., 512
Bagley, Demetrius H., 193
Baim, Roger, 515
Baker, Daniel G., 92
Baker, Stephen R., 609
Ball, Keith, 467
Balter, Richard, 694
Baptist, Selwyn J., 665
Barold, S. Serge, 197
Bartolomeo, Robert S., 199
Bases, Robert, 465
Bates, Margaret E., 265
Batra, Anil K., 389
Bellin, Lowell B., 619
Bennett, Boyce, 106
Berezin, Stuart, 227
Berkowitz, Richard L., 4
Bernstein, Martin, 389
Bernstein, Richard K., 200
Betzu, Robert, 660
Bevelaqua, Frederick A., 605
Bhargava, A., 685
Bhole, Anita, 227
Bittoun, Renée, 407
Blake, Gregory H., 354
Blinderman, Abraham, 77
Blöndal, Thorsteinn, 405
Bloom, M. L., 685
Bloom, Michael J., 97
Blum, Alan, 121, 282, 328, 350, 360, 445, 458
Blume, Sheila B., 56
Boros, Laszlo, 197, 611
Bowers, Charles, 4
Boyd, Arthur D., 605
Brandstetter, Robert D., 706
Brook, David W., 96
Brown, Jay, 164
Bruce-Chwatt, L. J., 682
Buchbinder, Shalom, 609
Budman, Daniel R., 601
Bussel, James B., 499
Byer, Barbara J., 534

Calcagno, David, 577
Califano, Joseph A., Jr., 360
Cantor, Susan, 694
Canver, Charles C., 101, 604
Carlson, Regina, 465
Carrasco, Noel J. M., 245
Chapman, Simon, 287
Chattopadhyay, T. K., 610
Chattopadhyay, Tushar K., 486
Cherubin, Charles E., 243
Chervenak, Frank A., 4
Chester, Douglas Lee, 469
Cho, Sam, 112
Clair, Michael, 694
Coady, Deborah J., 655
Cohen, Morris, 565
Cohen, Paul S., 97
Cohen, Robert, 64
Conn, Rex B., 680
Cooper, Richard, 344, 413
Costanza, Michael C., 480
Coulson, W. R., 357
Cowell, Michael J., 307
Coyle, P. K., 248
Craig, Thomas J., 57
Crocco, John A., 573
Cruz, Gladys, 599
Cunningham, Allan S., 658
Cush, John J., 110

Daniele, Ronald P., 162
Darrow, Mark, 647
Das, Sukanto K., 36
Davis, John S., 502
Davison, Edward T., 510
DeJunco, Joseph E., 485
Derby, Bennett M., 485
deR Kolisch, Paul, 484
Diamond, Herbert, 539
Dickerson, Dennis C., 143
Diem, Gail, 479
Dimant, Jacob, 202
DiTroia, Daniel J., 265
Dorfman, Sally Faith, 4
Douglas, Gordon W., 529
Drop, Maria J., 394
Duffell, G. Michael, 240
Duncanson, F., 702
Dutel, Marie, 665
Dwek, Joe H., 609
Dwyer, Patrick N., 647

Edell, Marc Z., 318
Edelman, Marian Wright, 128
Ehrenkranz, Richard A., 4
Eisenberg, Harvey, 704
Emrich, H., 685
Enck, Robert E., 662
Eng, Robert H. K., 243
Engebretsen, Bery, 55
Ernster, Virginia L., 335

Faierman, Daniel, 609
Fairbanks, Leland L., 464
Fani, Kazem, 40
Feldman, Joseph, 378, 482
Feldman, Joseph G., 693
Fink, Jordan N., 658
Fischbein, Alf, 8
Fischer, Paul, 465
Fisher, F. Craig, 484
Fitzgerald, Kathleen, 445
Fitzpatrick, J., 685
Fletcher, Brent, 512
Floering, David, 692
Flynn, John T., 27, 585, 694
Fontanetta, Eileen, 565
Foster, Roger S., Jr., 480
Fresko, Oya, 267
Friedman, Sandor A., 110
Fromowitz, Frank, 515
Fulton, H. B., Jr., 384
Furlong, Regina, 4

Gajera, R., 685
Galicich, Joseph H., 598
Garcia, Roger L., 37
Garner, Donald W., 322
Geary, Joseph E., 115
Gellhorn, Alfred, 590
Gerst, Paul H., 263
Gill, Liveleen M., 269
Gillooley, John F., 599
Gindoff, Paul R., 580
Ginzel, K. H., 299
Gisser, Stewart M., 318
Glantz, Stanton A., 470
Glaser, Gerald L., 9
Glichlick, D., 531
Godec, Ciril J., 222
Gogel, Edward, 694
Goldfarb, Alisan B., 95
Goldstein, Steven R., 655
Gomez, G. A., 685
Gonder, Maurice J., 200
Goodwin, Norma J., 145
Gopalaswamy, Chitra, 660
Gould, Lawrence, 660
Gould, Perry C., 199
Green, David, 486
Grunberg, Neil E., 310
Gutherz, Philip, 706

Hafiz, Mohammad Abdul, 220
Haley, N. J., 469
Halfens, Ruud, 394
Haller, John S., 484
Haller, John S., Jr., 207, 257
Hamby, Robert I., 485, 510
Han, Tin, 685
Hanauer, Peter, 369
Hannigan, Edward V., 195
Hansard, Paul, 238
Hansen, Bert, 488, 548
Harris, Bruce, 647
Hausner, Amos, 468
Henderson, M. L., 685
Henry, Mark C., 1
Herdman, Roger C., 634
Herman, Martin A., 366
Hilgenberg, Alan D., 527
Hirschman, Richard J., 109
Hirsh, David, 539
Hobbins, John C., 4
Hoffman, Gary, 502
Holder, Angela R., 4
Horgan, Michael J., 245
Horner, Richard J., 647
Hotchkiss, Edward J., 539
Howe, Suzanne, 59
Hoy, Wendy E., 534
Huffman, Jeffry L., 194
Huvos, Andrew G., 598

Immerman, Ronald S., 200
Imparato, Anthony M., 197
Imperato, Pascal James, 7, 637
Isenberg, Henry D., 539

Jagadha, V., 60, 596, 611
Jagadha, Venkita, 113
Jewelewicz, Raphael, 580
Jimenez, Fidelio A., 40
Johnson, Leon, Jr., 135

Kaali, Steven G., 98, 654
Kahn, Henry S., 472
Kahn, Leonard B., 539
Kapoor, V. K., 610
Kapoor, Vinay K., 36, 486
Katz, Steven E., 64
Kavaler, Florence, 14
Kearns, Richard J., 225
Keenan, Elizabeth, 409
Keller, Steven M., 95
Kelman, Howard R., 14
Kent, Deirdre, 422
Khuri, Fuad J., 205
Kimmel, David P., 197
Kinas, Helen Y., 37
King, Sharon B., 641
Kjønstad, Asbjørn, 403
Klass, Stephen C., 706
Klein, Robert M., 225
Knutson, David W., 534
Kolanchik, Gary J., 97
Koop, C. Everett, 290
Kozak, Alan J., 502
Kozlowski, Lynn T., 310
Kreipe, Richard E., 214
Kresky, Beatrice, 1
Kriplani, A. K., 610
Kuzminski, Antoinette M., 502

Landesman, Robert, 98, 525, 654
Lane, Donald J., 391
Lawrence, George J., Jr., 12, 576
Lebowitz, Arthur S., 509
Lee, Stanley L., 244
Lefor, Alan T., 34
Lehmann, Craig A., 692
Lehrman, Nathaniel S., 198
Leibowitz, Ian H., 110
Leiken, Alan M., 692
Lemkin, Joyce A., 115
Leon, Lucina, 665
Leonidas, Jean-Robert, 664
Levy, Julio, 263
Lichtman, Stuart M., 601
Lief, Beth J., 126
Liss, Arthur, 38
Little, Thomas, 585
Lore, John, 585
Lowen, MaryAnn, 694
Lowrey, Alfred H., 381
Luft, Benjamin, 694
Lugo, Emma, 37
Lyubsky, Sergey, 694

Maayan, Shlomo, 243, 704
MacDonald, J. Fred, 151
Madeley, John, 442
Magnus, Paul, 342
Magnússon, Gudjon, 405
Malfetano, John H., 242
Mangiacasale, Angela, 438, 439

Mann, William J., 515
Mannix, Henry, Jr., 237
Manton, Kenneth G., 623
Matas, A. J., 531
May, Allyn G., 534
May, John J., 393, 658
Maycock, Virginia A., 310
McBride, A. D., 125
McBride, David, 139
McBride, William, 157
McCormack, James J., 148
McCray, Richard S., 59
McGarvey, Michael R., 641
Meggs, Leonard G., 160
Merrill, Nancy, 502
Miller, Calvin, 428, 429, 431,
432, 434, 436
Miller, Frederick, 248
Miller, G. H., 313
Miller, Myron, 34
Miller, Steven N., 704
Millian, Stephen J., 10
Mir, Rabia, 539
Mishra, M. C., 610
Mishra, Mahesh C., 486
Montana, John B., 665
Montes, Mario, 585
Morehouse, Helen, 563
Morrissey, James M., 210
Morse, David E., 310
Morton, John H., 24
Mukhopadhyay, Ashok K., 36
Murphy, Patrick V., 72
Murray, Kathyrn I., 59
Musher, Daniel M., 574

Neglia, Walter, 706
Nelson, Stuart, 694
Newland, J. R., 237
Newman, Leonard J., 227
Nishi, Setsuko Matsunaga, 153
Nobler, Myron P., 109
Norstrand, Iris Fletcher, 40
North, Charles, 464

O'Gorman, Kevin M., 664
Olson, John P., 197
O'Sullivan, Sister Helene, 74
Ozer, H., 685

Pai, B. Narayan, 263
Panchacharam, P., 611
Papier, Cheri M., 416
Parsons, William J., 564
Patanaphan, Vinita, 220
Peartree, Robert J., 205
Peress, Nancy, 248
Perla, Elliott N., 704
Perry, Malcolm O., 577
Peter, Sebastian A., 242
Pillari, George, 38
Pinals, Donald J., 706
Pitchumoni, C. S., 702
Poon, T., 596
Poon, Tung Pui, 218
Poshni, Iqbal A., 10
Prabakaran, J., 702
Preisler, Harvey D., 269
Prior, John T., 6

Quinn, T., 531

Rakowski, Irene, 269
Ramaswamy, G., 60
Ramaswamy, Gita, 113, 218, 704
Randolph, Linda A., 131
Rapoza, Catherine R., 644
Ratz, Otto, 654
Raza, Azra, 269
Reddy, Sudhakar A., 218
Reese, P. A., 685
Reese, Richard, 502
Reich, Lloyd, 389
Reitz, Mary Ellen, 262
Renzin, Stephen, 654
Repace, James L., 381
Rice, Dan, 222
Richards, John W., Jr., 350
Richter, Gary C., 157
Ridgway, Derry, 679
Risemberg, Herman, 245
Rivers, Sylvia, 131
Robinson, Arvin E., 528
Rochelson, Burton L., 515
Rose, David P., 479
Rosen, Gerald, 598
Rosenkranz, Leon, 95
Rosenthal, Ronald E., 663
Rosenzweig, Murray J., 565
Roses, Daniel F., 262
Rothenberg, Robert E., 484
Rudavsky, Amiel Z., 565
Ruotolo, Ronald A., 205

Salazar, Omar M., 220
Saliba, Nicolas K., 242
Santos, Gil Hauer, 563
Saxena, Brij B., 525
Scally, Gabriel J., 396
Scheer, Robert L., 97
Schroeder, Conrad, 95
Schulman, Philip, 601
Schutzer, Steven E., 248
Schwartz, Daniel W., 67, 198
Scrimgeour, Euan M., 420
Seidman, Irving, 262
Seltzer, Vicki, 224
Selvaraj, S., 702
Sepkovic, D. W., 469
Shainess, Natalie, 664
Shapiro, Donald L., 560
Shapiro, Joseph, 539
Sharma, L. K., 610
Sharma, Lalit K., 36, 486
Sharp, Charles F., Jr., 235
Sharpe, William D., 61
Shean, Ruth E., 470
Shelley, Emer B., 400
Sher, Joanna Hollenberg, 40
Sherman, Lawrence M., 93
Shreedhar, Rakesh, 263
Shultz, James M., 302
Silane, Michael F., 577
Sills, Irene N., 251
Silverman, Andrew Y., 654
Simmons, Brian E., 344
Simon, Thomas, 512
Singhal, Hemant, 610
Sklarek, Howard M., 199
Slade, John D., 294
Smith, Sharon M., 243
Snyder, Jon R., 655
Soberman, R., 531
Sobo, Steven, 38
Sokal, David C., 467
Spohn, Lawrence, 455
Spradlin, Susan, 577
Srinivasan, K., 611
States, John D., 69
Stein, Richard A., 238
Steinberg, Harry, 539
Sterman, A. B., 248
Stern, Peter H., 201
Stokman, Cornelius L. J., 64
Stolfi, Julius E., 7
Subramanyan, Bala R., 655
Sundaresan, Narayan, 598
Sutherland, David E. R., 523
Svahn, David, 502, 647
Sweda, Edward L., Jr., 375
Szebenyi, Steven E., 502

Tafreshi, Michael, 38
Tash, Robert, 225
Tchertkoff, Victor, 113, 218
Tejani, Nergesh A., 265
Tellis, V. A., 531
Teng, Pin Kie, 112
Tierno, Philip M., Jr., 509
Traube, Charles, 267
Tucker, James B., 97
Tye, Joe B., 324

Upson, James F., 604
Urizar, Rodrigo E., 571

Vafai, Jalleh, 560
Valvo, John R., 205
Van Reek, Jan, 394
Vaules, David W., 502
Vega, Manuel, 485
Veith, F. J., 531
Verma, Uma L., 265
Vianna, Nicholas J., 91
Visconti, Ernest B., 59
Vogl, Steve, 224

Walts, Ann E., 191
Wang, Charles Pei, 153
Watts, Robert K., 148
Waxman, Henry A., 363
Waxman, Marian, 599, 663
Weingarten, Marvin J., 225
Weisburger, John H., 103
Weiss, R., 531
Welch, Ethan L., 115
Westerink, Maria Julia, 585
Wiechec, David M., 585
Wieczorek, Rosemary, 605
Wilson, Elizabeth, 444
Winkler, William P., 59
Wolfe, Samuel, 137
Wolinsky, Howard, 451
Wolintz, Arthur H., 18
Worden, John K., 480
Wright, Peter T., 502, 647
Wroblewski, Michael, 309
Wu, Richard H. K., 263
Wu, Tzee Chung, 227
Wynder, E. L., 469

Yacono, John V., 655
Yoffa, Jack, 512
Younger, Steven, 222

Zaino, Edward C., 513
Zisbrod, Zvi, 95

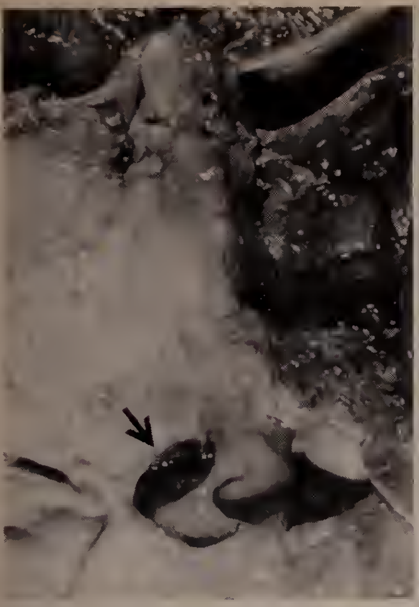


FIGURE 2. Partial separation of the posterior wall of the stomach from adhesion to diaphragm to reveal perforating ulcer with fistulous stoma (arrow). The unopened esophagus lies along the upper right margin. The opened left main stem bronchus lies in the upper right corner. B = Left main stem bronchus; E = Esophagus.

wedge pressures were 26/10 mm Hg and 8 mm Hg respectively, and cardiac output was 8 L/min. The persistence of shock was attributed to sepsis; this was supported by the elevated cardiac output and normal wedge pressure. The CVP line was removed since findings on auscultation demonstrated an increasing left pleural effusion. A thoracentesis revealed 800 cc of chocolate-appearing fluid. The pH of the fluid was 6.3, with microscopic examination demonstrating polymorphonuclear white blood cells and numerous mixed bacteria. Analysis of the fluid revealed the following: protein, 2.9 g/dL; LDH, 3,460 IU/L; bilirubin, 4.3 mg/dL; and glucose, 22 mg/dL. Chest tube insertion was attempted and surgical exploration entertained; however the patient died in refractory shock despite aggressive fluid management and vasopressor agents. The final chest roentgenogram (Fig 1) demonstrated a streak of air paralleling and probably lying just above the left hemidiaphragm in association with some fluid in the left pleural space. The appearance was felt to be that of a small loculation of air lying between the base of the left lung and the superior surface of the left hemidiaphragm. (Discussions with family members postmortem revealed that the patient's complaints of left shoulder pain included discomfort in the mid-epigastric region.) Antemortem blood cultures grew *Escherichia coli.*

Postmortem examination revealed a perforated high posterior wall gastric ulcer adjacent to the lesser curvature. The ulcer penetrated through the diaphragm and into a loculated pleural space formed by basilar adhesions (Fig 2). The general pleural cavity was infected from this space and was filled with 2 L of foul, brownish exudate consisting of mixed bacteria and *Candida albicans.*

DISCUSSION

The patient described herein had a well documented gastric ulcer that perforated through the diaphragm into the pleural space. Other than esophageal rupture and pancreaticopleural fistulas, it is unusual for an abdominal organ to have as associated nontraumatic pulmonary or pleural communication. Reviewing the literature has revealed that fistulas connecting the stomach and bronchus have occurred: diaphragmatic hernia[4]; trauma to the diaphragm[5]; and complications of a subphrenic abscess.[6,7]

The diagnosis of a ruptured gastric ulcer into the pleural space should follow the same sequential steps as that for a ruptured esophagus. A thoracentesis with pleural fluid revealing an extremely acidic exudate,[8] a low glucose, and an elevated bilirubin—as compared to blood values—should raise the suspicion of a gastro-pleural communication. It appears that the cause would be established by contrast studies utilizing meglumine diatrizoate, such as in esophageal rupture. These contrast studies are positive in 85% of esophageal ruptures.[2] In rare instances computerized tomography of the chest may be helpful in determining a gastro-pleural defect.[9]

A gastro-pleural rupture is associated with high mortality if it goes unrecognized without surgical intervention. The symptoms of actual perforation may be acute with possible hemodynamic decompensation. Thoracentesis should be carried out immediately when a pleural effusion is present under clinical and roentgenographic conditions such as those described, particularly when air is seen in the pleural space. If an extremely low pH and elevated bilirubin are found with polymorphonuclear white blood cells and mixed bacteria, chest tube insertion is imperative. Definitive diagnosis with meglumine diatrizoate instillation should be performed, as for suspected esophageal rupture.[2] Should this be positive, emergency surgery is warranted.

REFERENCES

1. Light RW: Pleural effusion secondary to diseases of the gastrointestinal tract, in *Pleural Diseases.* Philadelphia, Lea & Febiger, 1983, chap 16, pp 151-162.
2. Michel L, Grillo HC, Malt RA: Operative and nonoperative management of esophageal perforations. *Ann Surg* 1981; 194:57-63.
3. Tombroff M, Loicq A, DeKoster JP, et al: Pleural effusion with pancreaticopleural fistula. *Br Med J* 1973; 1:330-331.
4. Urizza JL, Ramirez Gama J: Fistula gastrobronquial consecutiva a hernia diafragmatic, no traumatica. *Rev Med Tuberc Aparato Resp* 1954; 15:208-211.
5. Seyfarth KA: Gastrobronchial fistula after traumatic rupture of the diaphragm. *Fortschr Roentgenstr* 1959; 91:408-409.
6. Mason LB: Subphrenic abscess. Report of a case with fistulous connections to the stomach, colon and bronchus. *Am Surg* 1965; 31:278-280.
7. Hathirat S, Renzetti AD Jr: Gastrobronchial fistula complicating subphrenic abscess. A case report. *Am Rev Respir Dis* 1969; 99:581-584.
8. Light RW, Macgregor MI, Luchsinger PC, Ball WC Jr: Pleural effusions: the diagnostic separation of transudates and exudates. *Ann Intern Med* 1972; 77:507-513.
9. Faling LJ, Pugatch RD, Robbins AH: The diagnosis of unsuspected esophageal perforation by computed tomography. *Am J Med Sci* 1981; 281:31-34

OMMISSION

The authors of "Silicone granulomas of the breast" (1985; 85:599-601) are grateful to the following physicians for the clinical information and follow up on the reported patients: Nancy Mellow, MD, Christopher B. Mills, MD, Ronald S. Levandusky, MD, and Steven Wong, MD.

MSSNY Medical Liability Reform Fund Honor Roll

In the September issue the *Journal* began publishing the list of physicians who have generously contributed to the campaign to inform the public about the crisis in medical liability litigation. The names of additional contributors are listed below.

John W Abbuhl, MD
Bhaskar T Achar, MD
Irving Nelson Agrin, MD
Margaret R Albanese, MD
Henry Robt Alderman, MD
Robert Edwin Alessi, MD
Burton Allyn, MD
Bertram E Alpert, MD
Richard Ament, MD
Paulo Emiliano Ampil, MD
Stavroula Angelakos, MD
Boonchuay Anunta, MD
John Gregory Apisson, MD
Herbert Michael Archer, MD
Ali I Arkan, MD
John Francis Assini, MD
Carl Irving Austin, MD
Jeanne Ruth Willner Baer, MD
Clyde Nelson Baker, MD
Seymour Saml Balkin, MD
Sawaran Singh Bambrah, MD
Charles Raymond Bannon, MD
Jacob Joseph Barie, MD
Clement Michael Barone, MD
Robert Michael Barone, MD
Anthony G Barraco, MD
Donald Alexander Barrett, MD
Sidney Saml Becker, MD
Harold Bedell, MD
Richard Harry Bennett, MD
Jerome Bernstein, MD
Martin Seymour Bernstein, MD
Gopal Achut Bhandarkar, MD
Kalyani Mohanbhai Bhuta, MD
David Biddle, MD
Louis U Bigliani, MD
William H Bloom, MD
David Allan Blum, MD
David A Blumenstock, MD
Robert David Bolinder, MD
Philip Peter Bonanni, MD
A Gale Borden, MD
Charles Changmin Bou, MD
Robert R Bowditch, MD
Frederick D Bowman, MD
George Clark Brady, MD
Donald F Brautigam, MD
Percy M Brazil, MD
B Lawrence Brennan, MD
Gail Brenner, MD
Philip Alan Bresnick, MD
Bruce W Brewer, MD
Eugene A Brody, MD
Bertram Elias Bromberg, MD
Earl Benedict Brown, MD
Matthew Jos Brown, MD
Norman Geo Brunner, MD
Wahid A Bserini, MD
Joseph T Bumatay, MD
Douglas Francisco Buxton, MD
Jorge Luis Camunas, MD
Daniel L Carlson, MD
Patrick A Carruthers, MD
Anthony P Caruso, MD
Anthony Stephen Casimir, MD
Fritz Casimir, MD
Arminius Cassvan, MD
Rogelio G Castillo, MD
Michael G Cedars, MD
Marcial Marcelo Cerruti, MD
William Risley Chaffee, MD
Betty Sauwai Wong Chan, MD
Joseph M Chandrankunnel, MD
Sreedevi Chandrasekhar, MD
Daniel Chansky, MD
Mohammed A Chauhdry, MD
Norman Lionel Chernik, MD
Herbert Sylvan Cherry, MD
Wha Ja Choi, MD
Young W Choi, MD
Jayanta Kumar Chowdhury, MD
Keng Jong Chung, MD
John Joseph Cirelli, MD
Frank James Cirincione, MD
Richard Ira Cohen, MD
Seymour M Cohen, MD
Mitchell David Cohn, MD
D Jackson Coleman, MD
George Leonard Collins, Jr., MD
David Andrew Collins, MD
Patrick S Collins, MD
Joseph R Connelly, MD
Leroy Henry Cooley, MD
Jerome Abraham Cooper, MD
Leon John Corbin, MD
Laura E Corio, MD
Robert Mande Corwin, MD
Patrick Rudolph Coseglia, MD
Robert Louis Cristofaro, MD
Dolores V Cuenca, MD
Michael Victor Cummings, MD
Krystyna Czarlinski, MD
Bernard Andrew Daly, MD
Daniel Asher Daniels, MD
Donald R Davidson II, MD
Morton I Davidson, MD
Fe Teresa De Jesus-Mc Carthy, MD
Frank Ross De Luca, MD
John Carroll De Mocker, MD
Roy Alan Debeer, DO
Ray Eugene Deuel, MD
Robert M Dewell, MD
Vincent Robert Di Gregorio, MD
James Edwin Dickson II, MD
Kishore Vajendra Divan, MD
Bernard F. Donovan, MD
Richard J Dranitzke, MD
Norma G Dreyfus, MD
Samuel Drix, MD
Alice Noak Dunajewski, MD
Abigail Durruthy, MD
Stephen Dvorin, MD
Naso Tom Dzongowski, MD
Wayne Albert Eckhardt, MD
David Robert Edelstein, MD
S Leonard Edelstein, MD
Gary E Eggleston, MD
Jalal Elahi, MD
John Michael Esposito, MD
Elizabeth G Estrin, MD
Michael R Ettenson, MD
Fredric Ira Fagelman, MD
Carl Peter Faso, MD
Richard Owen Fass, MD
Zafar Ul-Hasan Fatimi, MD
Joshua Mark Feibusch, MD
Frederick Steven Feiner, MD
Ernest Feldmar, MD
Jose L Fernandez, MD
Gary Richard Fink, MD
Solomon Fisch, MD
Frank Craig Fisher, MD
Eugene Somer Flamm, MD
Thomas M Flanagan, MD
Lewis M Flint, MD
Constantine E Flokas, MD
A David Flug, MD
Anthony A Formato, MD
Edith Millicent Forsyth, MD
Alvin Sidney Fox, MD
Cedric Francis, MD
Alvardo Mc Donald Fraser, MD
Michael Freedman, MD
Alvin Henry Freiman, MD
Julian Frieden, MD
Mary Anna Friederich, MD
Ira Hugh Friedman, MD
Richard E Fullerton, MD
John Francis Furey, MD
Edward Lyons Gallagher, MD
John Francis Gallagher, MD
Pamela M Gallagher, MD
Henry Francis Gardstein, Jr., MD
Paul David Garson, MD
Albert A Gartner, MD
Edward L Gaviola, MD
Abraham Geffen, MD
Samir M Geleil, MD
Seymour Gendelman, MD
Samuel M Gingold, MD
Bernard Gitler, MD
Peter Francis Goergen, MD
Alan Howard Gold, MD
Marcel Goldberger, MD
Grant Steven Golden, MD
Edmund Goldenberg, MD
David Goldgewert, MD
Michael Allen Goldsmith, MD
Stanley Goldstein, MD
William Goldwasser, MD
Mohinder Singh Goomar, MD
David Meyer Gordon, MD
Joseph Gordon, MD
Ezra Martin Greenspan, MD
Jack Michael Greenwood, MD
Orman Gregersen, MD
John Francis Griffin, MD
Joshua David Gross, MD
Bruce B Grynbaum, MD
Victor Guarneri, MD
Rama M Guntupalli, MD
Louis Robt Guzzetta, MD
Jamil Raouf Haddad, MD
Susan Jane Hadley, MD
Mark R Hagen, MD
Robert Douglas Hahn, MD
Anne Harrison, MD
William Jay Heidenberg, MD
Walter J Henry, MD
William Edwin Hermance, MD
Waldemar F Hermann, MD
Frederick Peter Hixson, MD
Ross Arthur Horsley, MD
Solomon Theodore Horwitz, MD
Ziver Huner, MD
Malcolm G Idelson, MD
Philip S Irie, MD
Hashim M Iyoob, MD
Theodore S Jackaway, MD
Josef Jahr, MD
Robert Allen Jakubowski, MD
William J Jameson, MD
Roger G Jammal, MD
Harry Jasper, MD
Francis M A Jaynal, MD
Fathi Abdul-Qader Jishi, MD
David N Johnson, MD
Paul G Jones, MD
Robert Michael Kalet, DO
Kaizer T Kamble, MD
Charles Joseph Kandler, MD
Gerald B Kara, MD
Debra Karnasiewicz, MD
Robin B Karpfen, MD
Himeko Kashiwabara, MD
Mark H Kasowitz, MD
Albert Stewart Katz, MD
Ralph Katz, MD
George Kaufman, MD
Walter Ernst Kaufmann, MD
George Adelbert Keating, MD
Phillip Keating, MD
Theodore N Keltz, MD
Michael Joseph Kendrick, MD
Erich Kim, MD
Syung Soo Kim, MD
Zung Wan Kim, MD
Michael Max Klein, MD
Russell George Knapp, MD
Baburao Koganti, MD
Young Hyun Koo, MD
Irene Levay Krisztinicz, MD
Vito S La Rocca, MD
Ranjit Kumar Laha, MD
Steven Miles Lapidus, MD
Dominador R Lapus, MD
Stephen Laufgraben, MD
Anthony Romuald Lazarus, MD
Arthur Stewart Lebowitz, MD
Harvey Lederman, MD
Gene Woong Lee, MD
Soo Duk Lee, MD
Young Sun Lee, MD
Norman E Leeds, MD
Martin R Leopold, MD
Fred S Levinson, MD
Roger Norman Levy, MD
Leon V Lewis, MD
Nathan Lieberman, MD
Ru-Kan Lin, MD
Tung-Hui Lin, MD
Robert William Linehan, MD
Benedetto A Lo Balbo, MD
Mario Benedict Lo Monaco, MD
Michael M Madden, MD
Jasbeer S Makhija, MD
Alfred Edgar Mamelok, MD
Albert Anthony Maniscalco, MD
Harold Edwin Marden, MD
Jon Owen Marks, MD
Robert A Marks, MD
Jacob David Matis, MD

Harold C May, Jr., MD
Thomas J P Mc Gowan, MD
Joseph B Mc Ilduff, MD
Robert John Mc Mahon, MD
Walter Charles Meier, MD
Bertram Meister, MD
Stanley M Mendelowitz, MD
Frederic A Mendelsohn, MD
Nandakumar Menon, MD
Carmen D Metildi, MD
John H Meyers, MD
Kenneth Lee Miller, MD
Richard J Miller, MD
Arnold Aaron Minsky, MD
Alexander Mintzer, MD
Mahendra J Mirani, MD
Anthony Joseph Mistretta, MD
Winston G Mitchell, MD
Leonard Joseph Mogilner, MD
Francis Edward Moore, Jr., MD
Maurice J Moore, MD
Gustavo J Moreno, MD
Kevin Peter Morrissey, MD
Paul Colhoun Morton, MD
Craig Moss, MD
Donald Moy, JD
Usha D Mukhtyar, MD
James Wm Murphy, MD
Albert Nadaf, MD
C Richard Nangle, MD
Ronald A Naumann, MD
Jose Florendo Nebres, MD
A Nemi, MD
Howard Roger Netter, MD
Peter R Neumann, MD
Michael George Neuwirth, MD
Michael Joseph Newton, MD
Charles Ney, MD
Lillian F Vitanza Ney, MD
Robert G Ney, MD
Edward Arthur Norris, MD
Mohamed Khalafalla Nour, MD
Patrick Emmett O'Hara, MD
Ferdinand A Ofodile, MD
Yak Sing G Ong, MD
Benjamin William Pace, MD
Horacio Acosta Padua, MD
David Thurber Page, MD
Donald G Paish, Jr., MD
Narhari M Panchal, MD
Harry John Pappas, MD
Richard L Parker, MD
Edgar Alan Parmer, MD
Gasper Parrino, MD
Robert Lee Patterson, MD
Daniel Joseph Paulo, MD
Gerald L Peer, MD
John Michael Pepe, MD
Daniel L Picard, MD
Harold Arthur Pike, MD
Dominic Anthony Pompa, MD
Jesse James Pone, Jr., MD
Robert A Press, MD
Sidney Issac Rabinowitz, MD
Heena P Rajdeo, MD
Raul Al Ravelo, MD
John Munn Recht, MD
Louis D Retz, Jr., MD
Warren Thomas Rinehart, MD
Benito Benj Rish, MD
Werner Joseph Roeder, MD
George Roff, MD
Charles Milton Rogers, MD
Frank L Rollo, MD
Francis Graham Ronan, MD
Floyd D Roos, MD
Pedro J Roque-Rodriguez, MD
Joseph Carl Rosenbluth, MD
Robert George Roth, MD
Lester D Rothman, MD
Sidney N Rothstein, MD
Clyde P Rowe, MD
Joseph F Rowley, MD
Ira Stephen Rubenstein, MD
Laurence Rubin, MD
Leonard Rupert Rubin, MD
Peter H Rubin, MD
Alan L Rubinstein, MD
Janusz E Rudnicki, MD
Theodore J Rusnack, MD
Thaddeus Rutkowski, MD
Albert E Sacknoff, MD
Jalal Sadrieh, MD
Ismet Sakarya, MD
Joseph Anthony Salerno, MD
Arnold Salop, MD
Robert E Sandroni, MD
Guillermo A Sangalang, MD
Michael Ottmar Sauter, MD
Roger G Schneider, MD
I Harvey Schotter, MD
Robert J Schuder, MD
Malcolm H Schvey, MD
Paul P Schwach, MD
Ammiel D Schwartz, MD
Gerald Vincent Schwartz, MD
Jerold Schwartz, MD
Harold N Schwinger, MD, P.C.
Robert Willard Scott, MD
Zia Segatol-Islami, MD
Ernesto Daniel Seldman, MD
Charles Louis Settembrini, MD
Rajasekar Sham, MD
Gregory Bernard Shankman, MD
Peter A Shapiro, MD
Suresh Chander Sharma, MD
Yogendra Datta Sharma, MD
Herbert S Sherry, MD
Albert Sherwyn, MD
Mohammed Y A Siddiqui, MD
Robert S Siffert, MD
Jay B Sigel, MD
Robert Arnold Silverman, MD
Roger Lawrence Simpson, MD
Lawrence H E Smith, MD
Antanas Snieska, MD
Barney Softness, MD
Hamilton Southworth, MD
Robert Lee Spiegler, MD
Howard A Spindler, MD
Edward A Stehlik, MD
Otto Steinbrocker, MD
Neil Sillars Stewart, MD
Irwin Kingsbury Stone, MD
Lee Ramsay Straub, MD
Margaret E Sullivan, MD
K N Sundaram, MD
Janet Hyunsook Sung, MD
Vito John Susca, MD
Pragna B Sutaria, MD
Norman Sveilich, DO
Samuel Simon Sverdlik, MD
John Knowles Swinburne, MD
Walter L Sydoriak, MD
Walter Bohdan Szpur, MD
Romeo Velasco Tamayo, MD
Apostolos P Tambakis, MD
Glory C Tancinco, MD
Merle Nito Tandoc, MD
Satayarayana V Tanguturi, MD
Robert Carl Tatelbaum, MD
James Ziegler Taylor, MD
Sanford Posnick Temes, MD
Richard Newman Terry, MD
Theodore F Thomas, MD
Tong Seng Tjoa, MD
Joseph C Todoro, MD
Norman Touger, MD
Clyde Winston Turner, MD
Morris Unher, MD
Claudia Azucena Useda, MD
Vincent James Valente, MD
Fred Charles Van Natta, MD
Jetse Van Vliet, MD
Cesar V Veloso, MD
Anthony J Viglietta, MD
Marcos Gilberto Viguera, MD
Henry Vinicor, MD
Earle W Voorhees, MD
Gary I Wadler, MD
Richard H Walden, MD
Henrietta L P Wallace, MD
George Edward Wantz, MD
Medical Society of the
County of Warren
David Anthony Webb, MD
Joseph Weiss, MD
Ernest A Weymuller, MD
Edward A Willens, MD
Donald Hagedorn Wilsey, MD
David Alan W Wilson, MD
Felix Wimpfheimer, MD
Robert Corey Yates, MD
Ernest L Yu, MD
Bernard S Yurick, MD
Harvey David Zara, MD

MEDICINE AND THE MANAGEMENT OF LIVING. TAMING THE LAST GREAT BEAST

By William Ray Arney, Bernard J. Bergen. 202 pp. Chicago, University of Chicago Press, 1984. $19.95 (hardcover)

William Ray Arney, a sociologist at Evergreen State College, and Bernard Bergen, a Professor of Psychiatry at Dartmouth Medical School, draw on a medical sociological framework to critically analyze changing attitudes and values in medical practice. *Medicine and the Management of Living* offers both a historical perspective on the nature of patient and physician relationships, and an extensive commentary on the expanded role of medicine today. The authors describe a revolution in medical practice during the last three decades that places the physician in the pivotal role of managing the living. Physicians, through broad systems and an ecological framework, no longer focus on disease but view their role as enablers in partnership with patients. They move patients along the continuum of life's processes, mediating between individuality and established normative processes. This is contrasted with a previous medical practice model that organized around objective disease processes and ignored the subjective reaction and participation of patients.

Following an introduction that sets the stage, chapter two describes a period during which disease was increasingly viewed as the enemy of life. Life processes were to be measured, dissected, differentiated, and classified. The physicians as warriors fighting the disease, lost the sense of the whole person, and patients lost their role in their own major life events. The title of the chapter, "The disappearance of the experiencing person," aptly describes this phenomenon and leads to chapter three entitled, "The return of the experiencing person." The authors focus largely in this chapter on modern medicine's alienation of people to their own deaths. Death had become "owned" by physicians who were enpowered to defeat death, estranging people from this central life and family experience.

The authors point to the 1950s as the time of a growing medical revolution and momentum that changed the concepts of doctor, patient, and disease. Goals of medical care, of health and cure, were being renegotiated. As doctors became concerned with the life cycle in its broader sense rather than technologies of "invasion and correction," an entirely new arena for intervention and responsibility developed. This new therapeutic alliance between physician and patient shares power and encompasses a multi-dimensional scope of influences and networks. In chapter four, Arney and Bergen view this changing structure as physician inspired and led rather than forced by consumers and larger societal pressures. Emphasis on biophysical, psychologic, and sociologic considerations become paramount.

Chapter five describes systems theory and how broad systemic thinking influences alternative approaches to treatment. Now moving beyond even mind-body considerations, the physician must comprehend relationships between the psycho-physiologic and the external world. This expectation to recognize, evaluate, prevent, and treat all the physician, psychologic, socio-economic, and cultural variables that relate to the disease process is a demanding responsibility. The authors believe that the responsibilities posed by a systems theory orientation is now an irreversible part of medicine's course.

Chapter six offers a full description of the changing role of physicians as team leaders, managers of the patient's health care, and resolvers of problems experienced by patients. "The patient with a problem, defined in terms of the patient's entire life, has displaced the disease as the object of medical attention." Management defined as the designing and implementation of the most effective program of care becomes a key concept and role.

The focus on life's processes reframe birthing, aging, and dying as a joint adventure with a beginning and eventual ending. Medicine offers willful and free individuals a map of the good life, assisting them in the achievement. Supportive experiences and support groups are considered a way individuals are helped to achieve positive social norms and more optimal standards. Risking this potential growth and change-oriented direction is the tendency to blame patients (victim blaming). On a larger societal scale, life styles become more health-impairing than the social causation of illness. Inequality of access to health care and social conditions are given less credence as a cause of illness than is control of individual choices through life style change. Also, expert social and technical knowledge determines for people the expectations that will guide and shape their behavior and reactions. This can be a subtle coercive force.

Finally, the authors present the conflicts inherent for physicians in responding to individual needs while being called upon to preserve order. In present terms, this may require decisions that result in rationing health care. The authors postulate that "the physician and patient who enter a therapeutic alliance are thereby pledged to reason together to find the ethically appropriate, socially harmonious course of action." They assert that the incorporation of spirituality, sociobiology, and ethics into medical discourse is necessary as medicine searches for a new model for order.

In ten brief chapters, there is a portrayal of an expanding calling that is extending medicine's reach. Rather than medicalize more aspects of life and move beyond boundaries, it is responsive to new parameters and environments.

The theoretical frameworks presented are often abstract, obscure, and obtuse. It is a difficult book to read and to comprehend despite the prevailing sense that the authors are building far-reaching concepts. Although the book is on a high intellectual level, one might question whether medicine has moved in the direction proposed by the authors. So much technology and disease-oriented medicine remains that it is difficult to envision this new order. The expanded role of the physician as a mediator between the individual and social forces that impact on health, and as a guide to the good life, seems more like a modern day ideal than reality. There is no doubt that medicine's role must encompass a complex sphere, but the authors are more convincing in presenting theory than in reporting on observable practices or on operational models.

ZELDA FOSTER, MSW
Veterans Administration
Medical Center
New York City 11209

TO PROVIDE SAFE PASSAGE: THE HUMANISTIC ASPECTS OF MEDICINE

Edited by David Rabin, MD and Pauline L. Rabin, MD. 258 pp. New York, Philosophical Library, Inc, 1985. $19.95 (hardcover)

The general theme of this book is the humanistic responsibilities of health care professionals toward incurable patients. The editors, a husband-wife team and both physicians, have edited a series of twenty essays written by parents, patients (some of whom are physicians), and physicians, all having personal knowledge of their subject material. David Rabin was, until his recent death, Professor of Medicine (Endocrinology) and Professor of Obstetrics and Gynecology; Pauline Rabin is an Associate Professor of Psychiatry at Vanderbilt University School of Medicine.

The text begins simply, if not deceptively, with Francis Peabody's landmark essay entitled, "The Care of the Patient." The Rabins' second chapter summarizes and expands its salient points including the concept of individualized personal care, the dehumanization of the hospital experience, and the care of the patient with no ostensible organic disease. This chapter uses this material as a springboard for a fourth key point; the care of the terminally ill and dying patient. Utilizing Jacob Bigelow's concept of the compleat physician as one who diagnoses, treats, and provides safe passage, the Rabins examine this emotionally laden issue with an admixture of authoritative detached concern and calculated sensitivity. In a sense, the reader can passively absorb this classroom presentation with the usual amount of professional distance. He is ill-prepared for the poignancy of David Rabin's trying ordeal with amyotrophic lateral sclerosis described in the following three chapters. The reader is thrust into the uncomfortable story of a physician's terminal illness. The forbidden topic of mortality cannot be avoided; its uncertainty and ambiguity must be grappled with and confronted. The social and professional isolation that David Rabin experiences makes the reader squirm; one can no longer don the white coat with so much compliance. As his disease progresses, Rabin becomes a pariah and lacking the "passport of the healthy" he is now marked with the "interdict of death." At parties, no one talks to him or his wife (premature widow syndrome). When he falls, even his colleagues do not help, pretending that they did not see the incident. Are not these episodes similar to most physicians' behavior with terminally ill patients? The interpersonal distance becomes greater as death approaches. Cursory "hallway" rounds become a substitute for more in-depth bedside contact.

The subsequent chapters deal with different aspects of death, dying, and the incurable patient. The chapter on chemotherapy is especially provocative as it is written by a physician patient suffering from lymphoma. The reader receives an "insider's" perspective into the social isolation, problems, and lessons of the patient's six month ordeal. The management of intractable pain, the care of patients with dementia, and genetic counseling are then discussed by physician experts. The focus is not so much on hard core medical fact, but rather on the difficult psychosocial aspects of chronic or progressive disorders (ie, pain perception, family stress).

The sections dealing with hospice care, the mentally retarded, and anger are written by lay people who have had first-hand experience with incurable illness. The humanistic aspect of illness rather than disease pathology is the consistent message. The concluding chapters consider the philosophical elements of the dying process from the patient's and also the physician's viewpoint. They concisely synthesize the preceding material, and then as if preparation for the inevitability of death, expose the reader to the acute grief process and the five stages following death as originally described by Kubler-Ross. No stone is left unturned. Family matters are addressed; the impersonal, mechanistic approach of current medical education is critiqued; and one last, indelible insight into the dying patient, David Rabin, is provided. The egocentric, joyless "Why me?" becomes the selfless, playful "Why not me?" This is the ultimate insight because it liberates the patient in order to accept the illness and move ahead in a positive, hopeful manner. The reader also greatly profits by appreciating the same truth.

There are several weaknesses in the text. For instance, a chapter on chronic illness (diabetes, hypertension, arthritis, ischemic heart disease) would have completed the spectrum of incurable illness. Such problems are common and frustrating because a cure is not possible and caring is often overlooked. Morbid curiosity aside, further details of Rabin's illness at the interpersonal (family) and intrapersonal (career) levels would have crystallized the issues for even the most insulated reader. For physicians, it is much easier to deal with relatively ambiguous theoretical material on death and dying because cold reality cannot be readily dismissed.

From this reviewer's perspective, this book cuts to the heart of the curing-caring dichotomy. Many physicians erroneously equate the two concepts. However, this is rarely the case. Chronic illness, in particular dementia, genetic handicaps, retardation, and mental illness are by definition incurable, whereas most infectious diseases, many forms of trauma, and surgical problems can be cured. Curing and caring constitute the complementary ends of a continuous spectrum. In order for the marriage to be solvent, elements of each must always be present. Care without cure is unacceptable in today's high technology medical world and cure without care reduces the physician-patient relationship to automated machinery.

At the very least, this book should be required reading for all medical students and house staff, and where possible, patients who suffer from an incurable illness. Ideally, the reading should form the core of a course devoted to humanism in medicine. Certainly, physicians who are trained to be "cure providers" must reckon with inherent limitations of the scientific method. They must also come to grips, preferably early on in their careers, with their own mortality. By so doing they can become adept "care-givers." At all times the compleat physician must dynamically balance the scientific aspects of "curing" with a goodly dose of "caring." He or she must be able to honestly and regularly reassure the incurable patient that all that can be done has been done, and allay overwhelming anxieties even unto death. Only then will safe passage be provided.

RICHARD BIRRER, MD
State University of New York
Downstate Medical Center
New York City 11203

DOUBLE EXPOSURE. WOMEN'S HEALTH HAZARDS ON THE JOB AND AT HOME.

Edited by Wendy Chavkin, MD. 276 pp, illustrated. New York, Monthly Review Press, 1984. $10.00 (paperback)

Occupational hazards face nearly everyone at the worksite. Specific hazards that affect the health and safety of women are more apparent in jobs that are primarily filled by women, especially when those hazards affect the reproductive system. This book encompasses background information pertaining to specific settings and risks that women face on the job. The primary purpose is to introduce the reader to current risks and problems at the worksite as well as to the history leading up to the recognition of these problems.

Dr Chavkin is a physician with a background in obstetrics and gynecology and occupational health. She has been involved in organizations concerned with the reproductive rights of women and with the issue of environmental hazards and reproduction. Dr Chavkin currently works with the New York State Department of Health on reproductive and occupational health issues. As editor, she has brought together numerous authors who have provided an important overall view of the types of problems women face on the job at the work site and on the job at home.

The book is divided into three sections: women at work, work and reproduction, and women at home and in the community. Information pertaining to specific risks as well as examples of such risks are presented for the office worker, the nurse, minority women, migrant and seasonal workers, and women who work at home (housework, wifework, and motherwork). An entire section is devoted to reproductive risks that include information on selective hiring practices, concerns of male reproductive risks as well as female risks, and specific needs of pregnant workers. A chapter on sexual harassment and how it affects health is also included.

This book is not intended to provide textbook information on occupational health. It is written to provide background information and examples of risks and problems women face at the worksite and how to actively try to improve this environment. Any compilation of chapters written by different authors yields differing backgrounds and writing styles. However, it is clear that Dr Chavkin provided solid direction for the authors. The book is written from an activist's perspective. One of the book's strongest assets is the reference section at the end of each chapter.

Double Exposure represents an excellent supplementary text for those interested not only in the history of occupational health but also in current and active concerns of women who are involved in educating the public about these problems. In addition it describes the methods necessary for changing working conditions that affect the health and safety of women who work.

BETSY T. KAGEY, MSPH
State University of New York
Downstate Medical Center
New York City 11203

ATLAS OF OSTEOARTHRITIS

By Allen D. Meisel, MD and Peter G. Bullough, MB, CHB. 224 pp, illustrated. Philadelphia, Lea & Febiger, 1984. $29.50 (hardcover)

This book is a welcome addition to the ever expanding literature on rheumatology. It is a pleasure to read because of its relatively small size and attractive graphic art, and is likely to be purchased and read cover to cover.

The authors' use of the term "osteoarthritis" is a refreshing step backwards in the right direction. Inflammation is recognized as playing a role in this condition, as evidenced by the widespread and effective clinical use of anti-inflammatory drugs and joint injections. Furthermore, as is pointed out in the introduction, the term "degenerative joint disease" is depressing and misleading to both patients and nonrheumatologists alike.

The title is somewhat of a misnomer; "An Introduction to Osteoarthritis" might be more appropriate. This volume is more than an atlas, but less than a comprehensive review of the subject. There are only about 180 pages of text (the pages are not numbered), the print is large, and most of the space is taken up with illustrations, graphs, and tables. For example, one half page is used for a single drawing on one method of leg length measurements.

The first chapter, which is the high point, is a very clear and concise review of joint biology, from its biochemistry through the pathomechanics of injury. The diagrams and pictures of gross and microscopic sections are excellent. The chapters on the various joints include sections on physical examination that contain useful diagrams which is a feature helpful to students. However, the text is too brief and sketchy to present a clear clinical picture of osteoarthritis and its effects on the patient. The beautiful pictures of the gross specimens and radiographs do not convey the symptoms of this disease to the reader.

The final chapter on treatment mainly covers drugs and is probably too basic for practicing physicians. The "throw-aways" are already replete with reviews of the nonsteroid anti-inflammatory drugs. It was disappointing to find only one paragraph on physical therapy and almost nothing at all on occupational therapy. Assistive devices are barely mentioned, such as raised toilet seats for osteoarthritis of the hip and knee, jar openers for those with osteoarthritis of the first carpal-metacarpal joint, and Thomas Heels with medial wedges for genu valgus.

Some clinical statements are surprising. For example, "osteoarthritis of the lumbar spine is the most common identifiable cause of back pain." Why not muscle spasm? And "L4-L5 is the most frequently involved disc space in lumbar disc disease." Why not L5-S1? A table of differential diagnoses of neck pain does not include fibrositis or trapezius syndrome. The authors at one point refer to the eighth cervical vertebrae. DISH syndrome (diffuse idiopathic skeletal hyperostosis) is not used to refer to ankylosing hyperostosis. Several pages are devoted to the examination of the knee for meniscus injury, whereas the anserine bursa (a more common source of knee pain) is neglected.

This atlas will be valuable for medical students and residents rotating on an arthritis service. It is not cluttered and it can be read in a few nights. I wonder how many owners of the standard arthritis texts ever read the chapters on osteoarthritis or physical examination. With an expanded discussion on the symptoms of osteoarthritis, and an added chapter on rehabilitation, I am looking forward to the second edition.

JAMES M. STROSBERG, MD
Sunnyview Hospital and
Rehabilitation Center
Schenectady, NY 12308

HEPATIC RADIOGRAPHY

Edited by Michael E. Bernardino, MD and Peter J. Sones, Jr, MD. 319 pp, illustrated. New York, MacMillan Publishing Co, 1984. $65.00 (hardcover)

This book concisely evaluates hepatic anatomy and imaging modalities that are used in the diagnosis of liver disease. It also discusses the radiologic evaluation of hepatic inflammatory and neoplastic diseases, with special emphasis on the non-surgical management of these problems. The editors and the various contributors to the volume adequately evaluate both simple plain film use and the use of advanced imaging techniques such as computed tomography (CT) and nuclear magnetic resonance (NMR) scans in diagnosing a variety of pathologic states.

Normal and abnormal plain film hepatic anatomy is carefully presented. This is followed by a discussion of the usefulness of hepatic scintigraphy, sonography, and CT and NMR imaging in diagnosing a variety of pathologic states. The presentation of hepatic angiography and splenic portography are adequate, but these discussions add no new information. However, interventional techniques outlining hepatic arterial and variceal embolization in both benign and malignant diseases are well presented.

The illustrative cases used in the book are excellent and the radiographs, CT, and NMR images are interesting and educational. While the plain radiographs, CT images, and nuclear scans reproduced are of good quality, the NMR images are small with poor resolution. The editors have organized the book so that each imaging modality is discussed separately. This is followed by a broad discussion of various hepatic diseases and then by a presentation of the advantages of the different imaging methods and their appropriate use in the workup of patients with specific diseases.

Recent references are provided at the end of each chapter. These are adequate in providing readers with additional sources of information. *Hepatic Radiography* addresses the important question of the appropriate and specific use of imaging techniques in diagnosing a variety of liver diseases. It fully explains the radiologist's role in diagnostic interventions such as in hepatic biopsy, hepatic drainage, and the treatment of hepatic disease. It is an excellent reference source for both experienced radiologists and house officers.

DANIELE SALVONI, MD
Cabrini Medical Center
New York City 10003

DIAGNOSTIC PROCEDURES IN CARDIOLOGY: A CLINICIAN'S GUIDE

Edited by James V. Warren, MD, Richard P. Lewis, MD. 494 pp. Chicago, Year Book Medical Publishers, 1985. $39.95 (hardcover)

Having recently reviewed a text on bedside cardiology in enthusiastic terms, it is more than curious that a text is now presented for review on the "era of high technology" in cardiology. From a clinician's standpoint, it's saving grace is that emphasis is placed, in spite of high technology, on the patient and not on the text.

A group of 28 contributors at Ohio State University, under the leadership of Drs Warren and Lewis, have compiled an excellent collection of articles on the various diagnostic tests in the evaluation of patients that are now available to the clinical cardiologist.

In the opening chapter, Dr Warren reminds us of Lewis Thomas' phrase, "halfway technology"—a state where expensive tests and therapies are provided in an attempt to arrest a disease process that could be so much more effectively and economically controlled if prevention were available. Much of cardiology today is in a state of "halfway technology."

In the second chapter, Dr John Neff discusses probability considerations in clinical medicine. He points out that physicians are inveterate testers (he might also have noted that cardiologists are inveterate gadgeteers), and therefore they must be able to evaluate the significance of any given test. He comments on accuracy, precision, and specificity in arriving at probability considerations.

The text goes on to emphasize the importance of the history and the physical examination as the basis of all cardiology evaluations. Interestingly, Dr Charles Wooley, who wrote this chapter, quotes Dr. A. Conan Doyle's Sherlock Holmes, who emphasized the importance of "the Thinking Person's Medical History" and "the Thinking Person's Physical Examination." The accumulation of factual data in a conversational (rather than an adversarial) setting, subjected to logical interpretation, sets the foundation for the evaluation of noninvasive and invasive testing.

The text is divided into two major sections, one on basic procedures and the other on clinical applications. The basic procedures include electrocardiography, cardiovascular radiology, systolic time intervals, echocardiography, exercise testing, nuclear cardiology, noninvasive arterial diagnosis, lipid studies, cardiac enzymes, electrocardiographic monitoring, electrophysiologic studies, cardiac catheterization, endomyocardial biopsy, and monitoring drug therapy. These chapters briefly describe the procedure and illustrate the physiologic significance of each type of testing.

The second section evaluates the various tests in relation to a number of different cardiac conditions. Although emphasis is placed on problems with ischemic heart disease, other conditions such as congestive heart failure, cardiac syncope, congenital heart disease, mitral valve prolapse, and prosthetic valves are discussed in terms of appropriate tests for proper evaluation.

This book is a succinct, practical guide to current state-of-the-art diagnosis of cardiac disorders. Residents and internists, as well as cardiologists, will find it most useful.

JOHN N. EDSON, MD
The Long Island College Hospital
New York City 11201

In addition to these listings, the Journal *will publish obituaries written by physician readers. Inquiries should first be made to the Editor.*

Ernest Ludwig Adler, MD, Gouverneur. Died April 30, 1985; age 78. Dr Adler was a 1932 graduate of Fachbereich Humanmedizin Justus-Liebig-Universität, Giessen. He was a Fellow of the American Academy of Family Practice and a member of the St. Lawrence County Medical Society and the Medical Society of the State of New York.

Rudolph F. Amyot, MD, Waterford. Died May 10, 1985; age 79. He was a 1934 graduate of Columbia University College of Physicians and Surgeons. Dr Amyot was a Diplomate of the American Board of Obstetrics and Gynecology, and a member of the Medical Society of the County of Rensselaer and the Medical Society of the State of New York.

Morris Binder, MD, Huntington. Died June 11, 1985; age 79. Dr Binder was a 1936 Licentiate of the Royal College of Physicians, Edinburgh. He was a Diplomate of the American Board of Psychiatry and Neurology, and a member of the American Psychiatric Association, the Suffolk County Medical Society and the Medical Society of the State of New York.

Samuel Blinder, MD, Bal Harbor, Florida. Died May 24, 1985; age 82. He was a 1926 graduate of George Washington University School of Medicine. Dr Blinder was a Fellow of the American College of Cardiology and the American College of Physicians, and a Diplomate of the American Board of Internal Medicine. He was a member of the New York Cardiological Society, the New York County Medical Society and the Medical Society of the State of New York.

George R. Bodon, MD, Key Largo, Florida. Died April 18, 1985; Age 81. He was a 1928 graduate of Semmelweis Orvostudomanyi Egyetem, Budapest. Dr Bodon was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Surgery, and a member of the Monroe County Medical Society and the Medical Society of the State of New York.

James William Bucci, MD, Albany. Died April 18, 1985; age 88. He was a 1924 graduate of Albany Medical College of Union University. Dr Bucci was a Fellow of the American College of Cardiology, and a member of the American Academy of Family Physicians, the Medical Society of the County of Albany and the Medical Society of the State of New York.

Louis Richard DeLuca, MD, Bronx. Died May 1, 1985; age 53. Dr DeLuca was a 1958 graduate of Facoltà di Medicina e Chirurgia Università de Roma. He was a Diplomate of the American Board of Psychiatry and Neurology and a member of the American Psychiatric Association, the Bronx County Medical Society and the Medical Society of the State of New York.

William Russell Dolan, MD, Lakeland, Florida. Died May 30, 1985; age 89. Dr Dolan was a 1922 alumnus of the State University of New York Upstate Medical Center. He was a member of the Onondaga County Medical Society and the Medical Society of the State of New York.

Egon Feldman, MD, Brooklyn. Died October 19, 1984; age 78. He was a 1939 graduate of the Universität Basel Medizinische Fakultät. Dr Feldman was a member of the Kings County Medical Society and the Medical Society of the State of New York.

Julia Fischler, MD, Bronx. Died May 5, 1985; age 80. She was a 1931 alumna of Medizinische Fakultät der Universität, Wein. Dr Fischler was a member of the American Academy of Family Physicians, the New York County Medical Society and the Medical Society of the State of New York.

Charles Collins Francis, MD, Sarasota, Florida. Died March 14, 1985; age 95. Dr Francis was a 1919 alumnus of New York University College of Medicine. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Mortimer Benjamin Genauer, MD, Staten Island. Died June 5, 1985; age 78. He was a 1931 graduate of the University of Louisville School of Medicine. Dr Genauer was a Fellow of the American College of Anesthesiologists, a Qualified Fellow of the International College of Surgeons, and a Diplomate of the American Board of Anesthesiology. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc., the Richmond County Medical Society and the Medical Society of the State of New York.

Edward I. Goldsmith, MD, New York. Died June 11, 1985; age 58. He was a 1950 graduate of Cornell University Medical College. Dr Goldsmith was a Fellow of the American College of Surgeons and the American College of Angiology, and a Diplomate of the American Board of Surgery. His memberships included the New York Society for Cardiovascular Surgery, the Academy of Medicine, the American Society of Tropical Medicine and Hygiene, the New York County Medical Society and the Medical Society of the State of New York.

Mervin Albert Henschel, MD, New York. Died June 8, 1985; age 82. Dr Henschel was a 1927 alumnus of Columbia University College of Physicians and Surgeons. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Medical Society of the State of New York

ANNUAL CME ASSEMBLY

Maintaining the Quality of
Health Care into the 21st Century.

March 7-9, 1986

and the

Annual Meeting of the House of Delegates

March 6-9, 1986

The New York Hilton
New York City

COURTESY N.Y. CONVENTION & VISITORS BUREAU

HIGHLIGHTS . . .

- 22 Sessions
- Panel Discussions
- Technical Exhibits
- President's Dinner Dance

CLIP & MAIL

Director of Front Office Operations
The New York Hilton
1335 Avenue of the Americas
New York, N.Y. 10019

Phone: 212-586-7000

for hotel use only

NAME ______________________ TITLE ______________
FIRM ______________________ PHONE ______________
STREET ______________________
CITY/STATE/ZIP ______________________
REMARKS ______________________
(please print)

NOTE: Reservations must be received no later than February 13, 1986 and will be held only until 6 p.m. on day of arrival unless later hour is specified.

please print

ARRIVAL DATE ______________________
☐ 2-4 p.m. ☐ 4-6 p.m. ☐ 6-8 p.m. ☐ after 8 p.m.
DEPARTURE DATE ______________________
(checkout time is 1 p.m.)

HAVE A CREDIT CARD? You can register at our QUICK CHECK desk. If you wish to take advantage of this credit card PAYMENT GUARANTEED reservation, please complete the information below.

☐ AMERICAN EXPRESS
☐ DINERS CLUB
☐ CARTE BLANCHE

Expiration Date ______________
____
Credit Card No.

Signature ______________________

MEDICAL SOCIETY OF THE STATE OF NEW YORK
Annual Meeting of the House of Delegates
March 6-9, 1986
Annual CME Assembly
March 7-9, 1986

Circle preferred rate:

Singles: $125 $135 $145
Double/Twin: $150 $160 $170
Executive Tower: $200, Single/Double
Suites: (P&1): $275 $340 $375
Suites: (P&2): $450 $485

Rates subject to 8¼% N.Y. State sales tax and $2.00 per room per night N.Y.C. occupancy tax. (If room at the rate requested is unavailable, one at the nearest available rate will be reserved.)

GENERAL INFORMATION

The Medical Society of the State of New York invites you to attend its 1986 Annual CME Assembly to be held March 7–9, at the New York Hilton. The Continuing Medical Education Sessions and Technical Exhibits are open to all physicians, their guests and the allied professions. The two and a half days of meetings will provide the individual physician with a possible 18 credits out of a total of 93 Category 1, CME credit hours being offered. Over 120 outstanding speakers, including a number from out-of-state, will present papers at 22 sessions.

The theme of the CME Sessions will be "Maintaining The Quality of Health Care Into The 21st Century." A few of the subjects to be presented are, Alcoholism and Gastroenterology; Realistic Uses of Computers in Medical Practice; Evaluation and Management of Temporal Mandibular Joint Disorders; Laser Therapy in Urogenital Disorders; and An Overview of Geriatric Psychiatry. For details, see the Preliminary Program which appears in the December 1985 issue of the *New York State Journal of Medicine*. Our program will offer the clinician new approaches in diagnosis and treatment of diseases in a wide range of medical specialties.

COURTESY NEW YORK CONVENTION AND VISITORS BUREAU

MUSEUM OF MODERN ART SCULPTURE GARDEN

Comments from attendees at the 1985 CME Assembly were: 'The Speakers are excellent and well-informed'; 'The presentation was timely, and the material was well illustrated'; 'My time was well spent'. Don't miss this opportunity to gain Category 1 credits over a weekend when you are free from office hours. Why not bring your family. There is no registration fee for MSSNY members. Professional representatives from more than 50 companies will be in the Rhinelander Gallery Exhibit Hall to discuss their products and services. The Technical Exhibits will include drug products, clinical laboratories, office equipment, computer services, educational, insurance protection, and financial services, career opportunities and more.

SPECIAL EVENTS

Thursday, March 6

The Annual Dinner Dance will be held in the Trianon Ballroom of the New York Hilton, in honor of Daniel F. O'Keeffe, M.D., President, preceded by a reception in the Mercury Ballroom at 7:00 p.m. All attendees are welcome. Tickets are $75.00 per person and should be ordered in advance from the Medical Society.

Friday, March 7 and Saturday, March 8

A reception for physicians, their guests, the allied professions and exhibitors will be held on both days in the Exhibit Hall between 5:00 p.m. and 6:00 p.m., compliments of the Medical Society of the State of New York. A free drawing for a color TV set will take place at both receptions.

HOTEL RESERVATIONS

Physicians must make their own reservations with the New York Hilton no later than February 13, 1986. After that date, rooms are on an availability basis only. If making reservations by telephone, please mention that you are attending the Medical Society Convention for billing at the special rate.

REGISTRATION

CME Registration will take place in the Rhinelander Gallery Exhibit Hall, located on the 2nd floor which is accessible by escalators from the lobby. Friday, March 7 and Saturday, March 8 from 8:30 a.m. to 5:00 p.m.; Sunday, March 9 from 8:30 a.m. to 12:30 p.m.

House of Delegates Registration will take place on the 2nd and 3rd floor promenades at the following hours: Wednesday, March 5, from 12 Noon to 7:30 p.m.; Thursday, March 6, Friday, March 7 and Saturday, March 8, from 8:30 a.m. to 5:30 p.m. and Sunday, March 9 from 8:30 a.m. to 12 Noon. All MSSNY members are welcome.

CME MEETING HOURS

Friday, March 7 —10 a.m.–1 p.m.
2 p.m.–5 p.m.

Saturday, March 8—10 a.m.–1 p.m.
2 p.m.–5 p.m.

Sunday, March 9 —10 a.m.–1 p.m.

EXHIBIT HOURS

Friday, March 7 —9 a.m.–11 a.m.
1 p.m.–6 p.m.

Saturday, March 8—9 a.m.–11 a.m.
1 p.m.–6 p.m.

Sunday, March 9 —9 a.m.–1 p.m.

1986
PRELIMINARY PROGRAM
Annual CME Assembly
March 7-9, 1986

Annual Meeting of the
House of Delegates
March 6-9, 1986

The New York Hilton
New York City

INDEX

Hotel Reservation Form 11A
General Information 12A
Scientific Program Committee 15A
Scientific Program 16A
Technical Exhibits 31A
Advance Registration 33A

REGISTRATION & MEETING HOURS

CME Assembly

Registration is open to MSSNY members, spouses, invited guests, non-member physicians, and members of the allied professions. There is no registration fee for MSSNY members, members of other state medical societies, residents/interns, speakers, medical students, medical assistants, and spouses. There is a registration fee for non-member physicians—$50; and allied professions—$10.

An advance registration form will be found in this section. For those who register in advance, your identification badge will be held at the CME Registration Desk located in the Rhinelander Gallery Exhibit Hall on the second floor of the hotel. For those who have not registered in advance, registration will take place on Friday, March 7, and Saturday, March 8, from 8:30 a.m. to 5:00 p.m., and on Sunday, March 9, from 8:30 a.m. to 12:30 p.m.

Identification badges are required for admittance to all CME Meetings and exhibits. All registrants are required to show identification at the registration desk before a badge is issued.

CME meeting hours will be on Friday, March 7 and Saturday, March 8, from 10:00 a.m. to 1:00 p.m. and from 2:00 p.m. to 5:00 p.m., and on Sunday, March 9, from 9:00 a.m. and 10:00 a.m. to 1:00 p.m.

Delegates

Registration for delegates will take place on the second and third floor Promenades as follows: Wednesday, March 5 from 12 Noon to 7:30 p.m.; Thursday, March 6, Friday, March 7, and Saturday, March 8, from 8:30 a.m. to 5:30 p.m.; and Sunday, March 9, from 8:30 a.m. to 12 Noon.

The 180th Annual Meeting of the House of Delegates will be called to order at 1:00 p.m. on Thursday, March 6, 1986 in the East Ballroom of the New York Hilton. The schedule follows: Thursday, March 6 from 1:00 p.m. to 5:00 p.m., Saturday, March 8 from 9:00 a.m.–5:30 p.m.; and on Sunday, March 9 from 9:00 a.m. to 1:00 p.m. (8:00 a.m.–2:00 p.m. if necessary)

AUXILIARY TO MSSNY

The Auxiliary House of Delegates meeting will take place on Friday, March 7 from 12:30 p.m.–5:00 p.m., and on Saturday, March 8 from 12:00 Noon–2:30 p.m. in the Mercury Ballroom of the New York Hilton.

CME CREDITS

The Medical Society of the State of New York designates the continuing medical education activities included in this CME Assembly as meeting the criteria for the designated number of credit hours in Category 1 of the Physician Recognition Award of the AMA, and/or MSSNY's Method #6.

Each program is acceptable for prescribed or elective credits by the American Academy of Family Physicians (AAFP) as designated. The Medical Society of the State of New York is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. The Medical Society's CME credit forms will be issued at the entrance to each meeting room at the opening of each session.

EXHIBITS

Technical exhibits will be located in the Rhinelander Gallery on the second floor of the New York Hilton. Exhibits will be on view Friday, March 7 and Saturday, March 8 from 9:00 a.m. to 11:00 a.m. and from 1:00 p.m. to 6:00 p.m.; and Sunday, March 9 from 9:00 a.m. to 1:00 p.m.

PHYSICIAN MESSAGE CENTER

Messages for the physicians attending the CME sessions may be left at the MSSNY Information Desk located in the Exhibit Hall on the second floor. Messages for Delegates may be left at the Delegates Information Desk on the third floor, West Promenade. The New York Hilton telephone number is 212-586-7000.

COFFEE BREAKS

Coffee breaks sponsored by the Medical Society of the State of New York, will be held in the Exhibit Hall on the second floor daily from 9:00 a.m. to 10:00 a.m. All registered attendees are welcome.

DINNER DANCE

On Thursday, March 6, the Annual Dinner Dance will be held in the Trianon Ballroom, preceded by a reception in the Mercury Ballroom at 7:00 p.m. Tickets should be ordered in advance from the Medical Society of the State of New York. Subscription is $75.00 per person. See the order form in this section.

RECEPTION

A reception, sponsored by the MSSNY, will be held on Friday, March 7 and Saturday, March 8 from 5:00 p.m. to 6:00 p.m. in the Rhinelander Gallery Exhibit Hall located on the second floor of the hotel. All physicians, their spouses, the allied professions and exhibitors are welcome. A free drawing for a color TV will be held at both receptions.

1986 ANNUAL CME ASSEMBLY

Medical Society of the State of New York
New York Hilton, New York City

Maintaining the Quality of
Health Care into the 21st Century

FRIDAY, MARCH 7

10:00 a.m. 1:00 p.m.	Cardiovascular Diseases, *Beekman Parlor*
	Gastroenterology/Alcoholism, *Sutton Parlor North*
	Obstetrics/Gynecology, *Sutton Parlor Center*
	Neurosurgery, *Sutton Parlor South*
	Physical Medicine & Rehabilitation, *Regent Parlor*
2:00 p.m. - 5:00 p.m.	Neurology, *Beekman Parlor*
	Pediatrics/Alcoholism, *Sutton Parlor North*
	Chest Diseases, *Sutton Parlor Center*
	Occupational & Environmental Health/Workers Comp., *Sutton Parlor South*
	Medical Liability Mutual Insurance Co., *Regent Parlor*
	Realistic Uses of Computers in Medical Practice, I, *Madison Suite*

SATURDAY, MARCH 8

10:00 a.m. 1:00 p.m.	Ophthalmology (All Day), *Beekman Parlor*
	Internal Medicine (All Day), *Sutton Parlor North*
	Hand Surgery/Anesthesiolgy (All Day), *Sutton Parlor South*
	Urology/Dermatology, *Sutton Parlor Center*
	Psychiatry (All Day), *Regent Parlor*
	Realistic Uses of Computers in Medical Practice, II, *Madison Suite*
2:00 p.m. - 5:00 p.m.	Ophthalmology, *Beekman Parlor*
	Internal Medicine, *Sutton Parlor North*
	Otolaryngology, *Sutton Parlor Center*
	Hand Surgery/Anesthesiology, *Sutton Parlor South*
	Psychiatry, *Regent Parlor*

SUNDAY, MARCH 10

*9:00 a.m. - 1:00 p.m.	General Surgery/Colon & Rectal Surgery, *Beekman Parlor*
10:00 a.m. - 1:00 p.m.	Allergy & Immunology, *Sutton Parlor North*
	Women's Medical Society of N.Y./Family Practice/Emergency Medicine, *Regent Parlor*
	Pathology, *Sutton Parlor Center*

CME CREDITS: Each program will be eligible for CME Category 1 credits on an hour-for-hour basis.

◆

Technical Exhibits

1986 ANNUAL CME ASSEMBLY

SCIENTIFIC PROGRAM COMMITTEE

Stephen Nordlicht, M.D., New York City, Chairman
Martin Lipkin, M.D., New York City, Vice Chairman
Peter B. Farnsworth, M.D., Lake Success, Director, Division of Scientific Activities

ASSOCIATE CHAIRMEN

Thomas S. Bumbalo, M.D., Alden
Samuel Cytryn, M.D., Levittown
John Davis, M.D., Cooperstown
David Hirsh, M.D., Scarsdale
Joseph J. Kaufman, M.D., Canandaigua
Marilyn M. S. Kritchman, M.D., Great Neck
A. W. Martin Marino, Jr., M.D., Brooklyn
Maxwell Spring, M.D., Bronx

MEMBERS REPRESENTING SPECIALTY SOCIETIES

Alcoholism

Anne Geller, M.D., Brooklyn
American Medical Society on Alcoholism-New York Chapter

Allergy & Immunology

Charles H. Feldman, M.D., New York City

Anesthesiology

Kenneth Freese, M.D., East Meadow
New York State Society of Anesthesiologists

Cardiovascular Diseases

Mohammad Zahir, M.D., Brooklyn
New York Cardiological Society

Chest Diseases

Nael Martini, M.D., New York City
American College of Chest Physicians

Dermatology

Eugene Bodian, M.D., Great Neck
New York State Society of Dermatology

Emergency Medicine

Lorraine M. Giordano, M.D., Jamaica
New York Chapter of American College of Emergency Physicians

Family and General Practice

Mark Krotowski, M.D., Brooklyn
New York State Academy of Family Physicians

Hand Surgery

Thomas Palmieri, M.D., Brookville
New York Society for Surgery of the Hand

Internal Medicine

Sidney Weinstein, M.D., Rochester
New York State Society of Internal Medicine

Occupational & Environmental Health & Workers' Compensation

Gottfried F. H. Lehmann, M.D., Jamaica

Neurology

Richard Satran, M.D., Rochester

Neurosurgery

James L. Budny, M.D., Buffalo
New York State Neurosurgical Society, Inc.

Obstetrics and Gynecology

Richard Schwarz, M.D., Brooklyn
American College of Obstetricians and Gynecologists, District II

Ophthalmology

Samuel Packer, M.D., Manhasset
New York State Ophthalmological Society

Orthopedic Surgery

David Hirsh, M.D., Scarsdale
New York State Society of Orthopedic Surgeons

Otolaryngology

Steven Parnes, M.D., Albany
New York State Society of Otolaryngology—Head and Neck Surgery

Pathology

Nathan S. Taylor, M.D., Rochester
New York State Society of Pathologists

Pediatrics

Marvin Blumberg, M.D., Jamaica
American Academy of Pediatrics, District II

Physical Medicine and Rehabilitation

Samuel Sverdlik, M.D., New York City
New York Society of Physical Medicine and Rehabilitation

Psychiatry

Richard Drooz, M.D., New York City
New York State Psychiatric Association

Radiology

Rita Girolamo, M.D., Valhalla
New York State Chapter of the American College of Radiology

Surgery

Murry Friedman, M.D., New Hyde Park
New York State Society of Surgeons

Urology

Pellegrino Tozzo, M.D., New Rochelle
New York State Urological Society

Women's Medical Society of New York State

Jeanne Smith, M.D., New York City

FRIDAY, MARCH 7, 1986

Cardiovascular Diseases
Joint Meeting With
The New York Cardiological Society

10:00 A.M.–1:00 P.M.
BEEKMAN PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

CRITICAL CARE MANAGEMENT OF PATIENTS WITH CARDIOVASCULAR DISEASE

JAMES T. MAZZARA, M.D., New York City, *Moderator*

Director of Medicine and Chief of Cardiology, St. Vincent's Hospital and Medical Center of New York; Professor of Clinical Medicine, New York Medical College

Objective: Participants will be provided with an update on the Intensive Care Unit management of critically ill patients with cardiovascular disease. The educational program will include discussion on hemodynamic monitoring, cardiac arrhythmias, congestive heart failure and shock.

10:00 A.M. **Hemodynamic Monitoring: An Overview**

JAMES T. MAZZARA, M.D., New York City

10:30 A.M. **Cardiac Arrhythmias in the Critically Ill Patient**

DAVID A. RUBIN, M.D., Valhalla

Director, Clinical Electrophysiology, Westchester County Medical Center; Assistant Professor of Medicine, New York Medical College

11:00 A.M. **Management of the Patient with Severe Congestive Heart Failure**

MICHAEL V. HERMAN, M.D., Valhalla

Chief of Cardiology and Professor of Medicine, New York Medical College

11:30 A.M. **Management of the Patient in Shock**

THEODORE SCHREIBER, M.D., New York City

Co-Director, Coronary Care Unit; Assistant Professor of Medicine, New York Hospital—Cornell Medical Center

12:00 NOON–1:00 P.M. **Panel Discussion**

The Above Speakers

Alcohol/Gastroenterology
Joint Meeting With
American Medical Society on Alcoholism & Other Drug Dependencies—New York State Chapter

10:00 A.M.–1:00 P.M.
SUTTON PARLOR NORTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

ALCOHOLISM: DIAGNOSIS AND EFFECTS ON THE LIVER

Objective: This symposium will provide the practicing physican with a plan for diagnosis and early management of the alcoholic who has not yet admitted his or her diagnosis. It will also offer a framework for understanding and managing the consequences of the toxic effects of alcohol on the liver.

10:00 A.M. **Welcome and Opening Remarks**

ANNE GELLER, M.D., New York City

Chief, Smithers Alcoholism Treatment and Training Center of St. Luke's-Roosevelt Hospital Center; Assistant Professor of Clinical Medicine, Columbia University College of Physicians and Surgeons

10:10 A.M.–11:00 A.M. **ANNEGELLER, M.D., New York City,** *Moderator*

10:10 AM **Early Medical Diagnosis of Alcoholism**

ROBERT S. SIOUSSAT, M.D., Cooperstown

Director, Alcohol and Substance Abuse, Mary Imogene Bassett Hospital; Assistant Professor of Clinical Medicine, Columbia University College of Physicians and Surgeons

10:30 A.M. **Discussion**

10:35 A.M. **Techniques of Presenting the Diagnosis and Referral for Treatment**

MICHAEL I. REHMAR, M.D., New York City

Medical Director, General Electric Company, New York City area; Assistant Clinical Professor of Medicine, Cornell University Medical College

10:55 A.M. **Discussion**

(continued)

11:00 A.M.–12:00 NOON **JOHN S. DAVIS, M.D., Cooperstown,** *Moderator*

Associate Director, Medical Education, Mary Imogene Bassett Hospital; Associate Professor of Clinical Medicine, Columbia University College of Physicians and Surgeons

11:00 A.M. **The Clinical Spectrum of Alcoholic Liver Disease**

ARTHUR M. MAGUN, New York City

Assistant Gastroenterologist, Presbyterian Hospital; Assistant Professor of Medicine, Columbia University College of Physicians and Surgeons

11:20 A.M. **Discussion**

11:25 A.M. **Dealing with the Consequences of Portal Hypertension**

HENRY S. POHL, M.D., Albany

Attending Physician, Division of Gastroenterology, Albany Medical Center Hospital; Associate Professor of Clinical Medicine, Albany Medical College of Union University

11:45 A.M. **Specific and General Discussion**

12:00 NOON–1:00 P.M. **THE ALBERT F. R. ANDRESEN MEMORIAL LECTURE: Hepatic, Nutritional and Metabolic Disorders Associated with Alcohol**

CHARLES LIEBER, M.D., Bronx

Director, Alcoholism Research and Treatment Center, Veterans Administration Hospital, Bronx; Professor of Medicine and Pathology, Mount Sinai School of Medicine of the City University of New York

---

Obstetrics and Gynecology
Joint Meeting With
The American College of Obstetricians and Gynecologists, District II

10:00 A.M.–1:00 P.M.
SUTTON PARLOR CENTER

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

Material for this program was not received by the printing deadline date to appear in this preliminary program booklet. Program will appear in the Final Program Booklet published in March and distributed at the meeting.

Neurosurgery
Joint Meeting With
New York State Neurosurgical Society, Inc.

10:00 A.M.–1:00 P.M.
SUTTON PARLOR SOUTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

PEDIATRIC NEUROSURGERY

JAMES L. BUDNY, M.D., Buffalo, *Moderator*

Assistant Clinical Professor of Neurosurgery, State University of New York at Buffalo

Objective: To educate pediatricians, doctors of family practice, and neurosurgeons as to the up-to-date and current thinking relative to Hydrocephalus and head injuries in children.

10:00 A.M. **Neonatal Hydrocephalus: Common Shunt Problems**

JEFFREY WISOFF, M.D., New York City

Assistant Professor of Neurosurgery, New York University Medical Center

10:45 A.M. **Questions and Answers**

11:00 A.M. **Head Injuries in Pediatrics**

KENNETH SHAPIRO, M.D., Bronx

Associate Professor of Neurosurgery; Associate Professor of Pediatrics, Albert Einstein College of Medicine of Yeshiva University and Montefiore Hospital and Medical Center

11:45 A.M. **Questions and Answers**

12:00 NOON–1:00 P.M. **Open Papers**

JAMES L. BUDNY, M.D., Buffalo

(continued)

Physical Medicine and Rehabilitation
Joint Meeting With
New York Society of Physical Medicine and Rehabilitation

10:00 A.M.–1:00 P.M.
REGENT PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

THE PHYSIATRIC APPROACH TO PROBLEMS OF LIMITED MOBILITY AND LOCOMOTION

SAMUAL S. SVERDLIK, M.D., New York City, *Moderator*

Clinical Professor, New York Medical College, Valhalla; Director, Department of Rehabilitation Medicine, St. Vincent's Hospital and Medical Center of New York

Objective: The problems of limited mobility and locomotion are and will be among the costliest and frustrating challenges facing physicians into the 21st century. This symposium will describe methods of prevention, evaluation, treatment, resources, and devices to modify or reduce these disabilities.

10:00 A.M. **Evaluation**

EUGENE MOSKOWITZ, M.D., Valhalla

Clinical Professor, Rehabilitation Medicine, New York Medical College; Director, Department of Rehabilitation Medicine, Lawrence Hospital, Bronxville

10:35 A.M. **Questions and Answers**

10:45 A.M. **The Patient with Neuromuscular Disability**

AUDREYRANDOLPH, M.D., Valhalla

Associate Professor, Clinical Rehabilitation Medicine, New York Medical College; Director, Department of Rehabilitation Medicine, Westchester County Medical Center

11:20 A.M. **Questions and Answers**

11:30 A.M. **The Patient with Musculoskeletal Disability**

LAWRENCE FREEDMAN, M.D., Stony Brook

Professor, Rehabilitation Medicine, State University of New York at Stony Brook; Chairman, Department of Rehabilitation Medicine, Nassau County Medical Center

12:05 P.M. **Questions and Answers**

12:15 P.M. **The Geriatric Patient**

ASA RUSKIN, M.D., Bronx

Associate Clinical Professor, Rehabilitation Medicine, Albert Einstein College of Medicine of Yeshiva University; Director, Department of Rehabilitation Medicine, Kingsbrook Jewish Medical Center, Brooklyn

12:50 P.M.–1:00 P.M. **Questions and Answers**

Neurology

2:00 P.M.–5:00 P.M.
BEEKMAN PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

CURRENT AND FUTURE CARE OF THE EPILEPTIC PATIENT

RICHARD SATRAN, M.D., Rochester, *Moderator*

Professor and Acting Chairman, Department of Neurology, University of Rochester School of Medicine and Dentistry

Objective: To review recent advances in the diagnosis and treatment of epilepsy and relate these to the ongoing care of the epileptic patient.

2:00 P.M. **Epilepsy: The Perspective of the Patient and the Role of the Physician.**

DAVID WANG, M.D., Rochester

Assistant Professor of Neurology and Pediatrics, University of Rochester School of Medicine and Dentistry

2:45 P.M. **Newer Electroencephalographic Techniques in Epilepsy**

MARGARET MCBRIDE, M.D., Rochester

Assistant Professor of Pediatrics and Neurology, University of Rochester School of Medicine and Dentistry

3:30 P.M. **Anticonvulsant Drugs: Benefits, Interactions and Side Effects**

RONALD KRALL, M.D., Skokie, Illinois

Director of Clinical Research, Lorex Pharmaceuticals

4:15 P.M.-5:00 P.M. **The Role of Alcohol in Seizures and the Care of the Alcoholic Epileptic**

DAVID GREEN, M.D., Troy

Clinical Professor of Neurology, Albany Medical College of Union University

(continued)

Pediatrics/Alcoholism and Substance Abuse
Joint Meeting with
American Medical Society on Alcoholism & Other Drug Dependencies—New York State Chapter
New York State District II of the American Academy of Pediatrics

2:00 P.M.–5:00 P.M.
SUTTON PARLOR NORTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

ALCOHOLISM AND DRUG ABUSE IN CHILDHOOD AND ADOLESCENCE

MARVIN L. BLUMBERG, M.D., Jamaica, *Moderator*

Chairman, Department of Pediatrics, Jamaica Hospital; Associate Professor of Clinical Pediatrics, State University of New York at Stony Brook

and

ANNE GELLER, M.D., New York City, *Moderator*

Chief, Smithers Alcoholism Center of St. Luke's-Roosevelt Hospital Center; Clinical Associate Attending, Neurology, The Presbyterian Hospital

Objective: To make professionals aware of the extent and prevalance of these problems among children and adolescents; to be able to detect early physical and emotional signs and symptoms; and to treat or to refer patients to proper specialists and/or agencies.

2:00 P.M. **Risk Factors for Alcoholism in the Adolescent Population**

LYNN HENNECKE, Ph.D., New York City

Lecturer and Author

2:30 P.M. **Questions and Answers**

2:45 P.M. **Patterns of Drug Use and Abuse Among Adolescents**

ALLAN LANS, D.O., New York City

Associate Chief, Smithers Alcoholism Center of St. Luke's-Roosevelt Hospital Center

3:15 P.M. **Questions and Answers**

3:30 P.M. **Suicide and Drug Abuse Among Adolescents**

CYNTHIA R. PFEFFER, M.D., New York City

Assistant Attending, Psychiatry, New York Hospital, University Hospital, and Hospital of the Albert Einstein College of Medicine

4:00 P.M. **Questions and Answers**

4:15 P.M. **Munchausen's Syndrome by Proxy, Another Form of Child Abuse**

MARVIN L. BLUMBERG, M.D., Jamaica

Chairman, Committee on Child Abuse, Medical Society of the State of New York

4:45 P.M.–5:00 P.M. **Questions and Answers**

Chest Diseases
Joint Meeting With
American College of Chest Physicians, New York State Chapter

2:00 P.M.–5:00 P.M.
SUTTON PARLOR CENTER

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

THE STATE OF THE ART IN DIAGNOSIS AND TREATMENT OF BRONCHOGENIC CARCINOMA

NAEL MARTINI, M.D., New York City, *Moderator*

Chief, Thoracic Surgery, Memorial Sloan-Kettering Cancer Center; Professor of Surgery, Cornell University Medical College

Objective: To present the current modes of diagnosis and treatment of Bronchogenic Carcinoma.

(continued)

2:00 P.M. **Chest Imaging**

ROBERT HEELAN, M.D., New York City

Associate Attending Roentgenologist, Memorial Sloan-Kettering Cancer Center; Associate Professor of Radiology, Cornell University Medical College

2:30 P.M. **The 35th Howard Lilienthal Memorial Lecture: Lasers in Bronchogenic Carcinoma**

MICHAEL UNGAR, M.D., Philadelphia, Pennsylvania

Associate Professor, Clinical Medicine, University of Pennsylvania School of Medicine; Associate Chief, Pulmonary Division, Presbyterian University of Pennsylvania Medical Center

3:00 P.M. **Indications for Surgery in Lung Carcinoma**

LARRY R. KAISER, M.D., New York City

Assistant Attending Surgeon, Thoracic Service, Memorial Sloan-Kettering Cancer Center; Assistant Professor of Surgery, Cornell University Medical College

3:30 P.M. **The Role of Radiation Therapy in the Treatment of Lung Carcinoma**

HAROLD BRENNER, M.D., New York City

Associate Professor of Oncology, Tel Aviv University, Tel Aviv, Israel; Visiting Radiation Oncologist, Memorial Sloan-Kettering Cancer Center

4:00 P.M. **The Role of Chemotherapy In Non-Small Cell Lung Carcinoma**

RICHARD GRALLA, M.D., New York City

Associate Attending Physician, Department of Medicine, Memorial Sloan-Kettering Cancer Center; Associate Professor of Medicine, Cornell University Medical College

4:30 P.M.–5:00 P.M. **Discussion**

Occupational & Environmental Health/ (Incl. Workers Compensation)

2:00 P.M.–5:00 P.M.
SUTTON PARLOR SOUTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

ENVIRONMENTALLY RELATED ILLNESS: AN ASSESSMENT

GOTTFRIED F. H. LEHMANN, M.D., M.A., Jamaica, *Moderator*

Consultant in Occupational Medicine and Workers Compensation; Formerly, Medical Director, Long Island Railroad

Objective: This program will present an approach to the diagnosis of illness, related to the work place and environment. Some treatment modalities will be discussed. Prevention of environmental cancers improves with identification of risks.

2:10 P.M. **Environmental Medicine: Screening for Unrecognized Illness**

ROBERT McCUNNEY, M.D., M.P.H., Boston, Massachusetts

Corporate Medical Director, Cabot Corporation; Assistant Professor of Public Health, Boston University School of Medicine

3:00 P.M. **Questions and Answers**

3:10 P.M. **Current Status of Heavy Metal Poisoning**

PHILIP J. LANDRIGAN, M.D., M.Sc., D.I.H., New York City

Professor and Director of Environmental and Occupational Medicine, Mt. Sinai Medical Center; Past Director, Division of Surveillance, Hazard Evaluations and Field Studies (NIOSH, CDC)

4:00 P.M. **Questions and Answers**

4:10 P.M. **Assessment of Attributable Risks in Environmentally Related Cancers**

ARTHUR C. UPTON, M.D., New York City

Professor and Chairman, Department of Environmental Medicine, New York University Medical Center

4:50 P.M.–5:00 P.M. **Questions and Answers**

(continued)

Medical Liability Mutual Insurance Company
Joint Meeting With The
Medical Society of the State of New York

2:00 P.M.–5:00 P.M.
REGENT PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

WHAT MAKES A PATIENT SUE

RALPH M. SCHWARTZ, M.D., New York City, *Moderator*

Associate Professor, Clinical Obstetrics and Gynecology, State University of New York Downstate Medical Center; Chairman, Patient Safety Committee, Medical Liability Mutual Insurance Company

Objective: The purpose of this seminar is to provide physicians in attendance with information about professional liability occurrences in the practice of medicine, the attitudes of patients toward their physicians and the modus operandi of a lawsuit. It will deal with problems that appear with some frequency in the review of claims and lawsuits, in the identification of areas of possible liability and their prevention and acquainting the physician with the legal intricacies of medical liability activity. It is hoped that this meeting will help to improve patient safety and reduce the number of potential liability events.

2:00 P.M. **Legal Terminology and Principles of Medical Liability**

EDWARD J. AMSLER, J.D., Syracuse

Assistant Secretary, Medical Liability Mutal Insurance Company; Partner, Fager and Amsler

2:30 P.M. **Understanding the System: Procedures and Components of a Lawsuit**

DONALD J. FAGER, J.D., New York City

Vice President, Medical Liability Mutual Insurance Company; Partner, Fager and Amsler

3:00 P.M. **Improving Documentation**

RALPH M. SCHWARTZ, M.D., New York City

3:40 P.M. **Improving Patient Communications**

JOHN F. DWYER, M.D., New York City

Associate Professor, Clinical Obstetrics and Gynecology, New York Medical College; Perinatologist, Stamford Hospital Stamford, Connecticut

4:00 P.M. **What Makes a Patient Sue**

ANGELO FARACHI, Esq., Rochester

Partner, Farachi, Guadagnino, Lange, and Johns

4:30 P.M.–5:00 P.M. **The Physician As A Witness**

Panel Discussion—The Above Speakers

Computers in Medicine

2:00 P.M.–5:00 P.M.
MADISON SUITE

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

THE REALISTIC USES OF COMPUTERS IN MEDICAL PRACTICES

LAWRENCE B. TILIS, M.D., Pittsford, *Moderator*

President, American Physicians Association of Computer Medicine, Inc.

Objective: To educate physicians about the uses of computers in the practice of medicine by in-depth discussion and practical demonstrations.

2:00 P.M. **Medical Billing Systems—How to select them: Uses in Practice Management and Teaching**

LAWRENCE B. TILIS M.D., Pittsford

3:00 P.M. **Questions and Answers**

3:30 P.M. **Demonstration of ELIZA**

LAWRENCE B. TILIS, M.D., Pittsford

4:30 P.M. -5:00 P.M. **Questions and Discussion**

SATURDAY, MARCH 8, 1986

Otolaryngology
Joint Meeting With
New York State Society of Otolaryngology—Head & Neck Surgery

2:00 P.M.–5:00 P.M.
SUTTON PARLOR CENTER

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

THE EVALUATION AND MANAGEMENT OF TEMPORAL-MANDIBLE JOINT DISORDERS

BRUCE CAMPBELL, M.D., Manhasset, *Moderator*

Clinical Assistant Professor of Surgery (Otolaryngology), Cornell University Medical College; Associate Attending, Surgery (Otolaryngology), New York Eye & Ear Infirmary

Objective: This symposium provides the otolaryngologist, dentist, oral surgeon, and radiologist with the latest developments in the area of Temporal Mandible Joint.

2:10 P.M. **Diagnosis and Treatment of Temporal Mandible Joint Disorders**

MARK FRIEDMAN, D.D.S., Mount Vernon

3:00 P.M. **Questions and Answers**

3:10 P.M. **Radiological Evaluation of the Temporal Mandible Joint Problem**

ARNOLD BERRETT, M.D., New York City

Associate Clinical Professor of Radiology, New York Medical College, Valhalla

3:50 P.M. **Questions and Answers**

4:00 P.M. **Surgical Management of the Internal Derangement of the Temporal Mandible Joint**

Warren Sordill, D.M.D., Schenectady

Director of Dental Education, St. Clare's Hospital

4:50 P.M.–5:00 P.M. **Questions and Answers**

Computers in Medicine

10:00 A.M.–1:00 P.M.
MADISON SUITE

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

THE REALISTIC USES OF COMPUTERS IN MEDICAL PRACTICES

LAWRENCE B. TILIS, M.D., Pittsford, *Moderator*
President, American Physicians Association of Computer Medicine, Inc.

Objective: To educate physicians about the uses of computers in the practice of medicine by in-depth discussion and practical demonstration. No knowledge of computers is required.

10:00 A.M. **Medical Record Keeping and the Computer: Costs, Problems and Solutions**

LAWRENCE B. TILIS, M.D., Pittsford

11:00 A.M. **Questions and Answers**

11:30 A.M. **Telephone Communications Accessing Databases**

WILLIAM K. WEISSMAN, M.D., New York City

12:45 P.M.–1:00 P.M. **Questions and Answers**

(continued)

Psychiatry
Joint Meeting With
New York State Psychiatric Association

10:00 A.M.–5:00 P.M.
REGENT PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 6 Hours, Category 1; AAFP, 6 Hours, Prescribed

PROGRESSIVE PSYCHIATRY NOW AND FOR THE TWENTY-FIRST CENTURY

RICHARD B. DROOZ, M.D., New York City, *Moderator*

Associate Professor of Psychiatry (Ret.), State University of New York Downstate Medical Center; Attending Psychiatrist (Ret.), Kings County Hospital Center, Brooklyn

Objective: To present a comprehensive, modern view of psychiatric issues in four major areas of outstanding current interest: Geriatric psychiatry, forensic psychiatry, schizophrenia, and future directions of psychiatry

10:10 A.M. **An Overview of Geriatric Psychiatry**

FREDERICK B. E. CHARATAN, M.D., Amityville

Clinical Director, South Oaks Hospital

10:50 A.M. **Clinical and Ethical Issues in the Operation of a Psychiatric Geriatric Program**

HARVEY BLUESTONE, M.D., Bronx

Professor of Psychiatry, Albert Einstein College of Medicine of Yeshiva University; Director of Psychiatry, Bronx-Lebanon Hospital Center

11:30 A.M. **Mania In the Elderly**

JOHN S. MCINTYRE, M.D., Rochester

Chairperson and Attending, Division of Psychiatry, St. Mary's Hospital; President, New York State Psychiatric Association

12:10 P.M.–1:00 P.M. **The Future Directions of Psychiatry in New York State**

STEVEN E. KATZ, New York City

New York State Commissioner of Mental Health; Professor of Clinical Psychiatry, New York University School of Medicine

2:00 P.M.–5:00 P.M.

2:00 P.M. **A History of the Treatment of Schizophrenia: The Lessons to be Learned**

LESTER E. SHAPIRO, Rockville Centre

Associate Professor of Psychiatry, School of Health Sciences, State University of New York at Stony Brook; Chairperson, Joint Committee on Schizophrenia, New York State Psychiatric Association

2:35 P.M. **Psychiatry and the Elderly in the Criminal Justice System**

NAOMI GOLDSTEIN, M.D., New York City

Clinical Associate Professor of Psychiatry, New York University School of Medicine; President, New York County District Branch, American Psychiatric Association

3:10 P.M. **Civil Issues in Psychiatry of the Elderly**

J. RICHARD CICCONE, M.D., Rochester

President-elect, American Academy of Psychiatry and the Law; Associate Professor of Psychiatry, University of Rochester School of Medicine and Dentistry

3:45 P.M. **The Complexities of Medicating Elderly Psychiatric Patients**

WILLIAM J. GERSHELL, M.D., New York City

Assistant Clinical Professor of Psychiatry, Mount Sinai School of Medicine of the City University of New York; President, New York Group for Geriatric Psychiatry

4:20 P.M.–5:00 P.M. **Suicide of the Elderly: Recognition and Liability**

HAROLD E. BERSON, M.D., Brooklyn

Attending Psychiatrist, Maimonides Medical Center; Attending Neuropsychiatrist, Interfaith Medical Center

STANLEY J. SCHER, L.L.B., New York City

Senior Partner, Garbarini, Scher, and DeCicco, P.C., specializing in medical malpractice litigation; Faculty, New York State Trial Lawyers Association

(continued)

Urology
Dermatology
Joint Meeting With
New York State Urological Society
and
New York State Society of Dermatology

10:00 A.M.–1:00 P.M.
SUTTON PARLOR CENTER

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

LESIONS OF THE MALE GENITALIA AS SEEN THROUGH THE EYES OF THE DERMATOLOGIST AND UROLOGIST

PELLEGRINO TOZZO, M.D., New Rochelle, *Moderator*

Chief, Urology, New Rochelle Hospital Medical Center; Associate Professor of Urology, New York Medical College

EUGENE L. BODIAN, M.D., Great Neck, *Moderator*

Associate Professor of Dermatology, New York University School of Medicine; Chief, Dermatology, North Shore University Hospital; President, New York State Society of Dermatology

Objective: A joint symposium of problems commonly seen by urologists and dermatologists. Clues to diagnosis and new therapeutic approaches, old diseases, as well as the current state of Acquired Immune Deficiency Syndrome will be discussed, including medical, surgical and laser.

10:00 A.M. **Common and Not So Common Lesions of the Scrotum**

EUGENE L. BODIAN, M.D., Great Neck

10:25 A.M. **Cancer of the Penis**

WILLIAM R. FAIR, M.D., New York City

Professor and Director, Urology Department Memorial Sloan-Kettering Cancer Center

10:50 P.M. **Questions and Answers**

11:00 A.M. **Skin Manifestations of Acquired Immune Deficiency Syndrome**

N. PATRICK HENNESSEY, M.D., New York City

Clinical Instructor, Department of Dermatology, New York University School of Medicine

11:25 A.M. **Treatment of Sexually Transmitted Diseases of the Urogenital Tract**

GEORGE OWENS, M.D., Valhalla

Assistant Professor of Urology, New York Medical College

11:50 A.M. **Questions and Answers**

12:00 NOON **Dermatologic Problems of the Male Genitalia Amenable to Laser Therapy**

ROY G. GERONEMUS, M.D., New York City

Chief, Surgery and Laser-Skin and Cancer Clinic, New York University School of Medicine

12:25 P.M. **Laser Therapy of Urogenital Disorders**

ROBERT HUBEN, M.D., Buffalo

Chief, Urologic Oncology, Roswell Park Memorial Institute

12:50 P.M.–1:00 P.M. **Questions and Answers**

(continued)

Hand Surgery/Anesthesiology
Joint Meeting With
The New York Society for Surgery of the Hand/NYS Society of Anesthesiologists

10:00 A.M.–5:00 P.M.
SUTTON PARLOR SOUTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 6 Hours, Category 1; AAFP, 6 Hours, Prescribed

UPDATE ON PRIMARY HAND CARE

THOMAS J. PALMIERI, M.D., New Hyde Park, *Moderator*

Assistant Professor of Clinical Surgery, State University of New York at Stony Brook; Physician-in-Charge, Hand Service, Long Island Jewish-Hillside Medical Center

Objective: This course is designed to provide the primary care physicians, orthopedic, plastic, and general surgeons, as well as nurses, therapists, and paramedical personnel with an update on the principles of hand surgery care: including the recognition and diagnosis of various diseases entities; and the latest information on the medical and surgical opinions for treatment, both initial and definitive.

10:00 A.M. **Examination of the Hand: Latest Techniques**

MARTIN A. POSNER, M.D., New York City

Chief, Hand Surgery, Lenox Hill Hospital, Mount Sinai Hospital, and Hospital for Joint Diseases; Consultant, Hand Surgery, Blythedale Children's Hospital, Valhalla, and City Hospital Center at Elmhurst

10:30 A.M. **Potential Pitfalls and Complications of Primary Care of the Hand**

JOSEPH LEDDY, M.D., New Brunswick, New Jersey

Clinical Associate Professor of Surgery, Division of Orthopedics, Department of Surgery; Director, Hand Surgery (Orthopedics), University of Medicine and Dentistry of New Jersey—Rutgers Medical School; Chief, Division of Orthopedics; Chief, Hand Surgery, St. Peter's Medical Center

11:00 A.M. **Latest Techniques and Drugs for Regional Anesthesia in Hand Surgery**

FARROKH R. MANEKSHA, M.D., Stony Brook

Clinical Assistant Professor of Anesthesia, State University of New York at Stony Brook; Director, Pain Center, University Hospital at Stony Brook

11:30 A.M. **Current Concepts in the Use of Prosthetics for Upper Extremity Amputations**

ROBERT W. BEASLEY, M.D., New York City

Professor of Surgery (Plastic), New York University School of Medicine; Director of Hand Surgery Service, Institute of Reconstructive Plastic Surgery, New York University Medical Center

12:00 NOON **Current Concepts in the Diagnosis and Treatment of Burns of the Hand and Wrist**

LESTER SILVER, M.D., New York City

Chief of Pediatric Plastic & Reconstructive Surgery; Attending, Plastic Surgery, Beth Israel Medical Center; Associate Professor of Clinical Surgery (Plastic), Mount Sinai School of Medicine of the City University of New York

12:30 P.M.–1:00 P.M. **Common Hand Tumors**

THOMAS J. PALMIERI, M.D., New Hyde Park

2:00 P.M. **Treatment of Skin Loss in the Hand**

JAMES B. MASSENGILL, M.D., Morristown, New Jersey

Associate Clinical Professor of Surgery; Chief, Division of Hand Surgery, Department of Surgery, Section of Orthopedic Surgery, University of Medicine & Dentistry of New Jersey, New Jersey Medical School

2:30 P.M. **Tendon Injuries—Primary and Secondary Treatment**

ROBERT SCHULTZ, M.D., Valhalla

Professor and Chairman, Department of Orthopedics; Director, Sylvester Carter Hand Service, New York Medical College; Director, Orthopedic Surgery, Westchester County Medical School

3:00 P.M. **Latest Concepts in the Treatment of Wrist Fractures and Dislocations**

CHARLES MELONE, Jr., M.D., New York City

Clinical Associate Professor of Orthopedic Surgery, New York University Medical Center; Director, Hand Surgery Service, Cabrini Medical Center

3:30 P.M. **Nerve Injuries: Primary versus Secondary Treatment**

STEVEN GREEN, M.D., New York City

Associate Attending Physician, Hospital for Joint Diseases, Orthopedic Institute, Assistant Clinical Professor of Orthopedic Surgery, Mount Sinai School of Medicine of the City University of New York

4:00 P.M. **Compression Neuropathies of the Upper Extremity and the Use of Electrodiagnostic Techniques**

CLAYTON A PEIMER, M.D., Buffalo

Associate Professor of Orthopedic Surgery; Chief, Division of Hand Surgery, Department of Orthopedic Surgery, Erie County Medical Center and Millard Fillmore Hospital

4:30 P.M.–5:00 P.M. **Early Treatment of Arthritis of the Hand and Wrist**

CHITRANJAN RANAWAT, M.D., New York City

Associate Attending, Orthopedic Surgery, The Manhattan Hospital for Special Surgery; Attending, Orthopedic Surgery, New York Hospital

(continued)

Internal Medicine
Joint Meeting With
New York Society of Internal Medicine

10:00 A.M.–5:00 P.M.
SUTTON PARLOR NORTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 6 Hours, Category 1; AAFP, 6 Hours, Prescribed

PLAYING THE ODDS: THE APPLICATION OF QUANTITATIVE CLINICAL DECISION MAKING TO CLINICAL MEDICINE

SIDNEY S. WEINSTEIN, M.D., Rochester, *Moderator*

Clinical Associate Professor of Medicine, University of Rochester School of Medicine and Dentistry

Objective: An introduction to newer techniques of clinical decision making. These papers are designed to assist us in better and more efficient patient work-ups, as well as to make our decisions more cost effective.

10:00 A.M. **Introduction to Quantitative Clinical Decision Making**

JEROME KASSIRER, M.D., Boston, Massachusetts

Professor of Medicine, Tufts University School of Medicine; Associate Chairman, Department of Medicine, New England Medical Center Hospital

10:45 A.M. **Selecting and Interpreting Diagnostic Tests: How to Do it With and Without the Computer**

ROBERT PANZER, M.D., Rochester

Assistant Professor of Medicine, University of Rochester School of Medicine and Dentistry

11:45 A.M. **Cost Effectiveness for the Clinician: What to Know and When to Care**

ALBERT MULLEY, M.D., Boston, Massachusetts

Assistant Professor of Medicine, Harvard Medical School; Chief, General Internal Medicine Unit, Massachusetts General Hospital

12:30 P.M.–1:00 P.M. **Qualitative and Ethical Issues in Quantitative Clinical Decision Making**

SAUL MOROFF, M.D., Bronx

Associate Professor of Medicine, Albert Einstein College of Medicine of Yeshiva University

2:00 P.M.–5:00 P.M.

2:00 P.M. **Ventilation/Perfusion Lung Scans in Pulmonary Embolism**

RAYMOND MAYEWSKI, M.D., Rochester

Associate Professor of Medicine, University of Rochester School of Medicine and Dentistry

2:35 P.M. **Diagnostic Decision: Diagnostic Tests in Breast Cancer**

ALVIN MUSHLIN, M.D., Rochester

Associate Professor of Medicine, University of Rochester School of Medicine and Dentistry

3:10 P.M. **An examination of the Evidence for the Treatment of Mild Hypertension**

MICHAEL ALDERMAN, M.D., Bronx

Professor and Chairman, Department of Epidemiology and Social Medicine, Albert Einstein College of Medicine of Yeshiva University

3:45 P.M. **Prognostic Stratification in Patients with Pneumonia**

EDGAR BLACK, M.D., Rochester

Assistant Professor of Medicine, University of Rochester School of Medicine and Dentistry

4:20 P.M.–5:00 P.M. **Panel Discussion**

Audience Participation

Ophthalmology
Joint Meeting With
New York State Ophthalmological Society

10:00 A.M.–5:00 P.M.
BEEKMAN PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 6 Hours, Category 1; AAFP, 6 Hours, Prescribed

MANAGEMENT OF OCULAR TUMORS BY THE PRACTICING OPHTHALMOLOGIST

SAMUEL PACKER, M.D., Manhasset, *Moderator*

Associate Professor of Clinical Ophthalmology and Radiology, Cornell University Medical College; Chief, Division of Ophthalmology, North Shore University Hospital

Objective: To identify those ocular tumors that should be managed without consultation with an ophthalmic oncologist.

(continued)

10:00 A.M. **Introduction**

SAMUEL PACKER, M.D., Manhasset

10:15 A.M. **Tumors of Eyelid**

ALBERT HORNBLASS, M.D., New York City

Chief, Ophthalmic Plastic and Reconstructive Surgery, Manhattan Eye, Ear and Throat Hospital; Clinical Professor of Ophthalmology, State University of New York Downstate Medical Center

10:30 A.M. **Panel Discussion**

DRS. HORNBLASS, GARBER, AND JAKOBIEC

11:00 A.M. **Tumors of the Conjunctiva**

FREDERICK A. JAKOBIEC, M.D., New York City

Professor of Clinical Ophthalmology and Pathology, Columbia University College of Physicians and Surgeons

11:20 A.M. **Panel Discussion**

DRS. JAKOBIEC, ABRAMSON, and R. RODRIQUEZ-SAINS

11:45 P.M. **Tumors of the Iris**

FREDERICK JAKOBIEC, M.D., New York City

12:15 P.M. **Panel Discussion**

DRS. JAKOBIEC, ABRAMSON, and R. RODRIQUEZ-SAINS

12:30 P.M. **Dysplastic Nevus Syndrome**

RENE RODRIQUEZ-SAINS, M.D., New York City

Associate Attending Surgeon, Manhattan Eye, Ear and Throat Hospital

12:45 P.M.–1:00 P.M. **Panel Discussion**

DRS. JAKOBIEC, ABRAMSON, and R. RODRIQUEZ-SAINS

2:00 P.M.–5:00 P.M.

2:00 P.M. **Choroidal Melanoma**

SAMUEL PACKER, M.D., Manhasset

2:20 P.M. **Panel Discussion**

DRS. JAKOBIEC, ABRAMSON, and R. RODRIQUEZ-SAINS

2:40 P.M. **Pediatric Ocular Tumors**

DAVID ABRAMSON, M.D., New York City

Clinical Professor of Ophthalmology, Cornell University Medical College

3:00 P.M. **Panel Discussion**

DRS. ABRAMSON, JAKOBIEC and HAIK

3:20 P.M. **Orbital Tumors**

BARRETT G. HAIK, M.D., New York City

Associate Professor of Clinical Ophthalmology, Cornell University Medical College

3:40 P.M. **Panel Discussion**

DRS. HAIK, ABRAMSON, and JAKOBIEC

4:10 P.M. **Evaluation and Treatment of Orbital Infection**

PERRY GARBER , M.D., Manhasset

Attending, Ocular Plastics Service, North Shore University Hospital; Physician, Long Island Jewish Hillside Medical Center

4:30 P.M.–5:00 P.M. **Panel Discussion**

DRS. GARBER and HORNBLASS

SUNDAY, MARCH 9, 1986

Allergy and Immunology

10:00 A.M.–1:00 P.M.
SUTTON PARLOR NORTH

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

CURRENT PERSPECTIVES IN ALLERGY AND IMMUNOLOGY

CHARLES H. FELDMAN, M.D., New York City, *Moderator*

Assistant Professor of Clinical Pediatrics, Columbia University College of Physicians and Surgeons; Director of Asthma Program, St. Mary's Hospital for Children, Bayside

Objective: This program will provide the specialist and the primary care physician with clinical updates on common problems in the field of allergy and immunology. The presentations will focus on the diagnosis and treatment of conditions frequently seen in the office setting.

(continued)

10:00 A.M. **Insect Allergy**

William M. Nicholas, M.D., New Hyde Park

Attending-in-Charge, Division of Allergy, Long Island Jewish Hillside Medical Center; Assistant Clinical Professor of Medicine, Columbia University College of Physicians and Surgeons

10:30 A.M. **Questions and Answers**

10:40 A.M. **Chronic Rhinitis**

John T. Connell, M.D., Englewood, New Jersey

Director, Nasal Diseases Study Center, Holy Name Hospital, Teaneck, New Jersey

11:10 A.M. **Questions and Answers**

11:20 A.M. **Long-Term Therapy of Asthma**

William J. Davis, M.D., New York City

Associate Professor of Clinical Pediatrics, Columbia University College of Physicians and Surgeons; Director, Division of Allergy, Department of Pediatrics, Columbia Presbyterian Medical Center

11:50 A.M. **Questions and Answers**

12:00 NOON **Office Testing of Pulmonary Function in Asthmatic Children**

Anthony L. Mansell, M.D., New York City

Associate Professor of Clinical Pediatrics, Columbia University College of Physicians and Surgeons; Director, Pediatric Pulmonary Laboratory, Columbia Presbyterian Medical Center

12:20 P.M. **Questions and Answers**

12:30 P.M. **Acquired Immune Deficiency Syndrome**

Michael H. Grieco, M.D., New York City

Director, R. A. Cook Institute of Allergy, St. Luke's-Roosevelt Hospital Center; Professor of Clinical Medicine, Columbia University College of Physicians and Surgeons

12:50 P.M.–1:00 P.M. **Questions and Answers**

General Surgery
Colon and Rectal Surgery
Joint Meeting With
New York State Society of Surgeons

***9:00 A.M.–1:00 P.M.**
BEEKMAN PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 4 Hours, Category 1; AAFP, 4 Hours, Prescribed

(1) SURGONOMICS: THE NEW ECONOMICS OF MEDICAL CARE
(2) COLONOSCOPY: 1986 - WHEN? WHERE? WHY? HOW?

Objective: (1) To introduce to physicians the change underway in medical financing that will effect their practice in medicine.
(2) This program will define current status of colonoscopy with emphasis on WHEN it is indicated, WHY it is the procedure of choice and HOW it should be performed

9:00 A.M.–10:45 A.M. **Murry Friedman, M.D., New Hyde Park,** *Moderator*

Representative, New York State Society of Surgeons, Scientific Program Committee, Medical Society of the State of New York; Attending Physician, Surgery, Long Island Jewish Hillside Medical Center, North Shore Hospital, Manhasset, and St. Francis Hospital, Roslyn

9:00 A.M. **The New Economics of Medical Care**

Eric Munoz, M.D., M.B.A., New Hyde Park

Head, Research Division, Department of Surgery, Long Island Jewish Hillside Medical Center

9:30 A.M. **The Shift from Voluntary to Proprietary Medicare**

Fred Goldman, Ph.D., New York City

Chairman, Health Services, The New School for Social Research

10:00 A.M. **Cost Containment and Quality Assurance: An Employer's Perspective**

Stephen Rosenberg, M.D., New York City

Associate Professor of Clinical Public Health (Health Administration), Columbia University College of Physicians and Surgeons; Medical Consultant, New York City Employee Benefits Program

10:30 A.M. **Panel Discussion**

Drs. Munoz, Goldman and Rosenberg

10:50 A.M.–1:00 P.M. **A. W. Martin Marino Jr., M.D., Brooklyn,** *Moderator*

10:50 A.M. **Colonoscopy: Origins and Rites of Passage**

William I. Woolf, M.D., New York City

Former Director of Surgery, Beth Israel Medical Center; Professor, Clinical Surgery, Mt. Sinai School of Medicine of the City University of New York

11:00 A.M. **The Polyp: Cancer Sequence, Early Detection, and Prudent Surveillance of the High Risk Colon**

Kenneth A. Forde, M.D., New York City

Professor of Clinical Surgery, Columbia University College of Physicians and Surgeons; Attending Surgeon, Presbyterian Hospital

(continued)

11:10 A.M. **Surgical Pathology**

BARTON HOEXTER, M.D., Great Neck

Clinical Associate Professor of Surgery, Department of Surgery, Cornell University Medical College; Chief, Department of Colon Rectal Surgery, North Shore Hospital, Manhasset

11:20 A.M. **Limitations and Complications**

RICHARD M. BURG, M.D., White Plains

Attending, Colon Rectal Surgery, White Plains Hospital and St. Agnes Hospital; Fellow, American College of Surgeons

11:30 A.M. **Panel Discussion**

DRS. WOOLF, FORDE, HOEXTER, AND BURG

11:45 A.M. **Indications and Contraindications**

DRISS HASSAN, M.D., Plattsburgh

Fellow, American Society of Colon and Rectal Surgery; Attending, General Surgery, Champlain Valley Physicians Hospital Medical Center

11:55 A.M. **Technique and Training**

VINCENT A. DEGENNARO, M.D., New York City

Chief, Surgical Education, St. Vincent's Hospital and Medical Center of New York; Assistant Professor of Surgery, New York Medical College

12:05 P.M. **Cost, Time, Clean-up, and Fees**

A. W. MARTIN MARINO, JR., M.D., Brooklyn

Clinical Professor of Surgery, State University of New York Downstate Medical Center; Chief, Colon and Rectal Surgery, The Brooklyn Caledonian Hospital

12:15 P.M. **Panel Discussion**

DRS. HASSAN, DEGENNARO, MARINO, AND HOEXTER

12:30 P.M.–1:00 P.M. **Video Tape Presentation**

ANNUAL MEETING

Women's Medical Society of New York State/Family Practice/Emergency Medicine
Joint Meeting With
New York Chapter of the American College of Emergency Physicians and New York State Academy of Family Physicians

10:00 A.M.–1:00 A.M.
REGENT PARLOR

CONTINUING MEDICAL EDUCATION CREDIT:
AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

CLINICAL PROBLEMS IN FAMILY PRACTICE AND EMERGENCY MEDICINE

MARK H. KROTOWSKI, M.D., Brooklyn, *Moderator*

Clinical Assistant Professor of Family Practice, State University of New York Downstate Medical Center

Objective: This program is meant to familiarize the family physician and the emergency medicine physician with areas of clinical medicine which confront the primary care physician almost daily.

10:00 A.M. **Update on Management of Type II Diabetes: The Second Generation Oral Agents**
Sheldon Bleicher, M.D., Brooklyn

Professor of Medicine, State University of New York Downstate Medical Center; Director of Medicine, Brooklyn Caledonian Hospital

10:30 A.M. **Controversies in the Etiology and Presentation of Ectopic Pregnancies**

THOMAS KWIATOWSKI, M.D., New York City

Physician-in Charge, Emergency Services, Long Island Jewish Hillside Medical Center

10:30 A.M.–11:30 A.M. **LORRAINE GIORDANO, M.D., Jamaica Estates,** *Moderator*

Fellow, American College of Emergency Physicians; Fellow, American Academy of Family Physicians

11:00 A.M. **New York State Emergency Medical System Development: Implications for Patients and Physicians**

MARK HENRY, M.D., Flushing

Chairman, New York State Emergency Medical Services Council; Director, Department of Emergency Medicine, Booth Memorial Medical Center

11:30 A.M.–1:00 P.M. **JEANNE SMITH, M.D., New York City,** *Moderator*

Fellow, American Academy of Pediatrics and American Academy of Psychoanalysis

(continued)

11:30 A.M. **Pattern of Violent Deaths in Women: Pathologic Findings**

NAFEES K. PERVEZ, M.D., New York City

Assistant Professor, Clinical Pathology, Mount Sinai School of Medicine of the City University of New York

12:00 NOON **The Single Parent Women Physician**

RITA ROSLYN NEWMAN, M.D., New Jersey

Attending in Psychiatry, Saint Barnabas Medical Center, Livingston, New Jersey

12:30 P.M.–1:00 P.M. **Exercise Today for Fitness Tomorrow: Pediatric Sports Medicine**

ELIZABETH CORYLLOS, M.D., Glen Head

Fellow, American Academy of Pediatrics; Fellow, American College of Surgeons

---

Pathology
Joint Meeting With
New York State Society of Pathologists

10:00 A.M.–1:00 P.M.
SUTTON PARLOR CENTER

CONTINUING MEDICAL EDUCATION CREDIT: AMA, PRA, 3 Hours, Category 1; AAFP, 3 Hours, Prescribed

DIAGNOSTIC TESTING IN THE PHYSICIAN'S OFFICE: OPPORTUNITIES AND PITFALLS

ROBERT BRECKENRIDGE, M.D., Elmer, New Jersey, *Moderator*

President, College of American Pathologists; Director of Laboratories, Elmer Community Hospital

Objective: To evaluate the role of laboratory testing in the physician's office from the perspectives of patient care, quality control, technology regulations, and economics

10:00 A.M. **Introduction**

ROBERT L. BRECKENRIDGE, M.D., Elmer, New Jersey

10:15 A.M. **Economic and Reimbursement Aspects of Office Testing**

DAVID TIERSTEN, M.D., Hackensack, New Jersey

Director of Laboratories, Hackensack Medical Center; Past President, New York State Society of Pathologists

10:45 A.M. **The Technology of Office Testing: Present and Future**

PETER J. HOWANITZ, M.D., Los Angeles, California

Associate Director of Laboratories, UCLA Center for Health Sciences; Chairman, Quality Assurance Service Committee, College of American Pathologists

11:30 A.M. **Quality Assurance, Licensure and Regulation of the Office Laboratory**

PAUL BACHNER, M.D., Port Chester

Chairman, Pathology and Laboratory Medicine, United Hospital; State Commissioner, Laboratory Accreditation Program, College of American Pathologists

12:00 NOON **The Clinical Perspective for Office Testing**

S. RAYMOND GAMBINO, M.D., Teterboro, New Jersey

Executive Vice President and Chief Medical Officer, Met Path; Adjunct Professor of Pathology, Columbia University College of Physicians and Surgeons

12:45 P.M.–1:00 P.M. **Discussion**

1986 ANNUAL CME ASSEMBLY

TECHNICAL EXHIBITS

RHINELANDER GALLERY
NEW YORK HILTON

Accumedic Computer Systems, Inc. New York, New York	Booth 46
AIS Corporation Bridgewater, New Jersey	Booth 30
American Physicians Service and Supply Edison, New Jersey	Booth 29
Asset and Financial Planning, Ltd. Poughkeepsie, New York	Booth 54
Boehringer Ingelheim Pharmaceuticals Ridgefield, Connecticut	Booth 40
Boots Pharmaceuticals Shreveport, Louisiana	Booth 50
Brentwood Instruments Torrance, California	Booth 45
Burlington Systems Burlington, Vermont	Booth 47
Carann Leasing Corp. Massapequa Park, New York	Booth 62
CIBA Pharmaceuticals Parsippany, New Jersey	Booth 55
CPI Pension Services White Plains, New York	Booth 57
Damon Clinical Laboratories Needham Heights, Massachusetts	Booth 43
Empire Blue Cross & Blue Shield New York, New York	Booth 41
Employee Leasing of New York New York, New York	Booth 53
Encyclopaedia Britannica USA Chicago, Illinois	Booth 52
First Investors Equity Management New York, New York	Booth 3
Gibraltar Securities Florham Park, New Jersey	Booth 56
Group Health, Inc. New York, New York	Booth 44
Healthcare Billing Systems, Inc. New City, New York	Booth 36
Hoechst-Roussel Pharmaceuticals, Inc. Somerville, New Jersey	Booth 38
International Preferred Assets Coppell, Texas	Booth 59
Knoll Pharmaceutical Company Whippany, New Jersey	Booth 1
Libin and Associates Bedford Hills, New York	Booth 24
Marketing Communications, Inc. Brooklyn, New York	Booth 25
Mead Johnson Nutritional Division Evansville, Indiana	Booth 26
Medical Business Services New York, New York	Booths 4 & 5
Medical Liability Mutual Insurance Co. **Donald J. Fager & Assoc., Inc.** New York, New York	37
Medical Quadrangle New York, New York	Booth 15
Merrill Lynch Pierce Fenner & Smith Garden City, New York	Booth 33
Metropolitan Life Insurance Co. New York, New York	Booth 23
Mutual Association of Professional Services New York, New York	Booth 60

CONTINUED NEXT PAGE

Medical Society of the
State of New York

Annual Dinner Dance

in honor of
Daniel F. O'Keeffe, M.D.
President

Thursday
March 6, 1986
Trianon Ballroom
The New York Hilton
53rd Street & Avenue of Americas

Cocktail Hour
7 P.M.

Superlative
Dinner

Subscription $75.00 per person

Reservation Form

Make check payable to:
Medical Society of the State of New York
420 Lakeville Road
Lake Success, New York 11042

Please send me _______ tickets for the
Annual Dinner Dance on March 6, 1986
Check enclosed for $_________

PLEASE NOTE: Reservations and ticket sales will close at 12 Noon on Thursday, March 6, one hour before the House Convenes.

Name______________________________

Address____________________________

_________________Zip_______________

Park Surgical Co. Brooklyn, New York	**Booth 42**
Physicians Reciprocal Insurers Manhasset, New York	**Booth 39**
Property Investment Corp. Newark, New Jersey	**Booth 19**
S. A. Ramirez & Co., Inc. New York, New York	**Booth 35**
Real-Time Management, Inc. Bridgeport, Connecticut	**Booths 31 & 32**
Roche Laboratories Nutley, New Jersey	**Booth 34**
W. B. Saunders Co. Upper Montclair, New Jersey	**Booth 22**
Schering Laboratories Matawan, New Jersey	**Booth 51**
Selective Leasing Co. East Brunswick, New Jersey	**Booth 21**
Charles J. Sellers Company, Inc. Athol Springs, New York	**Booth 58**
SenTech Medical Corporation Arden Hills, Maine	**Booth 27**
The Upjohn Company Garden City, New York	**Booth 49**
U.S. Army Medical Department Washington, D.C.	**Booth 14**
USV Laboratories Tarrytown, New York	**Booth 48**
Whittman Burke Refining Hawthorne, New Jersey	**Booth 28**

■

Note: Exhibitors who are not listed here will appear in the final program book which will be distributed at the CME Assembly registration desk during the meetings.

Central Park Lake

Washington Square Arch

COURTESY N.Y. CONVENTION & VISITORS BUREAU

Advance Registration Form

1986 Annual CME Assembly, March 7–9
The New York Hilton, New York City

Complete and return this form to the Medical Society address as indicated below.

Name______________________________
PLEASE PRINT *LAST* *FIRST*

Address______________________________

City/State/Zip______________________________

Medical Specialty______________________________

REGISTRATION FEE *Please check categories*

☐ Non-Member Physician*	$50.00
☐ Allied Profession	$10.00

* of any state medical society

- ☐ Nurse
- ☐ Physician Assist.
- ☐ Dentist
- ☐ Lab Technician
- ☐ PT/OT
- ☐ Other__________ *Specify*

NO FEE *Please check category*

- ☐ MSSNY Active Member*
- ☐ MSSNY Life Member
- ☐ Resident/Intern
- ☐ Medical Students
- ☐ Medical Assist.
- ☐ Speaker
- ☐ Spouse/Family
- ☐ News Media/Medical Writer

REMITTANCE ENCLOSED $__________

Please make check payable to:
Medical Society of the State of New York, and return this form with remittance to: Division of Meeting Services/MSSNY, 420 Lakeville Rd., P.O. Box 5404, Lake Success, N.Y. 11042.

Important: Your ID Badge will be held at the MSSNY Registration Desk located in the Exhibit Hall, on the second floor of the New York Hilton.

DOCTOR:

is your Medical Assistant keeping in step with you?

As medical practice becomes more complicated and more highly specialized, you need more highly trained medical assistants in your office.

Membership in the AMERICAN ASSOCIATION OF MEDICAL ASSISTANTS will help your assistants keep up-to-date and informed of new ideas and techniques. AAMA's continuing education program offers workshops and seminars that will enhance the professionalism of your office employees.

As the first professional organization for medical assistants (founded 1956), AAMA pioneered in developing the only certification program in this field. A medical assistant who successfully completes the examination is identified as a Certified Medical Assistant (CMA). More than 8,000 certificates have been earned since the first examination was given in 1963.

The AAMA pioneered in the development of curriculum standards for medical assisting programs. The American Medical Association, in collaboration with AAMA, is recognized as an official accrediting agency for such programs by the U.S. Office of Education.

On five different occasions the AMA House of Delegates has passed resolutions commending the objectives of the AAMA, endorsing its functions, and urging every physician to encourage medical assistants to join the association in order to benefit from its educational programs.

VISIT OUR EXHIBIT AT
THE CME ASSEMBLY

To help your medical assistants do a better job of helping you, urge them to join AAMA—the professional association dedicated to their continuing education. Fill in the attached coupon and mail it today. Your practice deserves the best!

I wish to inquire about membership for my medical assistant in the American Association of Medical Assistants, Inc. Please send more information to:

Name____________________

Business Address____________________

City/State/Zip____________________

County____________________ Phone____________________

Names and Addresses of Assistants____________________

Clip and mail to: AAMA, NYSS, Inc., c/o Department of Information, Medical Society State of New York,
420 Lakeville Road, P.O. Box 5404, Lake Success, New York 11042.

Jacob I. Lamstein, MD, North Miami Beach, Florida. Died May 22, 1985; age 82. He was a 1928 graduate of the University of Maryland School of Medicine, Baltimore. Dr Lamstein was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Eli-Herz Leneman, MD, Forest Hills. Died March 6, 1985; age 54. He was a 1957 graduate of the Faculty of Medicine St. Joseph's University, Beirut. Dr Leneman was a Diplomate of the American Board of Orthopaedic Surgeons, and a member of the American Academy of Orthopaedic Surgeons, the New York County Medical Society and the Medical Society of the State of New York.

Edward Opin, MD, Scarsdale. Died May 13, 1985; age 83. He was a 1925 alumnus of New York University College of Medicine. Dr Opin was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Ralph Peimer, MD, New York. Died May 29, 1985; age 69. He was a 1940 alumnus of Rush Medical College. Dr Peimer was a Fellow of the American College of Surgeons, a Diplomate of the American Board of Otolaryngology, and a member of the New York County Medical Society and the Medical Society of the State of New York.

Edward Kessinger Reid, MD, Rome. Died June 11, 1985; age 78. He was a 1933 graduate of the State University of New York Upstate Medical Center. Dr Reid was a Diplomate of the American Board of Radiology. His memberships included the American Board of Legal Medicine, the Radiological Society of North America, Inc., the Medical Society of the County of Oneida and the Medical Society of the State of New York.

Daniel N. Rosenberg, MD, Woodmere. Died March 19, 1985; age 71. Dr Rosenberg was a 1943 alumnus of Middlesex University School of Medicine. He was a Fellow of the American Academy of Family Physicians, and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Joseph E. Rubinstein, MD, New York. Died June 15, 1985; age 83. He was a 1930 graduate of McGill University Faculty of Medicine, Montreal. Dr Rubinstein was a Fellow of the American Psychiatric Association, a Diplomate of the American Board of Psychiatry and Neurology, and a member of the New York Neurological Society, the New York County Medical Society and the Medical Society of the State of New York.

William Arthur Sabatini, MD, Bronx. Died June 17, 1985; age 77. Dr Sabatini was a 1935 graduate of St. Louis University School of Medicine. He was a member of the American Academy of Family Physicians, the Bronx County Medical Society and the Medical Society of the State of New York.

Herbert John Smith, MD, New York. Died June 9, 1985; age 80. Dr Smith was a 1940 graduate of the University of Dublin School of Physics. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Frederick Stern, MD, New York. Died May 19, 1985; age 83. Dr Stern was a 1931 graduate of Rheinische Friedrich-Wilhelms-Universität Medizinische Fakultat Bonn, Prussia. He was a member of the American Geriatrics Society, the New York County Medical Society and the Medical Society of the State of New York.

Abraham Bernard Tamis, MD, Sun City, Arizona. Died June 5, 1985; age 86. He was a 1922 alumnus of New York University College of Medicine. Dr Tamis was a Fellow of the American College of Obstetricians and Gynecologists and the American College of Surgeons, and a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Academy of Medicine, the Bronx County Medical Society and the Medical Society of the State of New York.

Guido Vinci, MD, Long Island City. Died June 16, 1985; age 51. He was a 1961 graduate of Facoltà di Medicina e Chirurgia Università di Palermo. Dr Vinci was a member of the Queens County Medical Society and the Medical Society of the State of New York.

NATIONAL EMERGENCY SERVICES, INC. (N.E.S.)

Directorships, full- and part-time emergency department opportunities are available throughout New York State and the country for primary care physicians with ER experience. Enjoy a variety of practice settings, competitive remuneration, paid malpractice insurance, and flexible scheduling. For information and application materials contact the recruitment department at (516) 365-4010, (Outside N.Y. State 1-800-645-4848) or send C.V. to N.E.S., One Hollow Lane, Suite 304, Lake Success, N.Y. 11042.

INTERNIST

Internist with W.C. Board S.J. rating needed full time for my clinics in Buffalo and Rochester to perform evaluations and examinations.

Kick the hassle of practice. Enjoy banker's hours and high income. Reply with C.V. to: H. Lewis, M.D., 3641 Quail Hollow, Bloomfield Hills, Michigan, 48013.

Positions available in maturing Family Practice Department with fully-accredited Residency Program, required third year medical student and other academic advantages:

- **Community Medicine Coordinator and Assistant Program Director**
- **Assistant Coordinator of Medical Student Education**

Please respond with letter of interest and Curriculum Vitae to:

Dr. Alice E. Fruehan
Professor and Chairman
MS-516, Albany Medical College
Albany, New York 12208

- **Pleasant recreational/family living**
- **Attractive fringe benefits and Faculty Development package**
- **Vibrant colleagues, challenging opportunities**

EOAAE M/F

IMPORTANT NOTICE RE: **Malpractice Insurance**

PREFERRED RISK
MEDICAL LIABILITY INSURANCE
ON AN OCCURRENCE BASIS

ATTENTION
RESIDENTS
Part-time
Rates
Available

Medical Quadrangle, through Frontier Insurance Co. in Monticello, New York, offers a Preferred Risk Program with limits of $1 million/$3 million; specifically "tailored" part-time rates to meet your individual practice needs; beginning doctor rates and discounts for participating in our risk management program.

MEDICAL
MQ
QUADRANGLE

Call or Write:
Medical Quadrangle, Inc.
P.O. Box 368
103 Plainfield Avenue
Bedford Hills, NY 10507
(800) 624-6691

Send me information on your program. (Return this to address at left of coupon.)

Name ________________

Address ________________

City ______ State ______ Zip ______

Telephone ( ) ________________

Angina comes in many forms...

So does

SORBITRATE®

(ISOSORBIDE DINITRATE)

Unsurpassed flexibility in nitrate therapy.

© 1985 ICI AMERICAS INC.

See following page for brief summary of prescribing information.

SORBITRATE®
(ISOSORBIDE DINITRATE)

Please consult full prescribing information before use. A summary follows:

INDICATIONS AND USAGE: SORBITRATE (isosorbide dinitrate) is indicated for the treatment and prevention of angina pectoris. All dosage forms of isosorbide dinitrate may be used prophylactically to decrease frequency and severity of anginal attacks and can be expected to decrease the need for sublingual nitroglycerin.

The sublingual and chewable forms of the drug are indicated for acute prophylaxis of angina pectoris when taken a few minutes before situations likely to provoke anginal attacks Because of a slower onset of effect, the oral forms of isosorbide dinitrate are not indicated for acute prophylaxis

CONTRAINDICATIONS: SORBITRATE is contraindicated in patients who have shown purported hypersensitivity or idiosyncrasy to it or other nitrates or nitrites Epinephrine and related compounds are ineffective in reversing the severe hypotensive events associated with overdose and are contraindicated in this situation.

WARNINGS: The benefits of SORBITRATE during the early days of an acute myocardial infarction have not been established If one elects to use organic nitrates in early infarction, hemodynamic monitoring and frequent clinical assessment should be used because of the potential deleterious effects of hypotension.

PRECAUTIONS: General: Severe hypotensive response, particularly with upright posture, may occur with even small doses of SORBITRATE The drug should therefore be used with caution in subjects who may have blood volume depletion from diuretic therapy or in subjects who have low systolic blood pressure (eg, below 90 mmHg) Paradoxical bradycardia and increased angina pectoris may accompany nitrate-induced hypotension. Nitrate therapy may aggravate the angina caused by hypertrophic cardiomyopathy.

Marked symptomatic, orthostatic hypotension has been reported when calcium channel blockers and organic nitrates were used in combination. Dose adjustment of either class of agents may be necessary.

Tolerance to this drug and cross-tolerance to other nitrates and nitrites may occur. Tolerance to the vascular and antianginal effects of isosorbide dinitrate or nitroglycerin has been demonstrated in clinical trials, experience through occupational exposure, and in isolated tissue experiments in the laboratory The importance of tolerance to the appropriate use of isosorbide dinitrate in the management of patients with angina pectoris has not been determined However, one clinical trial using treadmill exercise tolerance (as an end point) found an 8-hour duration of action of oral isosorbide dinitrate following the first dose (after a 2-week placebo washout) and only a 2-hour duration of effect of the same dose after 1 week of repetitive dosing at conventional dosing intervals. On the other hand, several trials have been able to differentiate isosorbide dinitrate from placebo after 4 weeks of therapy and, in open trials, an effect seems detectable for as long as several months

Tolerance clearly occurs in industrial workers continuously exposed to nitroglycerin. Moreover, physical dependence also occurs since chest pain, acute myocardial infarction, and even sudden death have occurred during temporary withdrawal of nitroglycerin from the workers. In clinical trials in angina patients, there are reports of anginal attacks being more easily provoked and of rebound in the hemodynamic effects soon after nitrate withdrawal The relative importance of these observations to the routine, clinical use of isosorbide dinitrate is not known However, it seems prudent to gradually withdraw patients from isosorbide dinitrate when the therapy is being terminated, rather than stopping the drug abruptly.

Information for Patients: Headache may occur during initial therapy with SORBITRATE Headache is usually relieved by the use of standard headache remedies or by lowering the dose and tends to disappear after the first week or two of use.

Drug Interactions: Alcohol may enhance any marked sensitivity to the hypotensive effect of nitrates.

Isosorbide dinitrate acts directly on vascular smooth muscle, therefore, any other agent that depends on vascular smooth muscle as the final common path can be expected to have decreased or increased effect depending on the agent.

Carcinogenesis, Mutagenesis, Impairment of Fertility: No long-term studies in animals have been performed to evaluate the carcinogenic potential of this drug A modified two-litter reproduction study in rats fed isosorbide dinitrate at 25 or 100 mg/kg/day did not reveal any effects on fertility or gestation or any remarkable gross pathology in any parent or offspring fed isosorbide dinitrate as compared with rats fed a basal-controlled diet.

Pregnancy Category C: Isosorbide dinitrate has been shown to cause a dose-related increase in embryotoxicity (increase in mummified pups) in rabbits at oral doses 35 and 150 times the maximum recommended human daily dose. There are no adequate and well-controlled studies in pregnant women. SORBITRATE should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: It is not known whether this drug is excreted in human milk Because many drugs are excreted in human milk, caution should be exercised when SORBITRATE is administered to a nursing woman.

Pediatric Use: The safety and effectiveness of SORBITRATE in children has not been established

ADVERSE REACTIONS: Adverse reactions, particularly headache and hypotension, are dose-related In clinical trials at various doses, the following have been observed

Headache is the most common (reported incidence varies widely, apparently being dose-related, with an average occurrence of about 25%) adverse reaction and may be severe and persistent. Cutaneous vasodilation with flushing may occur. Transient episodes of dizziness and weakness, as well as other signs of cerebral ischemia associated with postural hypotension, may occasionally develop (the incidence of reported symptomatic hypotension ranges from 2% to 36%). An occasional individual will exhibit marked sensitivity to the hypotensive effects of nitrates and severe responses (nausea, vomiting, weakness, restlessness, pallor, perspiration, and collapse) may occur even with the usual therapeutic dose. Drug rash and/or exfoliative dermatitis may occasionally occur Nausea and vomiting appear to be uncommon. Case reports of clinically significant methemoglobinemia are rare at conventional doses of organic nitrates The formation of methemoglobin is dose-related and, in the case of genetic abnormalities of hemoglobin that favor methemoglobin formation, even conventional doses of organic nitrate could produce harmful concentrations of methemoglobin.

DOSAGE AND ADMINISTRATION: For the treatment of angina pectoris, the usual starting dose for sublingual SORBITRATE is 2.5 to 5 mg; for chewable tablets, 5 mg; for oral (swallowed) tablets, 5 to 20 mg, and for controlled-release forms, 40 mg

SORBITRATE should be titrated upward until angina is relieved or side effects limit the dose In ambulatory patients, the magnitude of the incremental dose increase should be guided by measurements of standing blood pressure.

The initial dosage of sublingual or chewable SORBITRATE for prophylactic therapy in angina pectoris patients is generally 5 or 10 mg every 2 to 3 hours Adequate controlled clinical studies demonstrating the effectiveness of chronic maintenance therapy with these dosage forms have not been reported.

SORBITRATE in oral doses of 10 to 40 mg given every 6 hours or in oral controlled-release doses of 40 to 80 mg given every 8 to 12 hours is generally recommended The extent to which development of tolerance should modify the dosage program has not been defined *The oral controlled-release forms of isosorbide dinitrate should not be chewed*

DOSAGE FORMS AVAILABLE: Sublingual Tablets (2.5, 5, 10 mg), Chewable Tablets (5, 10 mg); Oral Tablets (5, 10, 20, 30, 40 mg); Sustained Action Tablets (40 mg)

STUART PHARMACEUTICALS
Division of ICI Americas Inc.
Wilmington, DE 19897

STR-2282

Jan 17–19. **Confronting the Malpractice Crisis.** Hyatt Islandia, San Diego. 15 Cat 1 Credits. Info: Division of Continuing Medical Education, Wesley Medical Center, 550 North Hillside, Wichita, KS 66110. Tel: (913) 451-3103.

Jan 20–24. **9th Annual San Diego Postgraduate Assembly in Surgery.** 32 Cat 1 Credits. Info: Office of Continuing Medical Education, M-017. University of California San Diego School of Medicine, La Jolla, CA 92093. Tel: (619) 452-3940.

Jan 27–31. **Controversies and Challenges in Primary Care.** 25 Cat 1 Credits. Info: Extended Program in Medical Education, Registration Office, 1456 Ninth Ave, San Francisco, CA 94122. Tel: (415) 666-5808.

COLORADO

Jan 11–14. **Keystone Summit on Allergy, Immunology, Pulmonology, and Pharmacology.** 16 Cat 1 Credits. Info: Helga Cole, National Jewish Center for Immunology and Respiratory Medicine, 3800 East Colfax Ave, Denver, CO 80206 Tel: (303) 398-1379.

Jan 25–Feb 1. **Gastroenterology Update: 1986.** 20 Cat 1 Credits. Lion Square Lodge and Conference Center, Vail. Info: Jeanne Ryan, Program Coordinator, Office of Continuing Education, Johns Hopkins University School of Medicine, 720 Rutland Ave, Turner 22, Baltimore, MD 21205.

GEORGIA

Jan 24–26. **Educational Symposium: Breast Reconstruction.** 16 ½ Cat 1 Credits. St. Josephs Hospital. Info: Breast Reconstruction Symposium, John H. Hartley, Jr, MD, 3280 Howell Mill Rd, #111, Atlanta, GA 30327.

MASSACHUSETTS

Jan 19–31. **Program for Chiefs of Clinical Services.** Info: Office of Continuing Education, Harvard School of Public Health, 677 Huntington Ave, Boston MA 02115. Tel: (617) 732-1171.

Jan 24–29. **The Problem Foot and Ankle.** 20 Cat 1 Credits. Info: Department of Continuing Medical Education, Boston University School of Medicine, 80 East Concord St, Boston MA 02118. Tel: (617) 638-4605.

APPRAISALS-SALES-RELOCATION
Medical Practices—All Specialties

MEDICAL PRACTICES TRANSFER
Frederick Katz, Ph.D. & Assoc.

42-27 Union Street, Flushing, N.Y. 11355
Tel. (718) 445-9797 (any time)

PRACTICES WANTED

OPHTHALMOLOGIST, BOARD CERTIFIED desires to purchase practice in New York City or suburban area. Confidence maintained. Please contact Dept. 168, c/o NYSJM.

POSITIONS WANTED

PULMONARY INTERNIST, 30, ABIM certified, completing fellowship June 1986. Fully trained in all aspects of pulmonary medicine. Seeks hospital-based or subspecialty group practice affiliated with a teaching institution. Reply Harlan Weinberg, M.D., 337 N. Palm Drive, Beverly Hills, CA. 90210, Apt. 8.

GASTROENTEROLOGIST-ABIM, University-trained in all endoscopic procedures, ERCP, PEG, peritoneoscopy and consultative gastroenterology/hepatology. Seeking private practice associateship leading to partnership in the NYC/LI area. Available after completion of fellowship in July 1986. Reply Dept. 399 c/o NYSJM.

INTERNIST, relocating to upstate New York, seeks job/practice opportunity in internal medicine. Emergency room or locum tenums type work acceptable as well. Reply Dept. 400 c/o NYSJM.

M.D. ANST BC AVAILABLE. Cln. exp. ACLS. Deal direct full/part time position. J.D. 2401 Bay Blvd., #C, Indian Rocks Beach, Fla. 33535. (813) 595-2969.

EQUIPMENT

MEDICAL EQUIPMENT FOR SALE: Diagnostic Video Imager & Recorder by Matrix. Amino Acid Analyzer by Hitachi Perkin Elmer. Excellent condition, best offer. (516) 742-9614 Evenings after 7 P.M.

REAL ESTATE FOR SALE OR RENT

FOR SALE WEST 86TH STREET MANHATTAN. Large professional co-op. Move-in condition. Immediate occupancy. Private street entrance. 1500 sq. ft., 10 room suite. Excellent transportation. Call (212) 265-1310.

BROOKLYN, OCEAN PARK AT NEPTUNE AVE. Professional apartment in Trump Village. Prime, densely populated residential area. 5 room suite, 2 baths. Ideal doctor, dentist, psych., etc. Call Ms. Martin (718) 743-3375.

REAL ESTATE FOR SALE OR RENT—CONT'D

LOCATION, LOCATION NEAR 5 HOSPITALS. Great Neck corner property in Kensington. 3 examining rooms and lab, 4 bedrooms, 2 servants rooms, 4½ baths, double sinks. Front and back stairways. 2 car garage with electric openers. 4 blocks to station. $535,000. Telephone (516) 487-3474 or 751-8880.

APPROX. 1,000 SQ. FT., 57th ST., SUTTON AREA. Private street entrance, suitable office for Doctor, Dentist, Psychiatrist, Lawyer, C.P.A. Full kitchen, powder room. $2,750.00 per month. For more information call (212) 517-9743 or (212) 427-4624.

PROFESSIONAL CO-OP APARTMENT FOR SALE Prime location in the heart of Forest Hills, Queens. Suitable for any Medical Specialty. No doctor in 562 tenant building. Ground floor, convenient for subways and buses. Central air-conditioning, garage avaialble. Call (718) 263-0374, 1 P.M. to 6 P.M weekdays.

SPLENDID PERSONAL/PROFESSIONAL RESIDENCE, Rockville Centre, sprawling expanded ranch, 4 bedrooms 4 full baths, formal dining room, eat-in kitchen, 2 wood burning fireplaces, in ground swimming pool plus professional suite. 115 x 130 lot. Ideal professional or personal residence, $315,000.00. Frank Anderson, agent, Merrill Lynch Realty, Carl Burr Inc., (516) 872-8890.

HEWLETT HARBOR WATERFRONT. Historic sprawling 6 bedroom, 5½ baths, 2 fireplaces, Country French home, fully bulkheaded on open water, leaded glass windows, solid oak floors, beamed ceilings, Master Bedroom suite with cathedral ceiling and marble fireplace. Unusual property at end of cul-de-sac. A-one-of-a-kind opportunity. Principals only. $795,000. (516) 374-0660.

BAY SHORE, Leasing new luxurious medical building. Located near Southside Hospital. Minimum suite 740 sq. ft. Large parking area, handicapped designed. Individual gas heat, a/c-electric. (516) 665-4064, or 665-4482.

WANTED: MEDICAL OFFICE BUILDINGS. As principals we are seeking "existing buildings"—fully tennanted by physicians, dentists and health care services. Contact: Paul Gellert, Gelco Associates, 155 West 68th St., N.Y., N.Y. 10023; (212) 724-7900.

MANHATTAN'S EAST SIDE, 133 East 73rd St., N.Y.C. Lexington Professional Center, Inc. Part time & full time medical, dental, psychiatric office suites. Furnished & equipped. 24 hour answering service; receptionist. Mail service; cleaning. X-ray & clinical laboratory on premises. No leases necessary. Rent by the hour or full-time. (212) 861-9000.

MEDICAL BUILDING—Excellent N.Y.C. location. 3200 sq. ft., ground floor. Adjacent to expanding hospital in Queens. Near all transportation, heavy traffic. Ideal for Medical Clinic or Group Practice. Excellent opportunity, Sale or Lease. Call (718) 526-3368, owner.

PHYSICIANS IN RECOVERY

HELP!

The MSSNY wants the help of physicians who are recovering from chemical dependence (alcohol and other drugs) or mental illness. Other physicians attempting recovery need your guidance and support. The Committee for Physicians' Health (Physicians' Committee) can put you in touch. Please call or write.

We'll keep it confidential.

Barton A. Harris, M.D.
Medical Director
Committee for Physicians' Health
Medical Society of the
State of New York
P.O. Box 5404
Lake Sucess, NY 11042

516-488-7777

REAL ESTATE FOR SALE OR RENT—CONT'D

JERICHO, L.I., HOME + OFFICE. HOME: entry on fine residential street. OFFICE: entry on Broadway/Rt. 107. Contemporary decor distinguishes this residence that offers living room, dining room, bright new kitchen, big den plus family room, 3 bedrooms, 2½ baths. Spacious is the word for the 3 room + ½ bath office wing. Other features include 1/3 acre with private yard; 2 car garage; built-ins include central air, telephone intercom, etc. For information call Birch Tree (516) 496-3434.

TIMESHARING MEDICAL OFFICES. Start a new practice, expand an established practice, retire and practice part-time, without the hassle and expense of establishing or managing an office. Dr Share, a medical timesharing network, now has five new fully equipped prestigous Manhattan locations as part of our network. Please, write or call for more information, Dr Share, P.O. Box 8051, Garden City, NY (516) 486-8400.

16 ROOM HOUSE in beautiful Bay Ridge, Brooklyn, NY. Detached corner property, park-like surroundings, ample parking, near bus and subways. Excellent for home and office. Reasonably priced for quick sale. Call owner (718) 748-7088.

MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 110 E. 42nd Street, NYC. (212) 697-2950 or evenings (914) 723-0799.

ARE YOU UNDER INVESTIGATION BY THE STATE LICENSING AUTHORITIES? For consultation call Susan Kaplan, Attorney-at-Law, (212) 877-5998, concentrating in the vital legal problems affecting your professional license with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County.

1986 CME CRUISE/CONFERENCES on selected Medical Topics—Caribbean, Mexican, Hawaiian, Alaskan, Mediterranean. 7-12 days year-round. Approved for 20-24 CME Cat. 1 credits (AMA/PRA) & AAFP prescribed credit. Distinguished professors. Fly Roundtrip Free on Caribbean, Mexican, & Alaskan Cruises. Excellent group fares on finest ships. Registration limited. Pre-scheduled in compliance with IRS requirements. Information: Intenational Conferences, 189 Lodge Ave., Huntington Station, NY 11746. (516) 549-0869.

DOCTOR, YOU CAN'T BEAT the quality or the price! Holter Monitor Scanning Service. Physician owned, trained and supervised. No contracts to sign. We can arrange for lease/purchase of Holter equipment. New dual channel recorders with test cables $1,550. Now using UP Service for faster turn around time. DCG Interpretation (313) 879-8860.

MISCELLANEOUS—CONT'D

TAX ATTORNEY AND PENSION ACTUARY Specialist—Former IRS pension plan specialist and revenue agent—TEFRA amendments, pension and profit sharing plan annual administration including initial IRS qualification, annual filings, actuariat certification and employee statements of participation—partnership agreements and professional incorporation—No insurance required—references upon request—Jack Wachstock, PC, 3000 Marcus Ave., Lake Success, N.Y. 11530 (516) 437-4848.

DOCTORS: State-of-the-art dual channel Holter recorders for only $995.00. Will arrange lease or purchase. Excellent scanning service available. Only $50.00 for scanned report. Fast, courteous and professional service. Call today (516) 938-6400, ask for Diane Lipari.

PROFESSIONAL MISCONDUCT ATTORNEYS. William L. Wood Jr., formerly Executive Director of the New York State Office of Professional Discipline and Anthony Z. Scher, formerly Director of Prosecutions, announce the opening of their law office, Wood & Scher. Our recent tenure as chief enforcement officers has given us experience which will allow us to represent physicians in professional misconduct preceedings, malpractice licensure, license restoration, controlled drug proceedings, reimbursement disputes, purchase and sale of professional practices and all other matters affecting the professional lives and careers of practioners. Wood & Scher, Attorneys at Law, One Chase Road, Scarsdale, New York 10583, Telephone 914-723-3500.

PHYSICIANS SIGNATURE LOANS TO $50,000. Take up to 7 years to repay with no pre-payment penalties. Competitive fixed rate, with no points, fees or charges of any kind. Use for taxes, investment, consolidation or any other purpose. Prompt, courteous service. Physicians Service Association, Atlanta GA. Serving MD's for over 10 years. Toll-free (800) 241-6905.

HAVE YOU BEEN CONVICTED OF A CRIME? YOUR MEDICAL LICENSE MAY BE AFFECTED! For consultation call Susan Kaplan, Attorney-at-Law, (212) 877-5998, concentrating in the vital legal problems affecting your professional license with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County.

ARCHITECTURAL DESIGN AND CONSTRUCTION: Designers, builders of custom professional offices. Consulting services available. Whether you're opening a new office, relocating, remodeling or expanding an existing office in New York City area and adjacent New Jersey counties. Donald Young, Inc. Tel: (718) 948-5191.

PERSON WITH MEDICAL BACKGROUND WANTED to care for elderly lady on respirator in New York City. F.M.G. considered. Telephone: (212) 628-8626.

WEIGHT LOSS CONSULTANT. If your preprinted 1000 calorie diet doesn't work, an M.D. experienced in all aspects of weight reduction will help you set up an ethical, profitable, simple, effective weight loss program for your office practice. Contact Allan Lazar, M.D., 740 Carroll Place, Teaneck, NJ 07666. (201) 568-3742.

WEEKLY SEMINARS

Most major ski areas, Club Med, Disney World, Cruising aboard Sailboats in the Virgin Islands or a Mississippi Paddlewheeler.

Topic: Medical/Legal Issues

Accredited *Fee $175*

Current Concept Seminars, Inc. *(since 1980)*
3301 Johnson St., Hollywood, FL 33021
(800) 428-6069

MISCELLANEOUS—CONT'D

ARE YOU HAVING DIFFICULTIES GETTING YOUR MEDICAL LICENSE RESTORED? For consultation call Susan Kaplan, Attorney-at-Law, (212) 877-5998, concentrating in the vital legal problems affecting your professional license with extensive trial and administrative experience—formerly Assistant Chief of Prosecution and Deputy Director of Prosecution for New York State's Office of Professional Discipline (the state agency responsible for regulating NYS's 31 licensed professions), and as an Assistant District Attorney in Nassau County.

MEDICAL AND SCIENTIFIC WRITING AND EDITING. Experienced medical/technical writer and editor provides expedient, expert writing or editing of your manuscript, proposal, presentation, office manual, etc. Carl F. Husa, Jr., M.A., Husa's Paragraphics, P.O. Box 531, Olean, New York 14760 (716) 372-5284.

BACK COPIES NEW YORK STATE JOURNAL OF MEDICINE

For a limited time, back copies may be purchased at the following rates plus postage.

1971-1980 . . . $2.00
1981-1983 . . . 3.00

Exception: for the special issue of December 1983 which costs $5.00.

Send your order to:
Circulation Dept., New York State Journal of Medicine, 420 Lakeville Road, P.O. Box 5404, Lake Success, NY 11042.

Inquiries: (516) 488-6100, Circulation Dept.

Medical Society of the State of New York
ANNUAL CME ASSEMBLY
MARCH 7-9, 1986
The New York Hilton, New York City
Combined With The Annual Meeting of
The House of Delegates
MARCH 6-9

Safe Deposit.

Most people look for a safe place to deposit their money. A place where they can keep an eye on it.

Well, buying U.S. Savings Bonds is a safe and easy way to a safe deposit. Because Bonds can be replaced if lost, stolen, or destroyed.

Plus, Bonds offer guarantees: a guaranteed way to save, through the Payroll Savings Plan; a guaranteed interest rate; and guaranteed tax benefits. All guaranteed by America.

Add it all up. U.S. Savings Bonds are the safest deposit.

Keep your eye on them *and* watch them grow.

Take stock in America.

UNITED STATES SAVINGS BONDS

When you put part of your savings into U.S. Savings Bonds you're helping to build a brighter future for your country and for yourself.

Ad Council A public service of this publication and The Advertising Council.

Index to Advertisers

The Albany Medical College of Union University	34B
American Academy of Compensation Medicine	8A
Classified Advertising	6A,8A,34B,37A,38A
Eastern Educational Programs	6A
HealthWays	3rd Cover
International Oculoplastic Society Congress	5A
Knoll Pharmaceuticals	2A,3A,4A
Eli Lilly and Company	2nd Cover
Marion Laboratories	9A,10A
Medical Quadrangle	34B
A. H. Robins Company	7A,40A
Stuart Pharmaceuticals	35A,36A
Upjohn Company	4th Cover

This Publication is available in Microform.

University Microfilms International

Please send additional information
for ______________________ (name of publication)
Name______________________
Institution______________________
Street______________________
City______________________
State__________ Zip__________
300 North Zeeb Road, Dept. P.R., Ann Arbor, Mi. 48106

DAC
NDC 0031-8680-25
A·H·ROBINS
Robitussin®-DAC
EXPECTORANT
NASAL DECONGESTANT
ANTITUSSIVE
KEEP THIS AND ALL DRUGS OUT OF REACH OF CHILDREN. IN CASE OF ACCIDENTAL OVERDOSE, SEEK PROFESSIONAL ASSISTANCE OR CONTACT A POISON CONTROL CENTER IMMEDIATELY.
16 FL.OZ. (1PT.)

FOR SEVERE COUGHS, CAN YOU REMEMBER THESE LETTERS?

You know Robitussin®, and the "DAC" formula gives you just what you need to manage those severe coughs complicated with nasal congestion . . . Decongestant And Codeine!

Robitussin®-DAC Ⓥ

Each 5 ml (1 teaspoonful) contains:

Guaifenesin, USP	100 mg
Pseudoephedrine Hydrochloride, USP	30 mg
Codeine Phosphate, USP	10 mg

(Warning: May be habit forming)

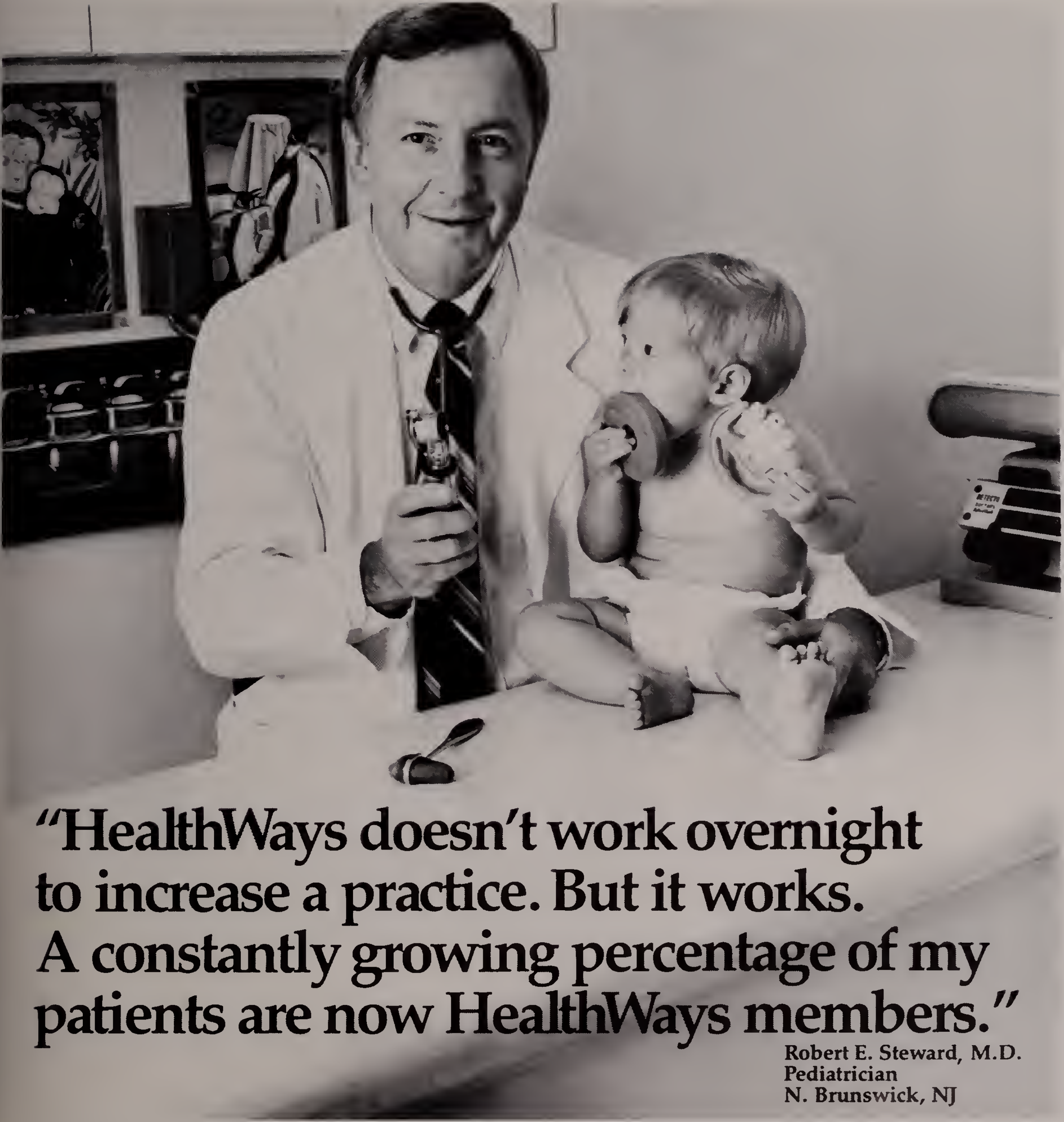
Alcohol 1.4 percent

℞ ONLY

A·H·ROBI

Pharmaceutical Division

Richmond, Virginia 23261-



"HealthWays doesn't work overnight to increase a practice. But it works. A constantly growing percentage of my patients are now HealthWays members."

Robert E. Steward, M.D.
Pediatrician
N. Brunswick, NJ

"When I first participated in HealthWays, they opened a whole new source of patients for me. Now it didn't grow overnight, but grow it did and continues to do so. And since many of my established patients have signed up for HealthWays, they continue to see me as always. After all, the big difference between non-HealthWays patients and HealthWays patients is that their health care costs them less. Plus the fact that they're covered for well care visits which are very important during a child's early years.

As far as having a good working relationship with HealthWays, I consider it the best. Their compensation policy is traditional fee-for-service. The bills go directly to them, and the payments are prompt. I prefer that to the capitation system where you are paid a fixed fee no matter how often you see a patient. And since I participate in HealthWays through my IPA (Individual Practice Association), and not as an individual, I have a strong corporate body of physicians representing me.

Will HealthWays help your practice as it did mine? I would certainly think it will."

To start HealthWays working for you and your patients, call HealthWays' IPA Department at 1-800-624-0720 in New Jersey or 201-636-6200 out-of-state.

HealthWays
THE WAY TO BETTER HEALTH CARE

New
Motrin® 800 TABLETS mg
ibuprofen

Extra strength
Convenience
Economy

Upjohn

The Upjohn Company
Kalamazoo, Michigan 49001 USA

© 1985 The Upjohn Company

J-5491 June 1985